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Theme Series: Accidental overdose poisoning mortality among Canadian populations: a review of coroner and medical examiner files

Scientific Guest Editors: Heather Palis and Amanda Slaunwhite

Guest Editors with Lived/Living Experience: Charlene Burmeister and Pam Young

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Editorial

Engagement of people with lived and living experience in the editorial process: reflections on the special series on the unregulated drug toxicity crisis in Canada

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Part of our "Accidental overdose mortality" theme series.

Introduction

Unregulated drug toxicity deaths (or "overdoses" or "poisonings") remain an ongoing national public health emergency in Canada.[1] Based on the available evidence, it is increasingly recognized that these deaths are a direct result of the criminalization of substance use, the failed war on drugs and outdated drug policy.[2] Coroner records are an important source of data on unregulated drug toxicity deaths, providing information about the circumstances of death (e.g. location of death, contact with health care prior to death, postmortem toxicology, etc.). Each province and territory has its own approach to collecting data, and these have not been previously gathered to examine national unregulated drug toxicity events. This special series includes five articles, each focusing on a specific topic, using data from across all provinces and territories, to provide a national picture of unregulated drug toxicity events in Canada.[3-7]

Engaging people with lived or living experience in research

This series had Guest Editors, including two people with lived or living experience (PWLLE) of substance use and two researchers. This is the first time that *Health Promotion and Chronic Disease Prevention in Canada* has engaged PWLLE in the editorial process.

Historically, PWLLE have not been effectively engaged in research.[8] PWLLE have reported experiencing stigma; facing power imbalances, disrespect, inequality, challenges in accessing meeting materials and lack of flexibility; and experiencing a lack of trauma-informed approaches.[8] Given this history, there is a need for meaningful engagement of PWLLE across the research process—from designing research questions, and collecting and interpreting data based on their lived experiences, to writing, presenting at conferences, peer review and engagement in editorial roles.

It is critical to work toward inclusive priority-setting in research to seek to respond to the drug toxicity crisis. This can be achieved by building partnerships between researchers and PWLLE that centre the expertise of PWLLE. Engagement of PWLLE from the inception of research is critical so that research questions reflect priorities for data collection that respond to the needs of communities. Such engagement can ensure that research findings are used in a manner that is helpful rather than harmful, for example, avoiding misinterpreting data used to inform priority setting (i.e. resource allocation, service provision, policy change) to address the current crisis.

Researchers working with PWLLE of substance use need to consider the ongoing harms of criminalization.[9,10] The Guest Editors highlighted that authentic and meaningful engagement of PWLLE is necessary to move toward emancipating people who use drugs. In this editorial, we report on the reflections of the Guest Editors on the contents of this special issue and the engagement process, taking this history into account.

Overview of the special series content: reflections and recommendations

This special series examined data from across Canada, and this issue reports more specifically on the unregulated drug toxicity crisis as it relates to chronic pain,[5] housing,[6] and impacts across life stages.[7] This series provides a first step toward a national picture of the circumstances of deaths, which has been an ongoing gap in the literature.

Nevertheless, there are important limitations. First, the data are from 2016. While this provides some historical context, there are significant limitations to the generalizability of the findings to the present day. Moreover, given that data were abstracted from coroner records from settings with different reporting structures and protocols, data relevant to the variables of interest were available only in some regions. The high proportion of missing data in some analyses limited the ability to reach conclusions.

To address these limitations, there is a need for more timely data and coordination of responses to allow these data to be

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accessed in near real-time, as was seen with the COVID-19 public health response.[11] Collection methods across regions need to be coordinated to support standardized reporting. This will allow for more meaningful comparisons, which are currently lacking.

The studies in this special series provide a historical snapshot of the unregulated drug toxicity crisis in Canada. Data can be interpreted relative to what we know today, representing a journey over time and showing changes in the unregulated toxic drug supply, expansion of drug user organizations, and increased availability of treatment and harm reduction services. While the implementation of harm reduction and treatment services has prevented deaths,[12] incremental efforts toward service provision have been insufficient to curb the ongoing unregulated drug toxicity crisis, which continues to be a public health emergency in 2024.

Overview of engagement of PWLLE as Guest Editors: reflections and recommendations

In the context of engaging front-line workers in academic activities, peer overdose response workers have emphasized the importance of their work being recognized (e.g. through financial compensation, co-authorship), organizational support and skills development,[13,14] all of which we prioritized in the process of engaging PWLLE as Guest Editors for this special series.

This engagement was facilitated through regular meetings of the researcher and PWLLE Guest Editors. Information was made available to the PWLLE Guest Editors ahead of these meetings; however, meetings did not begin with the expectation that they had reviewed the documents. Documents were reviewed together, to be sure everyone was starting from the same place. This was critical for reasons that are relevant to engaging PWLLE in future editorial roles: workload outside of editorial duties, learning-accommodation requirements, vicarious trauma and traumatic personal life experiences, and the many commitments PWLLE have outside of these meetings. The dedicated group meeting time was organized to suit the PWLLE Guest Editors' approach to the work, acknowledging the competing priorities in their community-based peer-led work.

The engagement with the Guest Editors was mutually beneficial. The PWLLE Guest Editors described this as a valuable experience, as they learned new academic language and research methods and gained confidence for engaging in similar activities in the future. In turn, the researcher Guest Editors were challenged to ask new questions about the data and bring a new lens to the review based on their PWLLE colleagues' input.

Engaging two peers was critical to alleviating the burden on one person. The PWLLE Guest Editors bounced ideas off one another, affirming or challenging one another's perspectives, which ultimately helped to move the discussion toward how to best revise each manuscript.

A key learning was that engaging PWLLE Guest Editors is a time-intensive process. Future engagement of PWLLE in Guest Editor roles across academic disciplines and journals should make deadlines more flexible to acknowledge the time needed for professional development and to develop new processes and protocols for engagement.

Including PWLLE as Guest Editors required significant time and thought as it was a new practice for the journal. For this special series, the PWLLE Guest Editors provided their expertise to ensure the included manuscripts were interpreted relative to the current real-world context of the unregulated drug toxicity crisis in Canada.

While guidelines exist for the engagement of PWLLE in the grant proposal and review process,[15] to our knowledge, such guidance does not exist in terms of PWLLE as Guest Editors in research and knowledge translation. The engagement of PWLLE as Guest Editors for this special series has led to extremely valuable insights that could serve as a foundation for developing such guidelines. This engagement is a process that could be replicated by other journals, potentially strengthening the meaning and impact of academic research across the field of public health.

Statement

The content and views expressed in this article are those of the authors and do not necessarily reflect those of the Government of Canada.

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Original quantitative research

Chronic pain and accidental acute toxicity deaths in Canada, 2016–2017

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This article has been peer reviewed.
Part of our "Accidental overdose mortality" theme series.

Abstract

Introduction: Multiple Canadian jurisdictions have reported a pattern of chronic pain among people who died from substance-related acute toxicity. This study examined the prevalence and characteristics of those with chronic pain using data from a national study of people who died of accidental acute toxicity.

