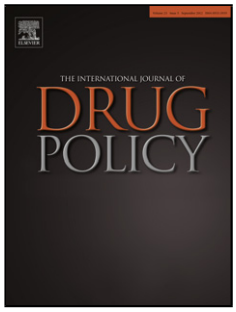

Title: Mortality related to Novel Psychoactive Substances in Scotland, 2012: an exploratory study

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Highlights

- Deaths involving Novel Psychoactive Substances (NPS) affect small numbers at population level, however the relatively high incidence of these substances is noteworthy when considering DRDs within the known problem drug user population.
- Deaths rarely involve NPS alone and typically involve a range of other substances.
- The majority of deaths involved Benzodiazepine-type NPS drugs, mainly Phenazepam.
- Differences in the profile of deaths involving Benzodiazepine and Stimulant-type NPS emerged.

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Mortality related to Novel Psychoactive Substances in Scotland, 2012: an exploratory study

Keywords

Novel Psychoactive Substances; drugs; mortality; overdose

Abstract

Background

The growth of Novel Psychoactive Substances (NPS) over the last decade, both in terms of availability and consumption, is of increasing public health concern. Despite recent increases in related mortality, the circumstances surrounding and characteristics of individuals involved in NPS deaths at a population level remain relatively unknown.

Methods

The Scottish National Drug Related Death Database (NDRDD) collects a wide-range of data relating to the nature and circumstances of individuals who have died a drug-related death (DRD). We conducted exploratory descriptive analysis of DRDs involving NPS recorded by the NDRDD in 2012. Statistical testing of differences between sub-groups was also conducted where appropriate.

Results

In 2012, we found 36 DRDs in Scotland to have NPS recorded within post-mortem toxicology. However, in only 23 of these cases were NPS deemed by the reporting pathologist to be implicated in the actual cause of death The majority of NPS-implicated DRDs involved Benzodiazepine-type drugs (13), mainly Phenazepam

(12). The remaining 10 NPS-implicated deaths featured a range of different Stimulant-type drugs. The majority of these NPS-implicated deaths involved males and consumption of more than one drug was recorded by toxicology in all except one case.

NPS-implicated deaths involving Benzodiazepine-type NPS drugs appeared to involve older individuals known to be using drugs for a considerable period of time, many of whom had been in prison at some point in their lives. They also typically involved combinations of opioids and benzodiazepines; no stimulant drugs were co-implicated.

Deaths where Stimulant-type NPS drugs were implicated appeared to be a younger group in comparison, all consuming two or more Stimulant-type drugs in combination.

Conclusion

This exploratory study provides an important insight into the circumstances surrounding and characteristics of individuals involved in NPS deaths at a population level. It identifies important issues for policy and practice, not least the prominent role of unlicensed benzodiazepines in drug-related mortality, but also the need for a range of harm reduction strategies to prevent future deaths.

Introduction

Although not a new phenomenon, the growth of Novel Psychoactive Substances (NPS) over the last decade, both in terms of availability and consumption, is of increasing public health concern. The number of new NPS reported to the European

Monitoring Centre for Drugs and Drug Addiction (EMCDDA) has increased year on year from 24 in 2009 to 81 in 2013 with over 350 substances now being monitored (EMCDDA, 2014). In addition, the United Nations Office on Drugs and Crime (UNODC) estimates that a total of 348 NPS had been identified by member states by mid-2013 (UNODC, 2014).

The scale of NPS use globally is less clear due to an absence of epidemiological data from robust population-based samples and only limited data from a few countries on specific substances and sub-populations (UNODC, 2013a). Moreover, a lack of common definitions, the large and increasing number and classes of substances regarded as NPS, and differences in legislation further complicate the ability to accurately understand use within and across countries.

