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Thematic Analysis of Medical Examiner Narratives to Understand the Socio-Spatial Context, Recency of Drug Use, and Likely Mechanism of Stimulant Toxicity Deaths

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Abstract

Background: Drug toxicity as a cause of death is challenging to establish and may be based on limited evidence, especially in deaths attributed to stimulants. We developed a method for characterizing stimulant deaths, focusing on potential mechanisms and opportunities for intervention.

Methods: We used medical examiner case narratives and medical records from a mixed methods study of fatal acute stimulant toxicity in San Francisco. We coded case narratives for circumstances surrounding death events, including physical location, bystander presence, decedent disposition, and evidence of recent street drug use; medical records provided data on potential mechanism of death when not present in case narratives.

Results: Of 101 deaths (70 stimulants-no-opioids, 31 stimulants-fentanyl), 85 were unwitnessed, including 69 unwitnessed deaths in spaces inaccessible to bystanders. Drug use was observed before collapse in 1 of 14 witnessed stimulant-no-opioid and 1 of 2 witnessed stimulant-fentanyl deaths. Among unwitnessed events, scene evidence of drug use was found in 36 of 56 stimulant-no-opioid and 25 of 29 stimulant-fentanyl deaths. Twelve of 14 witnessed stimulant-no-opioid deaths and none of two witnessed stimulant-fentanyl deaths included an apparent cardiovascular or cerebrovascular event.

Conclusions: Deaths occurred in physically and socially isolated contexts, limiting opportunities for bystander intervention. Compared to stimulant-fentanyl deaths, stimulant-no-

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opioid deaths may be more likely to be witnessed and involve a cardiovascular event, and less likely to involve recent drug use. Applying a thematic analysis of medical examiner records to a larger sample, including other opioid deaths, could guide prevention strategies.

Keywords

overdose; stimulants; medical examiner; coroner; methamphetamine; cocaine; toxicity

1. Introduction

Age-adjusted mortality attributed to acute methamphetamine or cocaine toxicity rose 14-fold in the U.S. from 2011–2021 (Spencer et al., 2023). While deaths involving stimulants without opioids increased (Spencer et al., 2023), most of the increase also involved opioids (Jones et al., 2020; Ciccarone, 2021; Hoopsick et al., 2023), including 61% of stimulant “overdoses” in 2021 (Hoopsick et al., 2023).

While the mechanism of opioid overdose death is well described, including rapid onset of respiratory depression after opioid use, death from acute stimulant toxicity is not clearly defined. Taking too large a dose of a stimulant is often called “overamping”; in contrast to opioid overdoses, these events vary in presentation and severity but are rarely fatal (Harding et al., 2022; Mansoor et al., 2022). Compared to fatal opioid overdoses (Stockings et al., 2019; Riley et al., 2022), deaths attributed to stimulants tend to occur in older people with more comorbidities and often have cardiovascular or cerebrovascular events listed as contributing causes (Turner et al., 2018; Stockings et al., 2019; Riley et al., 2022), suggesting many of these deaths may be sequelae of chronic diseases (Turner et al., 2018; Riley et al., 2022) and terms such as “overdose” or even “acute toxicity” may be misleading.

Cause of death determination is completed for legal purposes and the process can vary between jurisdictions (Slavova et al., 2015; Hochstatter et al., 2022). Drug toxicity as a cause of death is primarily based on toxicology results and scene examination with varying and often limited contribution from autopsy and medical records (Cina et al., 2011). Distinguishing drug toxicity from other causes of death may be impossible in some cases, particularly when decedents have multiple comorbidities (Merlin et al., 2022), and overdose is often a “diagnosis of exclusion” (Merlin et al., 2022). Moreover, these data provide limited insight into the dose or context of drug use.

As health professionals seek to reduce mortality from stimulant use, labeling these deaths “overdoses” may distract from the role of chronic disease and poverty and lead away from effective interventions. We developed a new method for characterizing deaths attributed to acute stimulant toxicity through review of medical examiner case narratives and medical records, with a focus on the socio-spatial context of the deaths, evidence of recent street drug use, and likely mechanism of death. We applied our method to 101 stimulant-associated deaths from a mixed methods study of stimulant deaths (Antolin Muñoz et al., 2025) and used the results to generate hypotheses for further investigation.