Methods: A cross-sectional analysis of accidental substance-related acute toxicity deaths that occurred in Canada between 1 January 2016 and 31 December 2017 was conducted. The prevalence of pain and pain-related conditions were summarized as counts and percentages of the overall sample. Subgroups of people with and without a documented history of chronic pain were compared across sociodemographic characteristics, health history, contextual factors and substances involved.

Results: From the overall sample (n = 7902), 1056 (13%) people had a history of chronic pain while 6366 (81%) had no documented history. Those with chronic pain tended to be older (40 years and older), unemployed, retired and/or receiving disability supports around the time of death. History of mental health conditions, trauma and surgery or injury was significantly more prevalent among people with chronic pain. Of the substances that most frequently contributed to death, opioids typically prescribed for pain (hydromorphone and oxycodone) were detected in toxicology more often among those with chronic pain than those without.

Conclusion: Findings underscore the cross-cutting role of multiple comorbidities and unmanaged pain, which could compound the risk of acute toxicity death. Continued prioritization of harm reduction and regular patient engagement to assess ongoing needs are among the various opportunities for intervention.

Keywords: *chronic pain, drug overdose, opioid overdose, opioid crisis, controlled substances, substance use, substance-related disorders, acute toxicity*

Highlights

- Between 2016 and 2017, at least one in ten of the people in Canada who died from an accidental acute toxicity had a documented history of chronic pain.
- People with chronic pain tended to be older and with no formal source of income.
- Mental health challenges, trauma and a previous surgery or injury were significantly more common among people with chronic pain than those without.
- Almost all individuals with chronic pain accessed health care services in the year before their death.

Introduction

Substance-related acute toxicity deaths are an ongoing, widespread and complex public health emergency in Canada.[1] Although this emergency is strongly tied to the use of increasingly toxic illegally manufactured drugs,[1] historic high rates of prescription opioid use for pain management also contributed to this crisis.[2] In 2017, Canada had the second-highest rate of daily opioid consumption in the world.[3]

A history of chronic pain was identified in 36% of all opioid-related deaths in Alberta in 2017.[4] In British Columbia, approximately 45% of the people who died from illicit drug acute toxicity in 2016 and 2017 had contacted health services for assistance with pain-related issues in the year before their death.[5]

Chronic pain is a widespread health concern and a major contributor to disability in Canada.[6] Certain populations are at greater risk of chronic pain: those with chronic conditions (e.g. diabetic neuropathy), older adults, postsurgical patients, people who have experienced an injury, and others.[7] There is also a significant mental health burden; concurrent symptoms of depression and anxiety, as well as suicidal ideation, are common among

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individuals with chronic pain.[8] As with substance-related harms, the prevalence and severity of chronic pain is often higher in populations affected by social inequities and discrimination.[2,9] However, critical limitations in the measurement of pain underestimate the true burden of chronic pain at the population level.[7]

In line with evidence on the increased risk of chronic pain for people who have experienced an injury, recent reports have outlined a link between substance-related acute toxicity deaths and employment in industries with a high risk of injury. In British Columbia, 52% of those employed at the time of death were employed in trades or transport or as equipment operators.[10] A similar pattern was reported in Alberta (53%)[4] and Ontario (approximately 30% worked in construction).[11,12] More recent data from Ontario (2018–2020) suggest that among people who died of opioid toxicity, people who worked in construction were more likely to be employed around the time of their death than those without a history of employment in construction (57.7% vs. 11.7%).[13] Of note, a history of chronic pain was common among both those with (37.2%) and without a history of employment in construction (37.9%).[13]

Unmanaged pain may lead to people seeking relief from pain using nonprescribed substances, substance use disorders[9] and an increased risk of overdose, especially among people diagnosed with opioid use disorder.[14] The estimated prevalence of chronic pain among people who use substances ranges from 31% to 55%.[9] A recent systematic review observed wide variability in the prevalence of substance use disorder or substance use–related challenges among patients with chronic non-cancer pain; prevalence of current substance use disorder ranged from 3% to 48%, while 16% to 74% had a lifetime history of any substance use disorder.[15] Similar rates of substance use disorder have been observed among patients with cancer (2% to 35%).[16]

Managing chronic pain is a particular challenge for people who use substances because of stigma and discrimination. A study in Vancouver, BC, found that 66.5% of a sample of people experiencing moderate to extreme pain who use substances reported being denied prescription analgesics by clinicians.[17] Of those who were denied prescription analgesics, many resorted to buying the requested pain medication (40.1%), a different pain medication (34.9%) or heroin (32.9%) on the street (participants were able to report multiple actions taken and may have taken one or all of these actions).[17] Use of nonpharmaceutical substances and diverted prescription medication has been increasingly implicated in the ongoing emergency of substance-related acute toxicity deaths in Canada.[1,12]

Taken together, the evidence suggests a consistent link between substance-related acute toxicity deaths and chronic pain, as well as a disproportionate burden of these deaths among people employed in construction and trades.

In this study, we estimated the minimum national prevalence of pain, and specifically chronic pain, among those who died from accidental substance-related acute toxicity in Canada between 2016 and 2017. We also examined differences between those with and those without a documented history of chronic pain by (1) sociodemographic characteristics, co-occurring health conditions and other known risk factors; (2) health-related encounters leading up to death, including history of prescription medication; (3) circumstances surrounding death and opportunities for intervention; and (4) toxicology findings.

Methods

Ethics statement

This study was reviewed and approved by the Public Health Agency of Canada Research Ethics Board (REB 2018-027P), the University of Manitoba Health Research Ethics Board (HS22710) and the Newfoundland and Labrador Health Research Ethics Board (20200153).

Data sources

This present study is a descriptive, cross-sectional analysis of those who died from accidental substance-related acute toxicity between 1 January 2016 and 31 December 2017 in Canada. Data were obtained from a retrospective review of coroner and medical examiner files to examine the characteristics, circumstances of death and substances involved among those who died from acute toxicity. Cases were defined as those who died from acute intoxication as a direct result of administering exogenous substance(s), with one or more of the substances involved being a drug or alcohol. Detailed information on data collection and study eligibility is published elsewhere.[18]

Where available, residential postal codes were linked to Statistics Canada's Postal Code Conversion File Plus to obtain area-based neighbourhood income quintile after tax (QAATIPPE).[19]

Study population

Between 1 January 2016 and 31 December 2017, 7902 people died of accidental acute toxicity across all the provinces and territories in Canada. The people who died were stratified by history of chronic pain as follows:

- With a history of chronic pain (N = 1056): Any person whose medical records and/or witness statements (from family or friends) mention any of the following around the time of death or in the past: chronic back pain; other pain disorder or chronic pain; long-term (> 90 days) treatment with opioid(s) for pain; fibromyalgia; or arthritis. Fibromyalgia and arthritis were included as they are common conditions catalogued under "chronic primary pain" and "chronic musculoskeletal pain," respectively, in the International Classification of Diseases 11th revision (ICD-11).[20]
- Without a history of chronic pain (N = 6366): Any person without a documented history of chronic pain (according to medical records or witness statements) and no documented history of any of the following conditions (which are often associated with chronic pain): cancer; stroke; vascular diseases; irritable bowel syndrome; inflammatory bowel disease; osteoporosis; chronic autoimmune disorders; or neurological disorders. Conditions associated with chronic pain were identified based on descriptions in the ICD-11.[20] These conditions are not included in the subgroup with a history of chronic pain as it is not possible to differentiate chronic pain from other possible primary symptoms or concerns associated with these conditions.