As with prevalence, there is a dearth of evidence on NPS-related harm. Most research to date on NPS-related harms is in relation to fatal poisonings and limited to deaths involving specific drugs (Corkery, Schifano, & Ghodse, 2012a) or individual case-studies (Maskell, De Paoli, Seneviratne, & Pounder , 2011). However, to our knowledge, there have been no published studies to date on population based cohorts of NPS-related death which describe, in detail, the characteristics of the individuals involved and the circumstances surrounding their deaths. Thus, the aim of this study was to provide an exploratory descriptive account of drug-related deaths (DRDs) involving NPS recorded by the Scottish National Drug Related Death Database (NDRDD) in 2012, including consideration of cases where NPS drugs are recorded within toxicology and a particular focus on cases where NPS drugs were deemed to be implicated in the cause of death

Methods

The UN categorises the current NPS market into six main groups of drugs: synthetic cannabinoids; synthetic cathinones; ketamine; phenethylamines; piperazines; plant-based substances; and a group of miscellaneous substances that contain recently identified NPS (e.g. tryptamines) that do not fit into any of these groups (UNODC, 2013b). Other pharmaceutical medications not licensed for use within the UK, for example benzodiazepines such as Phenazepam, have also been included within the broad definition of NPS by the UK Advisory Council on the Misuse of Drugs (ACMD) (ACMD, 2011) and the UK National Programme on Substance Abuse Deaths (Corkery, Claridge, Loi, Goodair, & Schifano, 2014).

The NDRDD adopts the same definition as used by National Records of Scotland (NRS) (2013) when including NPS within the dataset: *"The term 'New Psychoactive Substances' (NPS) is meant to cover the kinds of substances that people have, in recent years, begun to use for intoxicating purposes. NPS include so-called 'legal highs' (by which is meant substances which were legally available at the time of the death, whether or not they have since become controlled). In general, when an NPS first became available, it would not have been a controlled substance under the Misuse of Drugs Act 1971. Some NPS may still not be controlled under the Act. The definition of NPS therefore includes current so-called 'legal highs', and also substances which used to be described as 'legal highs' but are now controlled."*

NDRDD criteria for counting NPS-related deaths:

Inclusion and exclusion criteria for counting statistics on NPS-related deaths in a given year (here 2012) can be categorised in three ways:

- NPSs which were already controlled substances at the start of 2012;
- NPSs which became controlled substances during 2012 (i.e. whose classification changed during the period covered by these figures for deaths involving NPS); and
- NPSs which were not controlled substances at the end of 2012 (some of which have since become controlled substances).

NRS criteria for counting NPS-related deaths:

A death due solely to one of these drugs would be counted in the NRS National Statistics publication of drug-related deaths if the person died on or after the specified date that the drug became controlled. A death due solely to one of these NPS drugs would not be counted in NRS National Statistics on drug-related death (DRD) if it involved a drug that were not controlled at the time at the time of death.

NRS also provides additional information about whether the drugs recorded in toxicology were implicated in the death or not. This is based on pathologist reports which accompany the majority of DRDs. In the absence of such information NRS assumes all drugs mentioned on the death certificate were implicated in the death.

It is also important to note that NRS National Statistics are based on the date the death is registered rather than the date on which the person died. In Scotland, the numbers reported in a year are effectively the same because all deaths must be registered within eight days of death having been ascertained, without exception. In England and Wales, there tends to be a delay between the date of death and the date of registration because all sudden deaths are referred to the Coroner and are

not registered until they have been reported, This potentially leads to a lower number of deaths being registered in the latest reported year for deaths occurring in that year.

The Scottish NDRDD is notified of DRDs from local data coordinators. Cases are then matched against those reported by NRS. The two datasets have become closely matched over time (only 7% of NRS recorded DRDs were missing from NDRDD returns in 2012). The NDRDD therefore reports on a subset of DRDs in Scotland and is therefore not a National Statistics output for Scotland (which is provided by NRS) but a descriptive account of a cohort of deaths where further information was available.

Data on all deaths recorded in 2012 were obtained from the Scottish NDRDD, entered into a secure database, anonymised and analysed descriptively using SPSS v21. The Scottish NDRDD collects a wide-range of variables relating to the nature and circumstances of individuals who have died a DRD including their sociodemographics, drug use history, toxicology, known service contact and medical and psychiatric details (Hecht, Barnsdale, & McAuley, 2014). The overall sample was divided into two cohorts; those with NPS recorded in toxicology (36) (i.e. 'NPS-related') and those where NPS was deemed by the reporting pathologist to be implicated in the cause of death (23) (i.e. NPS-implicated). Six different NPS types were recorded in toxicology across 36 deaths (benzodiazeoines, tryptamines, phenethylamines, piperazines, cathinones arylalkylamines) which largely fell into two main pharmacological groups; 'Benzodiazepine-type' or 'Stimulant-type' NPS.