2. Methods

2.1 Sample and data collection

We used data from a parent study of unintentional deaths attributed to acute cocaine or methamphetamine toxicity, which combined informant interviews with review of medical examiner case narratives and medical records to explore factors associated with deaths attributed to acute stimulant toxicity. The parent study included a non-random sample of decedents identified through record review of the California Electronic Death Registry System (CA-EDRS) and the San Francisco Office of the Chief Medical Examiner (OCME) (Antolin Muñoz et al., 2025). The sample intentionally included 70 deaths involving stimulants without opioids (“stimulant-no-opioid”) and 31 deaths involving stimulants and fentanyl (“stimulant-fentanyl”) that occurred from June 2022 through December 2023. The parent study sought an even split between deaths attributed to cocaine and to methamphetamine: stimulant-no-opioid cases included 35 deaths attributed to cocaine, 32 to methamphetamine, and three to both; stimulant-fentanyl cases included 12 deaths attributed to cocaine, 15 to methamphetamine, and four to both.

The study was approved by the University of California San Francisco, Human Research Protection Program (#21-35305) and the State of California Health and Human Services Agency Committee for the Protection of Human Subjects (#2022-091).

2.2 Socio-spatial characteristics

Using the framework of reflexive thematic analysis (Braun and Clarke, 2006), case narratives were reviewed and coded for themes related to the social and physical contexts of death. Coding was performed by the study RN and physician (authors FB and POC), both of whom have training in qualitative analysis, with both blinded to which drugs the deaths were attributed. Coding was carried out iteratively until a set of stable thematic codes emerged, with coders meeting to discuss codes until consensus was reached. These codes were used to create five measures (Table 1) which were reviewed and discussed with co-authors, refined, and arranged in a nested categorization system (Figure 1).

2.3 Recent use of street drugs

Evidence of recent street drug use was analyzed separately for witnessed and unwitnessed deaths. For unwitnessed deaths, evidence was coded as present if the case narrative indicated possible street drugs (e.g. white powder, crystal substance) or associated equipment (e.g. glass pipes, syringes) found at the scene of death, excluding alcohol, tobacco, and cannabis products. For witnessed deaths, evidence was coded as present if the case narrative indicated that the decedent was seen using opioids, stimulants, or unspecified drugs in the period immediately prior to the medical emergency resulting in death (approximately one hour).

2.4 Mechanism of death

Although all deaths in the sample had acute stimulant toxicity listed as a cause of death, we sought to determine the likely physiological mechanism of the event resulting in death. We could only assess this if there was information about the nature of the event, limiting analysis to witnessed deaths. Case narratives and medical records were reviewed

by the study team physician (POC) to determine the likely mechanism of death, coded as cardiovascular, cerebrovascular, respiratory, or uncertain/other. If a clear mechanism was not described in the case narrative and medical records, we coded the event as follows: cardiac if the decedent experienced a sudden collapse after vigorous activity or signs of cardiovascular distress (e.g. clutching chest); respiratory if the decedent was described as having ceased breathing after using drugs without signs of cardiovascular distress; and uncertain in all other cases.

2.5 Analysis

We calculated frequencies and proportions of sociodemographic and socio-spatial characteristics of death, evidence of substance use, and mechanism of death. For age, which was continuous and skewed, we calculated a median and interquartile range (IQR). We presented results overall and separately for stimulant-no-opioid and stimulant-fentanyl deaths.

3. Results

3.1 Demographics

The median age of decedents was 58 (IQR: 47–61); 44% were White and 36% Black; 69% were unhoused or marginally housed (Table 2). Most decedents (75%) were classified as male.

3.2 Sociospatial characteristics of deaths

Of 101 deaths, 85 were unwitnessed, including 80% (56) of stimulant-no-opioid and 94% (29) of stimulant-fentanyl deaths; 68% of all deaths occurred unwitnessed in private spaces (Figure 2) and none of these decedents were resuscitated on scene or found by bystanders (Figure 3). In over half (52%) of all deaths, the decedent was found dead beyond resuscitative efforts in a private space after not being seen for more than 12 hours.

3.3 Evidence of drug use prior to death

Information on scene evidence of drug use was available for 83 of the 85 unwitnessed deaths. Of these cases, evidence was found for 36 (65%) of 55 stimulant-no-opioid deaths and 25 (89%) of 28 stimulant-fentanyl deaths.

Of the two witnessed stimulant-fentanyl deaths, one occurred immediately after the decedent injected a brown substance and stopped breathing. The other had minimal information about the scene of the event; the patient was transported to the hospital and died after a three-month intensive care unit stay. Among the 14 witnessed stimulant-no-opioid deaths, 13 (93%) did not involve drug use leading up to the event (e.g., decedent collapsed while grocery shopping or during an argument); in one case the decedent smoked from a glass pipe immediately before collapsing.