Of the 7902 people who died, 480 had no documented history of chronic pain but did have a history of a specific condition

associated with chronic pain; they were excluded from the comparison groups. It is possible that more people experienced chronic pain and/or medical conditions associated with chronic pain, but their histories were not documented in the death investigation files.

Variables

The variables included in this study describe interactions with health services, current or recently prescribed medications up to 6 months preceding death, sociodemographic factors, known risk factors and co-occurring conditions for substance-related harms, circumstances of death and the substances involved (refer to Table 1 for descriptions).

Specific medications prescribed for the management of chronic pain were identified based on the RxFiles *Pain Management & Opioids* mini-book.[21] Variables indicating a prescription for opioids, chronic pain medications and any potentially dangerous combinations of medications (e.g. opioids and gabapentinoids or opioids and benzodiazepines)[21] were included in this analysis because of their relevance to people with a history of chronic pain. Although available data on race, ethnicity and Indigeneity were extracted from death investigation files, these data are the focus of separate reports and are not included here.

Most of the variables were captured using "yes" and "no" values. Some were derived by coding "yes" and "no" for values captured in open text fields (e.g. pain medication names) or were categorical.

Statistical analyses

Frequencies and percentages for each variable were estimated for each subgroup. Statistically significant differences were identified through a Pearson chi-square test of independence. In accordance with privacy standards established for the original study,[18] all counts shown in this paper were randomly rounded to base 3 and the percentages were based on these rounded counts. As subtotals and totals were rounded independently from their components, tables might not always sum to 100%. In addition, frequencies less than 10 were suppressed.

All analyses and random rounding were performed using R statistical software version 4.2.1 (R Foundation for Statistical Computing, Vienna, AT).[23,24] As this study is based on a chart review of death investigations, where information on a person's entire life and medical history is not available, percentages represent the minimum proportions of people who had a given characteristic.

Results

Prevalence of pain among people who died of accidental acute toxicity

Of the 7902 people who died of accidental substance-related acute toxicity in Canada between 1 January 2016 and 31 December 2017, at least 1056 (13%) had a documented history of chronic pain whereas 6366 (81%) had no documented history of chronic pain. The remaining 6% had no documented history of chronic pain, but did have a medical condition associated with chronic pain and might have belonged in either group (data not shown). Unspecified type of pain (17%), chronic pain or other pain disorder (8%) and back pain (6%) were the most frequently recorded types of pain (Table 2). For most people with a history of back pain, the pain was chronic (68%).

Interactions with the health care system and history of prescription medication

There were significant differences between people with a history of chronic pain and those without for all interactions with the health care system and prescription medications examined ($p < 0.05$). People with a history of chronic pain had contact with the health care system in the year before their death more frequently (93%) than those without a history of chronic pain (65%). Moreover, the prevalence of contact with the health care system because of pain was almost 2 times higher among people with a history of chronic pain (30%) than among those without such a history (16%). Negative experiences or difficulty accessing the health care system (such as experiences of stigma) were also more prevalent among those with a history of chronic pain (4% vs. <1%). Some of these negative experiences may be related to difficulties accessing adequate pain management services, including pain medications (Table 3).

Among those who had or sought a prescription for opioid medication, those with a history of chronic pain had an opioid prescription reduced or denied in the 6 months prior to death more often (12%) than those without chronic pain (9%; $p < 0.05$). Recent prescriptions for medications typically used for managing chronic pain and potentially dangerous prescription combinations of opioids with gabapentinoids or benzodiazepines were more prevalent among those with a history of chronic pain (12% and 15%, respectively, vs. 1% and 2%, respectively).

Sociodemographic characteristics

For most sociodemographic characteristics, co-occurring health conditions and other examined risk factors, differences were significant between people with a history of chronic pain and those without ($p < 0.05$). Both those with and without a history of chronic pain were more often male; however, the proportion of males was higher among those without chronic pain (78%) than among those with chronic pain (57%) (Table 4).

People with a history of chronic pain tended to be older; 56% were aged 50 years or older compared to 26% of those without chronic pain. Irrespective of their history of chronic pain, the majority of people who died from accidental substance-related acute toxicity lived in neighbourhoods in the lowest or medium-low-income quintiles.

Among people with income source information, those with a history of chronic pain were less often employed (9%) than those without (24%). Among those who were employed, almost half of the people without a history of chronic pain worked in trades, construction or a related field. People with a history of chronic pain more commonly received disability support (11% vs. 5%) or were retired (3% vs. ≤1%). It is important to note that information on income source was unavailable for 59% of people with chronic pain and 45% of people without, and information on occupation was unavailable for 84% of people with chronic pain and 71% of people without.

Co-occurring health conditions and other known risk factors

More than half (53%) of all people with a history of chronic pain experienced a mental health condition compared to 27%

TABLE 1
Descriptions of variables included in the analysis of people who died of accidental acute toxicity, Canada, 2016–2017