This analysis briefly compares the toxicology of both cohorts before focussing in detail on the NPS-implicated cases, investigating the characteristics of the individuals involved and the circumstances surrounding their deaths. The decision to focus on the 23 NPS-implicated rather than all 36 NPS-related was taken on the basis that this group are the most likely to reflect actual cases where NPS has played a part in an individual deaths, as defined by the reporting pathologist.

In addition to this descriptive analysis, exploratory analyses of differences in findings between the two pharmacological sub-groups (i.e. 'Benzodiazepine-type' and 'Stimulant-type') NPS were conducted using Fisher's exact tests (Fisher, 1954).

Results

Toxicology

Table 1 details the drugs recorded and implicated in NPS deaths. The majority of NPS-related DRDs involved Benzodiazepine-type drugs (24), mainly Phenazepam (23). The other 12 NPS-related deaths featured a range of different Stimulant-type drugs. A similar breakdown is evident in the NPS-implicated cases where 12 involved Benzodiazepine-type drugs and 10 featured Stimulant-type drugs. In six of these deaths, multiple NPS drugs were found to be present.

[insert table 1]

Table 2 shows details from the toxicology reports, reporting both the drugs present and implicated in the deaths, overall and by drug type. Polydrug use was a key feature of the 36 NPS-related DRDs with presence of more than one drug recorded

by toxicology in every case (data not shown). The drugs most likely to be present in the body at post mortem alongside NPS were diazepam (20), methadone (14), alcohol (14), antidepressants (11), dihydrocodeine (9) and heroin/morphine (8). Both NPS drug type groups had co-presence of a wide range of other drugs, however, Benzodiazepine-type cases were more likely to also feature methadone (13/24) (p=0.01) while cocaine (5/12) and ecstasy/MDMA (3/12) were more likely to be recorded within the Stimulant-type group (p<0.01).

The extent to which certain drugs were reported as being implicated in each death, as opposed to being just present within the body at toxicology, revealed a slightly different picture. In total, just over two-thirds of NPS recorded as present in the body at post mortem were considered by the reporting pathologist to be implicated in the death (23/33). Again, both NPS drug type groups had co-implication of a wide range of other drugs, however, Benzodiazepine-type cases were more likely to be implicated alongside methadone (8/13) (p<0.01) and had no stimulant drugs co-implicated. In contrast cocaine (5/10) was more likely to be implicated within the Stimulant-type group (5/10) (p<0.01).

[insert table 2]

In addition, table 3 details the combinations of drugs in each NPS- implicated death. Stimulant type-cases (6/10) were more likely than Benzodiazepine-type cases (0/13) to have more than one NPS implicated (p<0.01) (data not shown) with all cases consuming two or more Stimulant-type drugs in combination.

[insert table 3]

Sociodemographics

Table 4 shows the sociodemographics of the individuals involved in the NPS-implicated deaths, overall and by type. The majority of the deceased were male (19/23) and accounted for most cases in each age-group. Deaths due to NPS were evident across all age groups [data not shown] with half in those aged 35 years and over. Benzodiazepine-type deaths were more likely to be aged over 35 years (p=0.04), whereas there were no Stimulant-type deaths in those aged over 45 (data not shown). Although NPS-implicated deaths were recorded in all deprivation quintiles, just over half (12) were from the most deprived area (SIMD quintile 1 (12).

[insert table 4]

Drug use history

The overwhelming majority of NPS-implicated DRDs (20/23) were among individuals known to have used drugs prior to their death, around half of these (8) for a considerable period of time (i.e. 11 years or more). All of these long-term users were in the deaths observed among the Benzodiazepine-type group (p<0.01).

A fifth (5/23) of cases were known to be on an opiate replacement therapy [methadone] prescription at the time of death, all via supervised consumption (data not shown). Again, all of those on methadone were observed among the Benzodiazepine-type group (p=0.04).

Medical and psychiatric history, and significant life events

Almost two-thirds (15/23) of NPS-implicated DRDs had a medical condition recorded in the six months prior to death, with two individuals experiencing more than one condition. A range of conditions were noted, the most common being psychiatric conditions (7), problem alcohol use (7) and respiratory conditions (5), .

A quarter (6/23) of individuals were known to have overdosed previously, although half of these only once or twice (3/6) and only one within the three months prior to their death (data not shown).