3.4 Mechanism of death

For the 16 witnessed deaths, 10 of the collapses (all stimulant-no-opioid) were likely cardiac, two (both stimulant-no-opioid) were cerebrovascular accidents, one (stimulant-

fentanyl) was likely respiratory, and three (two stimulant-no-opioid, one stimulant-fentanyl) lacked sufficient detail for determination.

4. Discussion

Through iterative analysis of medical examiner case narratives and medical records, we developed a system for characterizing deaths attributed to acute stimulant toxicity, applied this to a small sample of deaths, and developed three hypotheses for further investigation. First, most deaths attributed to acute stimulant toxicity occur in physically and socially isolated contexts where bystander response (e.g. with CPR or hypothetical reversal agents) would not be available, likely limiting the utility of such interventions. Second, there was limited evidence of recent street drug use among stimulant-no-opioid deaths, suggesting that many of these may not be driven by acute toxicity, but instead be the result of chronic disease contributed to by substance use. Third, stimulant-fentanyl deaths were generally unwitnessed and had more evidence suggesting recent drug use, and thus may be primarily attributable to fentanyl.

The deaths in our sample were characterized by marked social and physical isolation: 69 of 101 deaths occurred in private spaces without witnesses. In 53 of 101 deaths, over 12 hours had elapsed since the decedent was last seen; 33 of these decedents were found by professionals such as building staff or case managers rather than by family or friends. A typical case report for these deaths would be a body found decomposing in a public housing unit during a wellness check after the person had not been seen for several days. Even if the mechanism of death could be addressed by bystanders, the majority of these deaths had no opportunity for bystander intervention.

Two of 31 (6%) stimulant-fentanyl deaths were witnessed, compared to 14 of 71 (20%) stimulant-no-opioid deaths. Given that witnessed opioid overdoses should have a mortality rate under 1% (McDonald and Strang, 2016), and possibly lower in a city with ready access to overdose prevention services, the low rate of stimulant-fentanyl deaths being witnessed is not surprising if those events resulted from opioid-induced respiratory depression. In contrast, out-of-hospital cardiac arrest, a likely etiology for stimulant-no-opioid deaths (Turner et al., 2018; Riley et al., 2022), has a mortality rate approaching 90% (Tsao et al., 2022). The presence of witnesses would thus be less protective against death and could account for the finding of more witnessed stimulant-no-opioid deaths. These findings suggest that stimulant-fentanyl deaths should be considered as different from stimulant-no-opioid deaths.

If the deaths we studied were due to a common understanding of the term “overdose”, we would expect to find evidence of drug use shortly prior to the event resulting in death. While the one witnessed stimulant-fentanyl death with details about the event had such evidence, only 1 out of 14 witnessed stimulant-no-opioid deaths involved a report of witnessed drug use shortly preceding collapse. For the 83 unwitnessed deaths where information on scene drug evidence was available, evidence was more often present for stimulant-fentanyl (89%) than stimulant-no-opioid deaths (65%), suggesting that some stimulant-no-opioid deaths may not have been immediately preceded by drug use. Toxicology results imply some

recency of use but remain positive for hours for cocaine and days for methamphetamine. If deaths attributed to acute stimulant toxicity were not immediately preceded by stimulant use, hypothetical stimulant reversal agents (analogous to naloxone) would have no role in prevention. Instead, preventing stimulant-no-opioid deaths would demand attention to prevention and management of chronic cardiovascular and related diseases (Riley et al., 2022).

This study has several limitations. First, our work was exploratory with a small sample size and should be considered hypothesis-generating. Second, our sample was drawn from a parent study in which each decedent had at least one informant who was successfully interviewed, implying that the marked social isolation we observed may be an underestimate. Finally, our coding of recency of street drug use relied upon witness reports and scene evidence. Witness reports were likely specific for recent use, but may have been insensitive as decedents may have used drugs out of view; scene evidence may have been sensitive for recent street drug use, but less specific as the presence of a drug or associated supplies would not necessarily imply recent use. Nonetheless, these data were readily available from case narratives, and the consistent finding in both sets of data suggests that these proxies were informative.

5. Conclusion

We developed an approach to analyzing medical examiner case narratives that provides insight into the nature of deaths attributed to acute stimulant toxicity and the viability of interventions. In our pilot sample, such deaths often occurred alone in isolated settings, often without evidence of recent drug use, and in many cases may more appropriately be considered sequelae of chronic disease. In contrast to deaths involving opioids, which may be prevented by bystander overdose response strategies, preventing stimulant-no-opioid deaths may require upstream interventions to address chronic disease, poverty, and social isolation. Application of this approach to a larger dataset, including other opioid deaths, would be valuable to inform public health interventions targeting acute drug toxicity.