Variable	Description
Interactions with health services	
Contact with health services in the preceding year	The person who died accessed health services (inpatient or outpatient) in the year preceding their death (excluding any related to the acute toxicity event that resulted in their death).
Reason for contact with health services was pain related	The person who died accessed health services (inpatient or outpatient) in the year preceding their death, for pain-related issues.
Negative experiences or difficulties accessing health services	The death investigation file had evidence that the person who died had barriers to care such as negative experiences (e.g. stigma) with health services or difficulties accessing the health care system or services.
Opioid prescription reduced or denied in the 6 months preceding their death	The person who died had an opioid prescription reduced or denied in the 6 months preceding their death.
Pain medications	
Acetaminophen[21]	The person who died had been prescribed acetaminophen.
NSAIDs[21]	The person who died had been prescribed one or more of the following NSAIDs: celecoxib, diclofenac, ibuprofen, meloxicam, nabumetone, mefenamic acid, ketoprofen, indomethacin, etodolac or naproxen.
Antidepressants[21]	The person who died had been prescribed one or more of the following antidepressants: amitriptyline, nortriptyline, venlafaxine or duloxetine.
Gabapentinoids[21]	The person who died had been prescribed gabapentin and/or pregabalin.
Topicals[21]	The person who died had been prescribed one or more of the following topical pain medications: capsaicin, lidocaine or maxilene. It is possible that these medications may have been prescribed and administered as a nontopical formulation (e.g. lidocaine injection), but this specification is not available in the dataset.
Opioids, weak or atypical[21]	The person who died had been prescribed one or more of the following weak or atypical opioids: codeine, buprenorphine, tramadol, tapentadol or variations such as buprenorphine/naloxone.
Opioids, strong[21]	The person who died had been prescribed one or more of the following strong opioids: morphine, hydromorphone, oxycodone, fentanyl or methadone.
Opioids, unspecified	The person who died had been prescribed an opioid, but the specific opioid medication was unknown. Given that it was rare for opioids other than those listed in the strong and weak/atypical categories to be documented in the prescription history, those with an unspecified opioid prescription were assumed to have had a prescription for one or more of the strong or weak/atypical opioids listed in this table.
Opioids, any	The person who died had been prescribed one or more of the strong or weak/atypical opioids OR the specific opioid prescribed was unknown.
Muscle relaxants[21]	The person who died had been prescribed one or more of the following muscle relaxants: baclofen, cyclobenzaprine or tizanidine.
Miscellaneous other[21]	The person who died had been prescribed one or more of the following pain medications: trazodone, mirtazapine, carbamazepine, nabilone.
Medications commonly prescribed to manage chronic pain[21]	The person who died had been prescribed at least one of the substances in the acetaminophen, NSAID, antidepressant, gabapentinoid, topical, weak or atypical opioid, strong opioid, muscle relaxant or miscellaneous other pain medication categories described in this table.
Potentially dangerous prescription combinations	
Opioid and gabapentinoid	The person who died had been prescribed both an opioid and a gabapentinoid.
Opioid and benzodiazepine	The person who died had been prescribed both an opioid and a benzodiazepine (e.g. diazepam or alprazolam).
Sociodemographic characteristics	
Sex	The biological sex of the person who died (male or female).
Age group	The age group of the person who died based on their age at the time of death. The following age group categories were used to minimize suppression due to small cell counts across multiple categories (especially in the youngest and oldest age groups): <20 years; 20–29 years; 30–39 years; 40–49 years; 50–59 years; 60–69 years; ≥70 years.
Income source	The income source and/or employment status of the person who died at the time of death. Options include whether the person who died was employed, had specifically worked in construction and trades occupations, was receiving disability payments or was retired at the time of death. More than one option could be true for a single person. Data on income source were missing for 48% of the study population.
Area-based neighbourhood income quintile after tax	This linked variable from Statistics Canada's Postal Code Conversion File Plus (PCCF+)[19] indicates the after-tax income quintile of the neighbourhood of residence of the person who died. These quintiles are based on census metropolitan areas and census agglomerations to control for differences in the cost of living across Canada.
History of involvement with correctional services	The coroner or medical examiner file contained evidence that the person who died was incarcerated at the time of their death or had been incarcerated.

Continued on the following page

TABLE 1 (continued)
Descriptions of variables included in the analysis of people who died of accidental acute toxicity, Canada, 2016–2017

Variable	Description
Being unhoused	The person who died did not have stable, safe or appropriate housing or the immediate means or ability to acquire stable, safe or appropriate housing when they died. The person may have lived unsheltered on the street, stayed in emergency shelters and/or been temporarily accommodated by friends or family ("couch surfing"). The person may also have been at immediate risk of being unhoused because of job loss or eviction by a property owner, for example.
Co-occurring conditions and/or risk factors	
Depression	Medical records or witness statements (from family or friends) document the person who died as having a history of depression. Reports may have included signs of depression (undiagnosed) as well as clinically diagnosed depression.
Anxiety	Medical records or witness statements (from family or friends) describe the person who died as having a history of anxiety. Records may include signs of anxiety (undiagnosed) as well as clinically diagnosed anxiety disorder.
PTSD	Medical records or witness statements (from family or friends) describe the person who died as having a history of PTSD.
Suicidal ideation or attempt	Medical records or witness statements (from family or friends) describe the person who died as having a history of suicidal thoughts or attempts.
History of substance use (excluding alcohol)	The death investigation file describes a history of substance use, not including a history of alcohol use or of taking prescribed medication as directed.
Substance use disorder	Medical records or witness statements (from family or friends) describe the person who died as having a history of substance use disorder, including alcohol use disorder.
Past surgery and/or injury	Medical records or witness statements (from family or friends) described the person who died as having a past surgery and/or injury.
Potentially traumatic life events	Trauma results from "an event, series of events, or set of circumstances that is experienced by an individual as physically or emotionally harmful or life threatening and that has lasting adverse effects on the individual's functioning and mental, physical, social, emotional or spiritual well-being."[22,p.7] To assess exposure to potentially traumatic events, abstractors recorded any evidence in the coroner or medical examiner file that the person who died had experienced a traumatic event in their lifetime. Potentially traumatic events might include: a friend's or family member's health problem; intimate partner problem (e.g. divorce, discord) or other relationship problem (e.g. family argument); job- or school-related problem; financial problem; recent death by suicide of a friend or family member; other death of a friend or family member; criminal legal problem (e.g. arrest, jail, court case) or other legal problem (e.g. custody dispute, civil suit); interpersonal violence (as victim or perpetrator); child maltreatment experience; foster care experience; residential school experience; sexual or physical violence experience or assault. An abstractor might also have noted other potentially traumatic events. Also noted was whether any of the potentially traumatic events occurred within 2 weeks of the person's death. As it was not possible to determine the individual-level impacts of these adverse life events based on a standardized assessment tool (e.g. the PTSD checklist), the potential severity of these events is unknown.
Circumstances of death	
Witness was present at the time of substance use	Another person was present while the person who died consumed the substances that precipitated the fatal acute toxicity event.
Witness was present at the time of the acute toxicity event	Another person was present when the person who died was still alive and experiencing the acute toxicity event.
Person who died showed signs of opioid toxicity	Someone witnessed the person who died having one or more of the following signs of opioid toxicity: snoring or gurgling sounds, difficulty breathing, pinpoint pupils, unconscious or unresponsive, or blue lips or fingernails.
Naloxone was administered	Naloxone was administered to the person who died during the acute toxicity event that precipitated death.
Naloxone administered by a bystander	Whether naloxone was administered by a bystander. Naloxone might have been administered by another person as well as by the bystander.
Naloxone administered by a first responder	Whether naloxone was administered by a first responder (emergency medical services, law enforcement or fire services). Naloxone might also have been administered by another person as well as by the first responder.
Substances involved	
Substances detected on toxicology	The substances that were tested for and detected during toxicological analyses postmortem.
Substances that contribute to death	The substances identified in the death certificate, autopsy report or coroner or medical examiner report as contributing to death.
Substances prescribed to the person who died	The substances that were detected and/or identified as contributing to death had been prescribed to the person who died. The source of this information might include prescription history information or evidence at the scene (e.g. labelled pill bottles).
Substances diverted	The substances that were detected and/or contributed to death were prescribed to someone other than the person who died. The source of this information is usually evidence at the scene (e.g. labelled pill bottles).
Substances of nonpharmaceutical origin	The substances that were detected and/or contributed to death had nonpharmaceutical origin. This includes unregulated drugs and substances not intended for human use, such as industrial or household chemicals or veterinary medications.