Contact with services

Twelve of 20 individuals who suffered an NPS-implicated death were known to drug services, eight of whom had been in contact in the six months prior to death. Only one case was known to have had a recent detox.

Seven of the deceased had been in police custody at some point in their lives, all within the six months prior to their death. Indeed, two had been in police custody in the week prior to their deaths. Similarly, 11 of the 23 individuals had been in prison at some point in their lives, with three being released within six months of their death (two of whom had been released in the fortnight prior to their deaths). Those individuals in the Benzodiazepine-type group were more likely to have spent time in prison (9/13) compared with those in the Stimulant-type group (2/10; p=0.04).

Death circumstances

Nine individuals were pronounced dead in their own home and seven were pronounced dead at hospital (including Accident & Emergency departments). There were persons present for almost three-quarters of the deaths (16/22). Of the 16 who were present at the time of death, over half (9) were known to be in the same room at the time of the fatal overdose (data not shown).

Ambulance attendance was recorded at the scene of most deaths (19/23), however resuscitation was attempted in just under half of cases (11), most of which were carried out by the ambulance staff themselves (6, data not shown). The remaining cases attended by the ambulance service were deemed to be 'beyond resuscitation'.

The majority of cases were classed as 'accidental poisoning' (19/23) with the remainder classed as an 'event of undetermined intent, poisoning'; no suicides were recorded within the cohort.

Discussion

This analysis provides the one of the first detailed accounts of the circumstances surrounding deaths due to NPS, albeit based on a very small number of deaths upon which only tentative conclusions can be drawn. Despite recent increases in NPS-related mortality (Corkery, Claridge, Loi, Goodair, & Schifano, 2014; National Records of Scotland, 2014), the circumstances surrounding and characteristics of individuals involved in NPS deaths at a population level remain relatively unknown, in particular cases where NPS was deemed to implicated in the cause of death.

In 2012, 36 DRDs had NPS recorded within post-mortem toxicology which accounted for 8% of the total (479) DRDs registered on the Scottish NDRDD that year (Hecht, Barnsdale, & McAuley, 2014). However only 23 of these NPS drugs were considered by the reporting pathologist to be implicated in the actual cause of death. The NPS drugs recorded and implicated in the deaths could be pragmatically grouped into two categories: Benzodiazepine-type or Stimulant-type NPS. Official statistics on DRDs in England & Wales reported an increase in NPS recorded in death certificates from 9 in 2007 to 52 in 2012 (Office for National Statistics, 2013). The 2012 data for England and Wales were made up of deaths mainly related to Stimulant-type NPS drugs (cathinones (18/52), GHB/GBL (13/52) and piperazines (9/52)); no other NPS drugs were reported. Using a different definition of NPS, the UK National Programme on Substance Abuse Deaths reported an increase in NPS implicated in DRDs from 10 in 2009 to 68 in 2012 (Corkery, Claridge, Loi, Goodair, & Schifano, 2014). Again, most of these deaths in 2012 were related to Stimulant-type NPS drugs (cathinones (24/68) and 'amphetamine-type substances' (23/68)).

The NDRDD in Scotland is unique in providing detailed descriptions of the demographics of each individual involved in DRD and the context surrounding each case. However, with only 23 deaths in the NPS-implicated cohort, just one year of data for analysis, and lack of a sufficient control population to compare against, caution should be taken when interpreting these initial results and applying the findings more widely. This caveat is particularly salient for the comparative analysis between NPS-implicated cases involving Benzodiazepine-type and Stimulant-type NPS which should be used cautiously to inform future analysis in this field. Given the

exploratory nature of this research, no attempt at multiple comparisons correction was made.

Benzodiazepine-type NPS-implicated deaths

The Benzodiazepine-type NPS-implicated deaths were predominantly in males, often in those aged over 35 years old, and known to have used drugs for a long time. They were often known to drug treatment services but not always in receipt of opiate replacement therapy, and were likely to have previous experience of spending time in prison at some point in their lives. Generally there was polydrug consumption of opioids with benzodiazepines by individuals in this group.

Interestingly, the 13 individuals whose death involved Benzodiazepine-type NPS shared many similar characteristics to the mainly opioid-related DRD cohorts in Scotland and elsewhere that has emerged in recent years (Hecht, Barnsdale, & McAuley, 2014; Darke, 2014). These findings are important in that they potentially identify existing or past clients of traditional drug treatment services; therefore there is an urgent need for such services to recognise the broad range of NPS drugs and their impact and to manage them accordingly within care plans.