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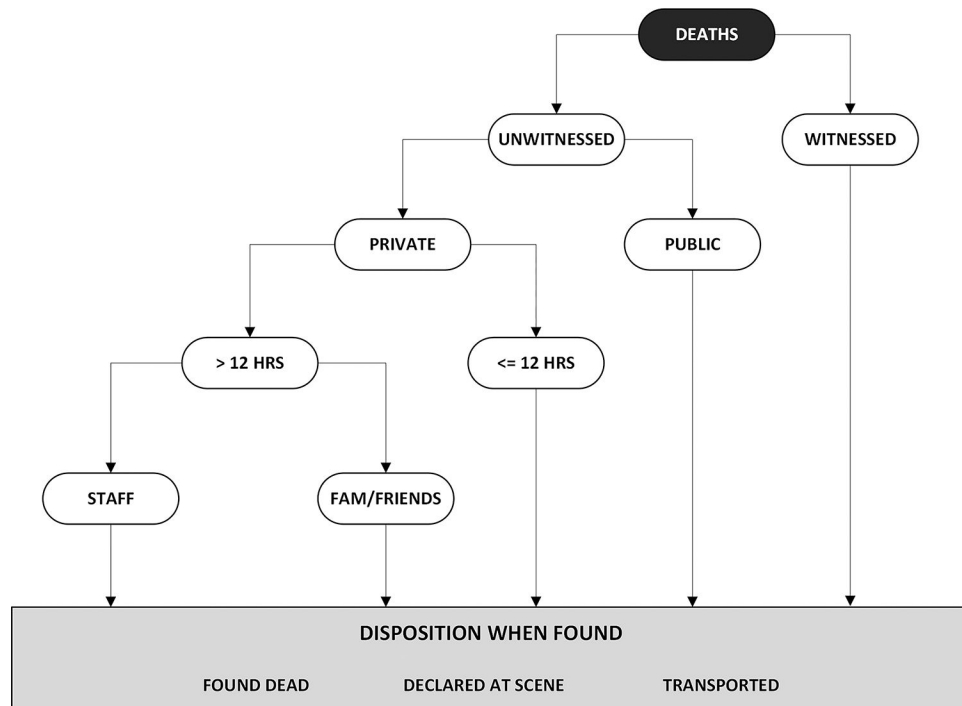


Figure 1: Nested death categorization system

All deaths were characterized by disposition of decedent when found; unwitnessed deaths were additionally categorized by public vs. private location of death. Deaths occurring in private were coded by time elapsed since decedent was last seen alive; decedents not seen within 12 hours were also coded by who found the body.

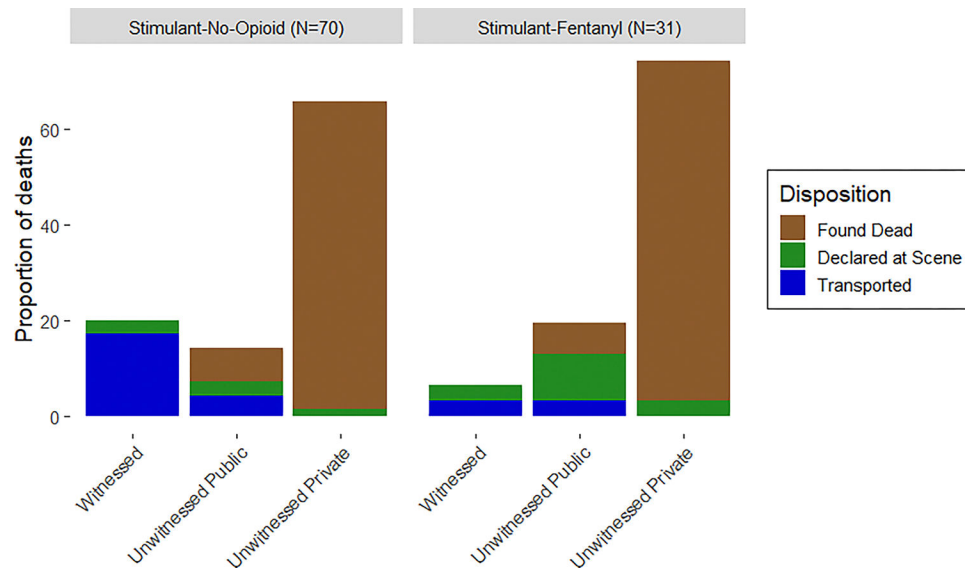


Figure 2: Spatial contexts of deaths attributed to stimulants among decedents in a psychological autopsy study by fentanyl involvement (N=101).

Witnessed deaths include events that occurred in public and private spaces; unwitnessed deaths are shown broken down by public vs. private.