Abbreviations: NSAID, nonsteroidal anti-inflammatory drug; PTSD, posttraumatic stress disorder.

TABLE 2
History and types of pain among people who died of accidental substance-related acute toxicity, Canada, 2016–2017, N = 7902

History and type of pain	n	%
History of pain		
No history of chronic pain	6366	81
Any pain (chronic or acute)	2418	31
Chronic pain	1056	13
Acute pain only	72	≤1
Types of pain		
Pain, unspecified type[a]	1359	17
Chronic pain or other pain disorder[b]	612	8
Back pain	507	6
Acute back pain	Suppressed	Suppressed
Chronic back pain	348	68
Unspecified back pain	138	27
Arthritis	258	5
Long-term (>90 days) treatment with opioids for pain[c]	141	2
Fibromyalgia	99	2
Acute pain (excluding back pain)	63	≤1

Note: Numbers <10 and percentages based on such numbers have been suppressed.

[a] Includes any relevant medical history of pain where it was not possible to distinguish between acute and chronic pain.

[b] Includes any relevant medical history of chronic pain or other pain disorder that was not captured as "back pain" and/or "pain, unspecified type."

[c] Long-term treatment that uses opioids for pain management reflects a short- or long-term chronic pain that typical nonsteroidal anti-inflammatory drugs (NSAIDs) and other pain medication management did not alleviate.

of people without chronic pain (Table 4). Depression, anxiety, posttraumatic stress disorder and thoughts of suicide were all more prevalent among those with chronic pain.

While a history of substance use was more common among those without a history of chronic pain (82% vs. 74%), substance use disorder was more common among those with chronic pain (31% vs. 22%). More than half of all people with a history of chronic pain had experienced an injury or surgery (55%) compared to 13% of those without. About one in four people with a history of chronic pain had a combined history of substance use, a mental health condition and a past injury or surgery (Figure 1). In addition, those with a history of chronic pain more often had evidence of a potentially traumatic event in their lifetime (51%) than those without chronic pain (36%; Table 4). Experience of being unhoused were less common among people with a history of chronic pain.

Circumstances surrounding death

Similar proportions of people with and without a history of chronic pain used substances in the presence of others prior to the fatal acute toxicity event (Table 4). However, those with a history of chronic pain were more likely to have had a witness present at the time of death (21% vs. 10%). In addition, those with a history of chronic pain more often showed signs of opioid toxicity during the fatal event (42%) compared to those without (25%).

Naloxone was more commonly administered for those without a history of chronic pain (18% vs. 14%).

Substances involved

Substances that contributed to more than 10% of deaths among people both with and without a history of chronic pain were fentanyl, cocaine, ethanol (alcohol), methamphetamine and morphine (Table 5). Hydromorphone and oxycodone more often contributed to deaths of people with a history of chronic pain, while diacetylmorphine (heroin) and amphetamine more often contributed to deaths of those without. Opioids, which are frequently used to treat chronic pain, and other medications commonly used to treat chronic pain were among the substances that most frequently directly contributed to deaths of people in both groups. Potentially dangerous combinations of opioids with gabapentinoids or benzodiazepines were detected in the toxicology results of 20% and 43%, respectively, of people who had a history of chronic pain. These combinations were prescribed to more than half of people who died.

For all substances and combinations examined, people with a history of chronic pain were more commonly prescribed the substance that was detected in toxicology results than those without (Table 5). For fentanyl and amphetamines, the lower percentage of detections due to substances of nonpharmaceutical origin among those without a history of chronic pain is likely due to higher percentages of substances having an unknown origin (not shown) when information about their medical history is also lacking. Detections of a diverted pharmaceutical medication occurred less than 1% of the time for both populations (data not shown).

Discussion

The persisting high number of substance-related acute toxicity deaths in Canada continues to reflect the role of a toxic and unregulated drug supply[1] within the broader context of factors influencing substance use and related harms. A pattern of injury and chronic pain among people who died from substance-related acute toxicity has been recorded in multiple jurisdictions in Canada.[4,5,10-12] A history of chronic pain was documented in the coroner and medical examiner files for at least 13% of the people who died of accidental acute toxicity between 2016 and 2017.

Important differences in sociodemographic and other equity-relevant factors were noted between those with and those without a history of chronic pain. Most of those with a history of chronic pain were 40 years and older and resided in low- or medium-low-income neighbourhoods; compared with those without chronic pain, they were more often unemployed, receiving disability supports or retired at the time of death. These findings align with earlier work mapping the association between older age and lower socioeconomic status and increased prevalence of chronic pain and disability.[7,26]

TABLE 3
Interactions with the health care system and history of prescription medication among people who died of accidental substance-related acute toxicity, by history of chronic pain, Canada, 2016–2017

	Chronic pain (N = 1056)		No chronic pain (N = 6366)	
	n (N)[a]	%	n (N)[a]	%
Interactions with the health care system				
Any contact with the health care system in the year preceding death	984	93	4119	65
Contact with the health care system for pain-related issues in the year preceding death	315	30	999	16
Negative experience or difficulty accessing the health care system	45	4	36	≤1
History of prescription medication				
Prescription for opioids reduced or denied in the 6 months prior to death among those who had or sought a prescription	93 (747)	12	57 (666)	9
Acetaminophen	90	9	102	2
NSAIDs[b]	228	22	216	3
Antidepressants[c]	270	26	273	4
Gabapentinoids[d]	381	36	279	4
Topicals[e]	Suppressed	Suppressed	Suppressed	Suppressed
Opioids, any	714	68	507	8
Opioids, weak/atypical[f]	63	6	93	2
Opioids, strong[g]	237	22	201	3
Opioids, unspecified	435	41	228	4
Muscle relaxants[h]	69	7	39	≤1
Miscellaneous other[i]	162	15	234	4
Opioid and gabapentinoid	129	12	69	1
Opioid and benzodiazepine	153	15	126	2

Abbreviation: NSAID, nonsteroidal anti-inflammatory drug.

Notes: Numbers <10 and percentages based on such numbers have been suppressed.
Chi-square test, $p < 0.05$.

[a] N is specified when the available sample is a subset of the overall sample, depending on the subgroup examined and/or missing data.

[b] One or more of celecoxib, diclofenac, ibuprofen, meloxicam, nabumetone, mefenamic acid, ketoprofen, indomethacin, etodolac or naproxen.

[c] One or more of amitriptyline, nortriptyline, venlafaxine or duloxetine.

[d] Gabapentin and/or pregabalin.

[e] One or more of capsaicin, lidocaine or maxilene.

[f] One or more of codeine, buprenorphine, tramadol, tapentadol or variations such as buprenorphine/naloxone.

[g] One or more of morphine, hydromorphone, oxycodone, fentanyl or methadone.

[h] One or more of baclofen, cyclobenzaprine or tizanidine.