The association between benzodiazepines and DRDs is well established (Bird & Robertson, 2011; Darke, 2014), but not yet fully understood and should be a priority for future research. The most prevalent NPS recorded and implicated in 2012 DRDs by the Scottish NDRDD was Phenazepam, a long-acting benzodiazepine developed in the former Soviet Union in the 1970s (Corkery, Schifano, & Ghodse, 2012b). Although not licensed for use in the UK, it has recently emerged as a drug of

concern due to increasing use and related harm reported in Western Europe and the USA (Maskell, De Paoli, Seetohul, & Pounder, 2012), with the first seizures in the UK recorded in Scotland in 2008 (Corkery, Schifano, & Ghodse, 2012b). Hospital admissions related to Phenazepam followed soon after prompting the Scottish Government to issue a warning in 2010 about its availability and risks associated with consumption ('Warning over online Valium', 2010).

The first deaths in the UK where Phenazepam was implicated were recorded in 2011 (Maskell, De Paoli, Seetohul, & Pounder, 2011). Indeed the UK National Programme on Substance Abuse Deaths reported Phenazepam presence within toxicology in 14 deaths in both 2012 and 2013, but implicated in only 9 and 5 of those cases respectively (Corkery, Claridge, Loi, Goodair, & Schifano, 2014). Phenazepam became controlled as a Class C drug in the UK in June 2012. Phenazepam presents additional cause for concern given its high levels of toxicity; it is reported to be particularly effective at 0.5-1.0mg, or 10% of a standard 10mg dose for diazepam (Kyle, Brown, Bailey, & Stevenson, 2012). The extent to which Phenazepam is being used as a substitute or supplemental benzodiazepine, or perhaps being marketed as diazepam and being taken unwittingly, is currently unknown. Indications from Phenazepam seizures suggest that the latter theory is the most plausible with markings suggestive of diazepam-like features (Corkery, Schifano, & Ghodse, 2012b). Future study, including qualitative enquiry, should aim to explore such hypotheses in more detail. The impact of Phenazepam's change of legal status should also be considered. Indeed it will be important to determine responses by both sellers and consumers since the Phenazepam ban came into force and any associated impacts on harm. Death data from NRS suggests that there has been no

immediate impact of the ban, at least in relation to mortality, with Phenazepam implicated in, or potentially contributing to, the cause of death in 34 deaths in 2013, an increase from 13 deaths in 2011 and 19 deaths in 2012 respectively (National Records of Scotland, 2014).

Stimulant-type NPS-implicated deaths

The deaths where Stimulant-type NPS were implicated shared some similarities with the Benzodiazepine-type group detailed above, but the data were suggestive of some differences in terms of the individuals involved and the circumstances of their deaths which merit further exploration in future reports. They appeared to be a younger group, and were unlikely to be known to have used drugs on a long-term basis, partly due to their younger age. Although some were known to drug treatment services, none were in receipt of opiate replacement therapy. Equally, they were less likely to have been in prison during their lives, again possibly due to their younger age. Like the Benzodiazepine-type cases, polydrug use was common, often in the company of others, typically combinations of stimulant type drugs including other NPS. Indeed, these ten Stimulant-type cases more closely mirrored the risk factor profile described in analysis of a Mephedrone-related death cohort by Corkery and colleagues (2013).

Despite their small numbers, deaths involving Stimulant-type NPS have attracted particular media attention, possibly due to the younger age of the individuals involved (Scottish Government, 2011; European Commission, 2014) or as a result of a specific symbolic framework used by the media to represent problems associated with such drugs (Manning, 2006). Indeed, the additional focus of the press on

alleged mephedrone-related fatalities in 2010 appeared to have the unintended consequence of actually increasing interest in purchasing the drug (Forsyth, 2011). Future NPS research should consider the role of such external influences in either mitigating or facilitating future harm.