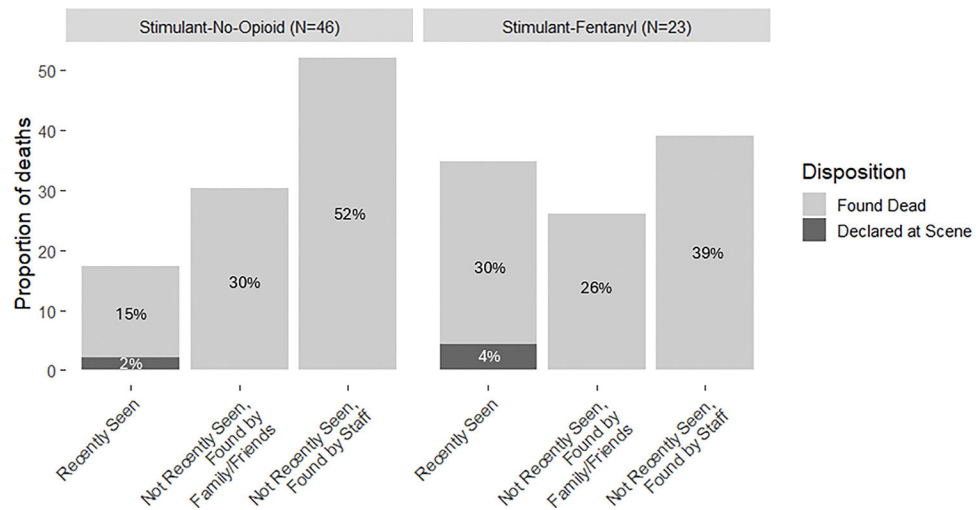


Figure 3: Social contexts of deaths attributed to stimulants among decedents in a psychological autopsy study by fentanyl involvement (N=69).
 Recently seen decedents were seen within 12 hours of death; decedents not recently seen are shown broken down by who found the body.

Table 1:

Socio-spatial death characterization codes

Variable	Levels
Presence of witnesses	Witnessed: event (e.g., collapse, loss of consciousness, physical distress) was observed in real time by other people
	Unwitnessed: event was not seen in real time (e.g., no evidence of potential witnesses present, or potential witnesses did not observe event)
Location of death	Public: visible and accessible to passersby (e.g., street, lobby, parked car)
	Private: not visible and not accessible to passersby (e.g., residence, hotel room)
Time since decedent was seen prior to event ^a	≤12 hours
	>12 hours
Person who discovered decedent	Family/friend: person with a personal relationship with the decedent (e.g. roommate, partner, family member)
	Staff: person acting in a professional role (e.g. healthcare worker, housing staff)
	Bystander: person with no relationship to the decedent
Disposition of decedent when found	Found dead: decedent was clearly beyond resuscitative efforts when found
	Declared at scene: resuscitation was attempted but ceased and death was declared at scene
	Transported: transportation was initiated before death was declared (e.g., declared in ambulance, emergency department, or hospital inpatient unit)

^a 12 hours was chosen for cutoff as decedents seen within 12 hours were generally using drugs within a social context while decedents who had not been seen for greater than 12 hours generally had not been seen for multiple days.

Table 2:
Sociodemographic Characteristics of Decedents from Acute Cocaine or Stimulant Toxicity in San Francisco,
June 2022-December 2023 (N=101) % (n)

	All deaths (n=101)	Stimulant-no-opioid (n = 70)	Stimulant-Fentanyl (n = 31)
Age	58 (median), IQR 47–61	58 (median), IQR 52–61	50 (median), IQR 43–59
Sex^a			
Female	25 (25%)	16 (23%)	9 (29%)
Male	76 (75%)	54 (77%)	22 (71%)
Race/ethnicity			
Black	36 (36%)	29 (41%)	7 (23%)
White	44 (44%)	28 (40%)	16 (52%)
Hispanic/Latine	14 (14%)	7 (10%)	7 (23%)
Asian/Pacific Islander	4 (4%)	4 (6%)	0 (0%)
Multiracial	3 (3%)	2 (3%)	1 (3%)
Housing status^b			
Unhoused	20 (20%)	12 (17%)	8 (26%)
Marginally housed	49 (49%)	37 (53%)	12 (39%)
Stably housed	32 (32%)	21 (30%)	11 (35%)

^aSex was determined post-mortem and may represent sex assigned at birth or gender at time of death.

^bUnhoused includes living on streets or in a vehicle. Marginally housed includes temporary housing (e.g. hotel, halfway house). Stably housed implies owning or renting one's own place, living in an adult residential facility, or staying with family.