[i] One or more of trazodone, mirtazapine, carbamazepine or nabilone.

Although there are reports linking substance-related acute toxicity deaths and employment in industries with a high risk of injury,[4,10-13] we found employment in construction and trades to be more common among people with no history of chronic pain. This may be because of the overall lower prevalence of employment among people with chronic pain. However, this study was limited by the amount of missing information on employment history; as such, people who had been employed in trades (and incurred injuries leading to chronic pain or disability) may be undercaptured. Moreover, the seasonal and often time-limited nature of work in construction may serve as an accessible source of employment for people who use substances.[13] The relationship between acute toxicity deaths and employment in construction and trades may also be underpinned by the mutual clustering of men in younger age groups.[13] More research is needed to better characterize the association between employment in construction and trades and substance-related harms, taking into account the recency and duration of employment.

Closer examination of the potential cross-cutting role of multiple interrelated factors revealed considerable overlap between substance use, mental health conditions and past injury or surgery among those with chronic pain. Mental health conditions, history of trauma and past injury and/or surgery were significantly more prevalent among people with a history of chronic pain. These findings are unsurprising given the often-bidirectional association between pain, mental health and substance use–related issues. Rayner et al.[27] reported that patients with depression were more likely to indicate heightened pain-related interference in daily functioning and more generalized pain. Traumatic events are also associated with an increased likelihood of functional somatic syndromes

TABLE 4
Sociodemographic characteristics, co-occurring health conditions, other known risk factors and circumstances surrounding death from accidental substance-related acute toxicity, by history of chronic pain, Canada, 2016–2017

	Chronic pain (N = 1056)		No chronic pain (N = 6366)		*p* value[b]
	n (N)[a]	%	n (N)[a]	%	
Sociodemographic characteristics					
Sex					
Male	597	57	4965	78	<0.05
Female	459	44	1401	22	
Age group, years					
<20	Suppressed	Suppressed	162	3	<0.05
20–29	63	6	1350	21	
30–39	159	15	1830	29	
40–49	243	23	1386	22	
50–59	402	38	1200	19	
60–69	150	14	396	6	
≥70	39	4	42	≤1	
Area-based neighbourhood income quintile[c]					
Q1 (lowest)	420 (942)	45	1878 (4614)	41	>0.05
Q2 (medium-low)	192 (942)	20	978 (4614)	21	
Q3 (middle)	144 (942)	15	714 (4614)	16	
Q4 (medium-high)	117 (942)	12	579 (4614)	13	
Q5 (highest)	72 (942)	8	462 (4614)	10	
Source of income[d]					
Employed around time of death	99	9	1521	24	<0.05
Employed in construction and trades	18 (99)	18	690 (1521)	45	<0.05
Retired	30	3	54	≤1	<0.05
Received disability support	114	11	333	5	<0.05
Co-occurring conditions and other known risk factors					
Any history of the following mental health conditions and symptoms:	555	53	1731	27	<0.05
Depression or depressive symptoms	426	40	1251	20	<0.05
Suicidal ideation	180	17	426	7	<0.05
Anxiety disorder	252	24	714	11	<0.05
PTSD	45	4	126	2	<0.05
Any history of substance use (excluding alcohol)	786	74	5223	82	<0.05
Any history of substance use disorder (including alcohol use disorder)	327	31	1389	22	<0.05
Any history of potentially traumatic life events	543	51	2271	36	<0.05
Any potentially traumatic life event in the 2 weeks prior to death	54 (543)	10	234 (2271)	10	>0.05
Unhoused	51	5	621	10	<0.05
Past injury and/or surgery	582	55	837	13	<0.05
History of involvement with corrections	63	6	471	7	<0.05
Proximal circumstances surrounding death					
Substances used in the presence of others	204	19	1344	21	>0.05
Acute toxicity event was witnessed	216	21	621	10	<0.05
Witness recognized that an acute toxicity event was occurring	63	6	354	6	>0.05
People who died showed signs of opioid toxicity	441	42	1569	25	<0.05
Naloxone was administered	99 (441)	22	438 (1569)	28	<0.05
Naloxone was administered by bystanders	Suppressed	Suppressed	66 (1569)	4	–
Naloxone was administered by first responders	63 (441)	14	276 (1569)	18	>0.05

Note: Numbers <10 and percentages based on such numbers have been suppressed.

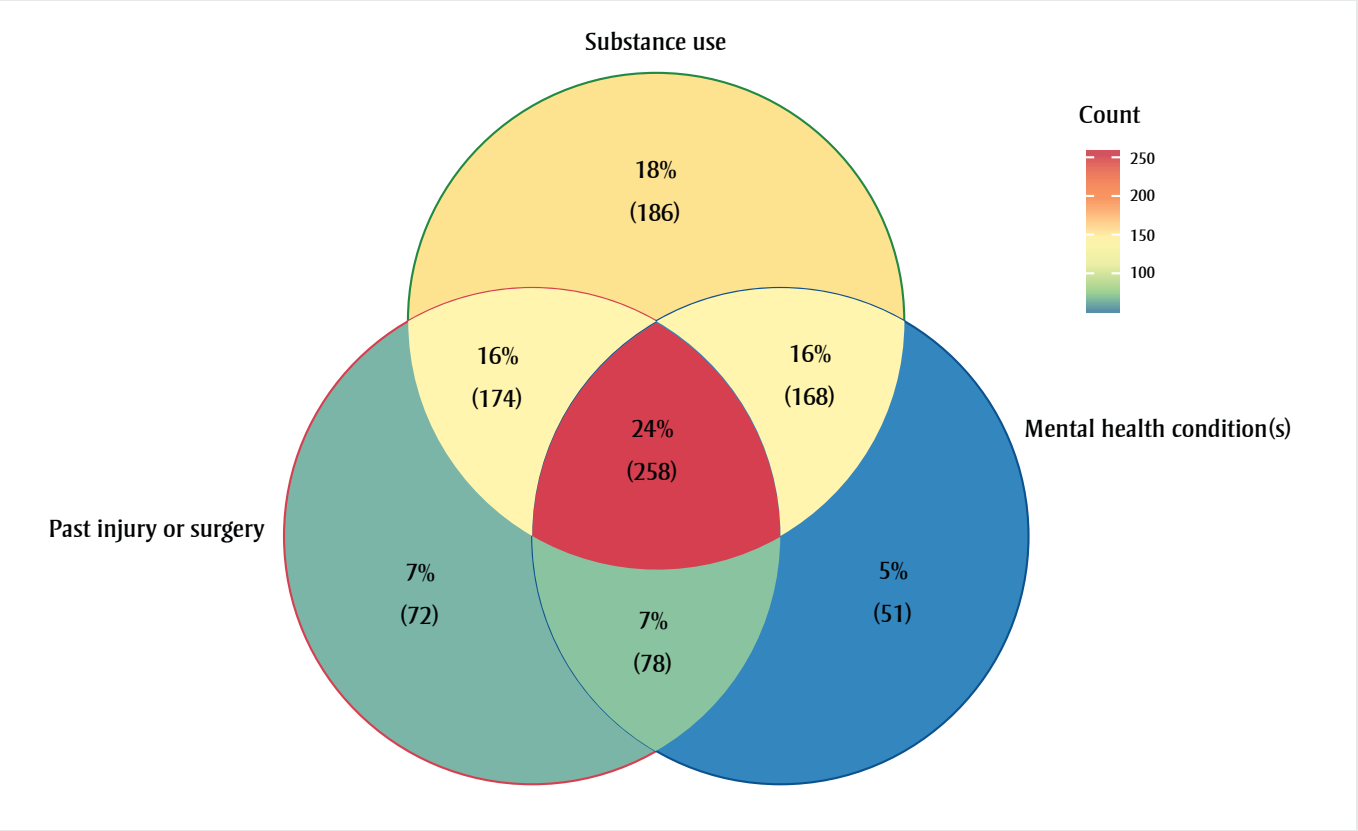
[a] N is specified when the available sample is a subset of the overall sample, depending on the subgroup examined and/or missing data.