Although trends in NPS-related mortality appears to be increasing (Corkery, Claridge, Loi, Goodair, & Schifano, 2014; National Records of Scotland, 2014), accurately determining the scale of their impact on harm at a population level to inform policy and practice is challenging. The social construction of NPS and their related harms is directly influenced by which substances are defined within that term, some of which are neither new nor psychoactive (King and Nutt, 2014). Given the variations in definitions across countries this has the potential to over or under estimate the impact depending on how broad the NPS definition is. For example, had Scotland adopted the UN categorisations outlined in the introduction of this paper, then Phenazepam would not be included as an NPS and the scale of reported mortality would be much reduced. Furthermore it is also likely that some NPS won't be tested for unless circumstantial evidence or witness testimony of consumption is forthcoming at time of death. Such omissions also give scope for potential under-reporting.

In addition to inconsistencies surrounding definition and reporting, the subjectivity of whether drugs are recorded or implicated in the actual cause of death merits consideration. As noted earlier in this paper, this categorisation is largely determined by the reporting pathologist or by NRS in the absence of such information. Should a large number of pathology reports on implication be missing then the robustness of

the implicated figures could be challenged. However, in 2012, pathologists provided NRS with information (in time) for 31 out of the 32 for which NPSs were implicated, and all of the 15 deaths (in 2012) for which NPSs were present but not implicated (Frank Dixon, personal communication). Such completeness with regard to pathology reporting provides a degree of reassurance as to the robustness of the implicated figures. The consistency of how different pathologists (or labs) interpret drugs as recorded or implicated is largely unknown, however, and certainly merits further investigation.

Conclusion

Given the limited evidence base on mortality where NPS is involved more widely, this exploratory study provides an important addition to the literature and offers scope for further research. Although based on a small population-based sample, it suggests some important issues for policy and practice, not least the prominent role of unlicensed benzodiazepines in drug-related mortality, but also the need for different harm reduction strategies to target two apparently distinct groups; one which seems to mirror the established ageing cohort of DRDs, the other more closely aligned to the recent reported increases in recreational use of Stimulant-type NPS by younger people. Future analysis will be important in determining the extent of NPS involvement in DRDs and the circumstances surrounding each death to inform policy and public health interventions. This is of particular importance given the increasing number and variations of NPS drugs appearing on the market.

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Table 1: Substances recorded/implicated in NPS-related DRDs, Scotland, 2012

a) Benzodiazepine-type deaths (Recorded base: n=24; Implicated base: n=13)

Substance name	Recorded	Implicated
Phenazepam	23	12
Etizolam	1	1

b) Stimulant-type deaths (Recorded base: n=12; Implicated base: n=10)[1]

Substance name (chemical group)	Recorded	Implicated
AMT (Tryptamine)	5	5
6-APB (Phenethylamines)	3	2
BZP (Piperazines)	3	2
API (Phenethylamines)	2	2
Mephedrone (Cathinones)	2	2
MPA (Arylalkylamine)	1	2
Methylethcathinone (Cathinones)	1	1
TFMPP (Piperazines)	1	1
5-MeO-DALT ((Tryptamine)	1	1

[1] Number exceeds the base total as six of the deaths had more than one NPS recorded at toxicology.

Table 2: Selected drugs recorded/implicated by toxicology in NPS-related deaths, by drug type, 2012

	Number of deaths with drugs present				Number of deaths with drugs implicated			
	All deaths n/total (%)	**Benzodiazepine-type n/total (%)**	**Stimulant-type n/total (%)**	***p*-value[a]**	**All deaths n/total (%)[a]**	**Benzodiazepine-type n/total (%)**	**Stimulant-type n/total (%)**	***p*-value[a]**
Drugs recorded								
Alcohol	14/36	11/24	3/12	0.29	3/23	3/13	0/10	0.23
Anti-Depressants	11/36	7/24	4/12	0.99	0/23	0/13	0/10	0.99
Cocaine	7/36	2/24	5/12	0.03*	5/23	0/13	5/10	<0.01*
Diazepam	20/36	15/24	5/12	0.30	3/23	2/13	1/10	0.99
Dihydrocodeine	9/36	7/24	2/12	0.69	2/23	2/13	0/10	0.49
Ecstasy/MDMA	3/36	0/24	3/12	0.03*	2/23	0/13	2/10	0.18
Heroin/Morphine	8/36	5/24	3/12	0.99	4/23	2/13	2/10	0.99
Methadone	14/36	13/24	1/12	0.01*	8/23	8/13	0/10	<0.01*
NPS	36/36 (100.0)	24/24 (100.0)	12/12 (100.0)	n/a	23/23 (100.0)	13/13 (100.0)	10/10 (100.0)	n/a

[a] Fishers exact test
*denotes significant variables

Table 3: Combinations of drugs in NPS-implicated deaths, 2012

Case No.	Drugs Implicated[a]				
Benzodiazepine-type					
1.	**PHENAZEPAM**				
2.	**PHENAZEPAM**	METHADONE			
3.	**PHENAZEPAM**	METHADONE			
4.	**PHENAZEPAM**	METHADONE			
5.	**PHENAZEPAM**	METHADONE			
6.	**PHENAZEPAM**	METHADONE	HEROIN		
7.	**PHENAZEPAM**	METHADONE	CODEINE		
8.	**PHENAZEPAM**	METHADONE	DIHYDROCODEINE	DIAZEPAM	
9.	**PHENAZEPAM**	METHADONE	HEROIN	GABAPENTIN	
10	**PHENAZEPAM**	DIAZEPAM	ALCOHOL		
11.	**PHENAZEPAM**	BUPRENORPHINE	ALCOHOL		
12.	**PHENAZEPAM**	BUPRENORPHINE	ALCOHOL		
13	**ETIZOLAM**	DIHYDROCODEINE	TRAMADOL		
Stimulant-Type					
14.	**MEPHEDRONE**	COCAINE			
15.	**MEPHEDRONE**	MDMA	AMPHETAMINE	**METHYLETHCATHINONE**	**MPA**
16.	**AMT**	**BZP**	DIAZEPAM	TEMAZEPAM	MORPHINE
17.	**AMT**	**API**	**5-MEO-DALT**	KETAMINE	
18.	**AMT**	COCAINE			
19.	**AMT**	COCAINE			
20.	**AMT**	COCAINE			
21.	**APB**	**MPA**	COCAINE		
22.	**APB**	**API**	ECSTASY		
23.	**BZP**	**TFMPP**	HEROIN	BENZODIAZEPINE	

[a] NPS drugs in **bold** text; drugs listed in no particular order

Table 4: Characteristics of individuals involved and circumstances surrounding each NPS-implicated death, by drug type, 2012

Base = 23 NPS implicated cases	NPS-implicated DRDs n/total[a]	Benzodiazepine-Type n/total	Stimulant-Type n/total	*p*-value[b]
Sociodemographics				
Male	19/23	10/13	9/10	0.60
Aged >35 years	11/23	9/13	2/10	0.04*
Resident in most deprived quintile (q1)	12/23	9/13	3/10	0.10
Lived alone	11/22	7/12	4/10	0.67
Had children	7/23	3/13	4/10	0.65
Drug Use History				
Known drug user	20/23	12/13	8/10	0.56
- Known drug user >11years		8/10	0/6	<0.01*
Known injector	9/20	7/12	2/8	0.20
- Known injector >11years		3/6	0/2	0.46
Recent detox	1/20	1/12	0/8	0.99
Methadone prescription	5/23	5/13	0/10	0.05*
Medical, Psychiatric History and Significant Life Events				
Known medical conditions in the six months prior to death	15/23	9/13	6/10	0.69
Significant life event in the six months prior to death	12/23	6/13	6/10	0.68
Previous overdose at any time	6/23	5/13	1/10	0.18
Contact with services				
Drug treatment service contact	12/20	9/12	3/8	0.17
- Drug treatment service contact 6 months prior to death		6/9	2/3	0.99
Police custody contact	7/20	4/11	3/9	0.99
- Police custody contact in the 6 months prior to death		4/4	3/3	0.99
Prison contact	11/23	9/13	2/10	0.04*
- Prison contact in the 6 months prior to death		1/9	2/2	0.20

Death circumstances				
Used drugs in own home	11/19	6/11	5/8	0.99
Pronounced dead own home	9/23	7/13	2/10	0.20
Pronounced dead at hospital	7/23	2/13	5/10	0.17
Persons present	16/22	7/12	9/10	0.16
Resuscitation attempted	11/23	5/13	6/10	0.41
Ambulance attended	19/23	10/13	9/10	0.60
Cause of death (ICD10 code)				
Accidental poisoning (X40 – X44)	19/23	11/13	8/10	0.99
Undetermined intent, poisoning (Y10 – Y14)	4/23	2/13	2/10	0.99

[a] Not all variables total 23 cases due to missing data, [b] Fishers exact test
*denotes significant variables