[b] *P* value for chi-square test.

[c] Information on the neighbourhood income quintile was unavailable for 11% of people with chronic pain and 28% of people without.

[d] Information on income source was unavailable for 59% of people with chronic pain and 45% of people without. Information on occupation was unavailable for 84% of people with chronic pain and 71% of people without.

FIGURE 1
Venn diagram of the co-occurrence of a history of substance use (excluding alcohol), past injury and/or surgery and mental health conditions among people with a history of chronic pain who died of accidental acute toxicity, Canada, 2016–2017 (N = 1056)



Notes: Mental health condition(s) include one or more of depression or depressive symptoms, suicidal ideation, anxiety or posttraumatic stress disorder (PTSD) mentioned in the death investigation file.

The R package *ggVennDiagram*[25] was used to produce this Venn diagram.

such as fibromyalgia.[28] A history of trauma, such as maltreatment in childhood, and mental health conditions are also intricately linked and may result in greater pain severity and interference.[29]

Fentanyl was the leading contributor to death for both people with a history of chronic pain (24% of deaths) and those without (52% of deaths). When fentanyl contributed to death, people with a history of chronic pain were more often prescribed fentanyl (26%) than those without (1%). This suggests that fentanyl was frequently used for pain management among people with a history of chronic pain. However, the chart review dataset does not have data on the indications for the prescriptions that people were given. Since 2017, the last year of the study period, guidelines for the treatment of chronic non-cancer pain have recommended against opioid therapy,[21,30,31] precipitating an expected decrease in the proportion of patients with chronic pain being prescribed fentanyl and other opioids. In contrast, the detection of fentanyl in drug seizure samples from law enforcement agencies has increased each year until 2021 in Canada, and it remains at high levels.[32]

The substances that most frequently directly contributed to death were more commonly prescribed to people with a history of chronic pain than those without, although nonpharmaceutical substances were also often detected in people with such a history. This does not mean that the prescriptions were necessarily inappropriate; the person who died may have taken more than their prescribed dose or supplemented or combined their medication with another pharmaceutical or nonpharmaceutical substance.

In our study, we found that people with a history of chronic pain had opioid prescriptions reduced or denied in the 6 months prior to death more often than those without such a history. Restricting access to pharmaceutical pain medications has been shown to steer people to illegal drug supplies, which are often more toxic and unpredictable.[17] Of note, less than 1% of the substances that most frequently contributed to death were diverted prescription drugs. A harm reduction approach to prescribing for people with a history of chronic pain that emphasizes patient education about the substances they are prescribed and the potential risks of using other substances in combination with their prescriptions may reduce the risk of accidental death. People with a history of chronic pain had high contact rates (93%) with health services in the year preceding their deaths. About a third of the time, the contact was related to pain, providing opportunities for health care providers to review their patients' prescriptions and talk about the use of pain medications and other approaches to pain management.

TABLE 5
Distribution of substances that contributed to most of the accidental acute toxicity deaths or that are associated with chronic pain, detection during toxicology testing, contribution to death, and substance origin, by history of chronic pain, Canada, 2016–2017

Substance or combination of substances	History of chronic pain (N = 1056)					No history of chronic pain (N = 6366)				
	Deaths where substance or combination was detected	Deaths where substance or combination contributed to death	% of detections due to prescribed medications[a]	% of detections due to non-pharmaceutical origin substances[a]	% of detections due to unknown origin[a]	Deaths where substance or combination detected	Deaths where substance or combination contributed to death	% of detections due to prescribed medications[a]	% of detections due to non-pharmaceutical origin substances[a]	% of detections due to unknown origin[a]
Fentanyl	27	24	26	71	Suppressed	53	52	1	46	53
Cocaine	28	22	n/a	100	0	45	38	n/a	100	0
Hydromorphone	26	16	63	n/a	0	7	4	14	n/a	0
Oxycodone	23	16	71	n/a	0	6	4	18	n/a	0
Ethanol (alcohol)[b]	29	15	n/a	n/a	0	34	23	n/a	n/a	0
Methamphetamine	16	12	n/a	100	0	28	24	n/a	100	0
Morphine[c]	24	11	42	29	24	22	15	4	35	60
Multiple drug toxicity[d]	n/a	11	n/a	n/a	100	n/a	4	n/a	n/a	100
Diacetylmorphine (heroin)	6	6	n/a	100	0	13	12	n/a	100	0
Amphetamine[c]	13	2	11	83	7	24	15	3	47	51
Any opioid	86	71	64	25	18	79	75	9	33	53
Any chronic pain medication	90	72	70	23	14	79	70	14	33	62
Opioid and gabapentinoid	20	4	64	n/a	0	4	1	16	n/a	0
Opioid and benzodiazepine	43	13	53	n/a	0	17	6	12	n/a	0

Abbreviation: n/a, not applicable.

Notes: Percentages are based on counts randomly rounded to base 3; percentages based on counts <10 are suppressed.

Substances detected are those found in toxicology testing, including in trace amounts. Substances can be detected irrespective of whether they are taken as prescribed or consumed intentionally or unintentionally. Detected substances do not always contribute to death, as determined by the investigating medical examiner or coroner. Detections of a single substance of prescribed and nonpharmaceutical origin occurred in <10 deaths for both populations and are not shown in this table. Detections of a diverted pharmaceutical medication occurred less than 1% of the time for both populations and are not shown in this table.

A substance can have more than one origin, e.g. fentanyl can be prescribed or acquired via the illegal drug supply. Some substances are solely of pharmaceutical origin (e.g. hydromorphone, oxycodone, gabapentinoids), and some are solely of nonpharmaceutical origin (e.g. cocaine, methamphetamine). As pharmaceutical fentanyl was not widely available, in the cases where the origin of the fentanyl was unknown, it was likely of nonpharmaceutical origin.

[a] The denominators in these columns are the number of deaths where the substance or combination of substances was detected.

[b] Ethanol (alcohol) originates in the alcoholic beverage industry and is classified as neither pharmaceutical nor nonpharmaceutical; it can sometimes be toxicologically detected as a result of decomposition.

[c] Morphine and amphetamine are active metabolites of other substances (diacetylmorphine [heroin] and methamphetamine, respectively) and their presence may be because they or the parent substance was consumed.

[d] Multiple drug toxicity denotes deaths for which multiple substances contributed to death, but the specific substances involved were not listed.

It is important that these opportunities for intervention not be missed through negative experiences such as prevailing stigma associated with chronic pain and substance use. In this study, people with a history of chronic pain had negative experiences when accessing health care services more often than those without such a history. These negative experiences further marginalize people who live with chronic pain and people who use opioids or other substances, possibly preventing them from receiving adequate services. Our findings are supported by those of a qualitative study examining the lived experiences of people who use substances; Dassieu et al.[33] also describe the challenges in accessing interdisciplinary pain management services, the relative inaccessibility of nonpharmacological therapies for pain (such as physiotherapy) and the resultant potential for self-management with illegal drugs as a last resort.

Strengths and limitations

The chart review study is based on coroner and medical examiner files, which

have different formats and investigation protocols across jurisdictions. The variables of interest in this analysis have different availabilities across jurisdictions; therefore, we are only able to present minimum proportions.

Capacity for toxicology testing also varies by jurisdiction and over time. The dataset does not include information about dosage and regimen duration, both of which contribute to the degree of risk for acute toxicity. However, the overseeing coroner or medical examiner would have assessed specifics on prescribed medications when determining the cause of death. Some medications have multiple on-label and off-label purposes; for example, buprenorphine, methadone and long-acting morphine can be prescribed for pain or opioid agonist therapy, and these opioids may have been misclassified as pain medications in the absence of information on indication(s) for use.

The ICD-11 diagnostic codes for chronic pain were published in 2018 and were not available to health care providers during the study period. In addition, some people living with chronic pain might not have sought a diagnosis or formal care, especially if they had previously faced barriers in accessing health care services (e.g. stigma). It is also possible that people with no known social contacts (such as family or friends) were missed if there was no one who could report on their experiences of chronic pain and/or barriers in accessing health care services.

Certain conditions associated with chronic pain (such as endometriosis) could not be separated from broader categories and were not excluded from the group with no history of chronic pain, resulting in some misclassification of people captured as having no history of chronic pain. In addition, a documented history of chronic pain may have influenced the collection of other medical and prescription histories. Given these limitations, differences observed between those with and those without a history of chronic pain are susceptible to bias. Furthermore, the cross-sectional nature of this study precludes causal inference.

Finally, it is important to note that the onset of the COVID-19 pandemic affected substance use patterns and harms.[34] Accidental opioid toxicity deaths have almost doubled in Canada since March 2020 and remain higher than pre-pandemic trends, owing to various factors including the supply of increasingly unpredictable and toxic illegally manufactured drugs.[34] While this present study examined a previously undescribed national population, future work should investigate the role of chronic pain in the sustained increase in substance-related acute toxicity deaths since the COVID-19 pandemic to guide policy planning and actions.

Conclusion

Many cross-cutting and interacting factors likely influence the distinct burden of substance-related harms, including acute toxicity deaths, among people with chronic pain. Almost all the individuals with a documented history of chronic pain accessed health care services before their death, and almost a third of these interactions were for pain-related reasons. More than one in 10 people with chronic pain had an opioid prescription denied or reduced in the 6 months before their death. These findings signal unmanaged pain and the need for safe, adequate and accessible pain management solutions.

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Conflicts of interest

The authors have no conflicts of interest.

Authors' contributions and statement

JV: Conceptualization, Formal analysis, Investigation, Writing – Original draft, Writing – Review & Editing.

AV: Conceptualization, Formal analysis, Investigation, Writing – Original Draft, Writing – Review & Editing.

LY: Conceptualization, Investigation, Writing – Review & Editing.

KH: Validation, Investigation, Writing – Review & Editing.

JFL: Conceptualization, Investigation, Writing – Review & Editing.

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Original quantitative research

Housing status and accidental substance-related acute toxicity deaths in Canada, 2016–2017

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Part of our "Accidental overdose mortality" theme series.

Abstract

Introduction: There is a complex relationship between housing status and substance use, where substance use reduces housing opportunities and being unhoused increases reasons to use substances, and the associated risks and stigma.

Methods: In this descriptive analysis of people without housing who died of accidental substance-related acute toxicity in Canada, we used death investigation data from a national chart review study of substance-related acute toxicity deaths in 2016 and 2017 to compare sociodemographic factors, health histories, circumstances of death and substances contributing to death of people who were unhoused and people not identified as unhoused, using Pearson chi-square test. The demographic distribution of people who died of acute toxicity was compared with the 2016 Nationally Coordinated Point-In-Time Count of Homelessness in Canadian Communities and the 2016 Census.

Results: People without housing were substantially overrepresented among those who died of acute toxicity in 2016 and 2017 (8.9% versus < 1% of the overall population). The acute toxicity event leading to death of people without housing occurred more often in an outdoor setting (24%); an opioid and/or stimulant was identified as contributing to their death more frequently (68%–82%; both contributed in 59% of their deaths); and they were more frequently discharged from an institution in the month before their death (7%).

Conclusion: We identified several potential opportunities to reduce acute toxicity deaths among people who are unhoused, including during contacts with health care and other institutions, through harm reduction supports for opioid and stimulant use, and by creating safer environments for people without housing.

Keywords: *drug overdose, opiate overdose, poisoning, mortality, homeless persons, unhoused, unsheltered, homelessness*

Highlights

- 8.9% of people who died of accidental substance-related acute toxicity in 2016 and 2017 were unhoused at the time of their death compared to less than 1% of the general population in Canada.
- One in four of the acute toxicity events that lead to death occurred outdoors.
- People who were unhoused at the time of their death had an opioid and/or stimulant identified as contributing to their death more often than those with housing.
- Toxicology tests detected opioids and stimulants in combination in more than half of the people who were unhoused at the time of their death.

Introduction

The overdose crisis in Canada is a significant public health concern, with 36 442 deaths related to apparent opioid toxicity between January 2016 and December 2022.[1] Provincial and municipal reports show a greater impact on some populations than others, including people who are unhoused.[2-5] An estimated 235 000 people were unhoused in Canada in 2016, including 22 190 people who were in shelters on any given night.[6,7]

In this paper, we have chosen to use "people who were unhoused" or "people without housing" rather than "people who were experiencing homelessness." A house is a physical shelter, but a home encompasses more than a physical location and is tied to personal meanings and social connections.[8-11] A person without access to a stable or safe physical shelter may still have a home in the people around them, the spaces they live in and wider community.

People who are unhoused have higher rates of substance use than the general

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population.[12,13] The relationships between housing insecurity ("the loss of, threat to, or uncertainty of a safe, stable, and affordable home environment"[14,p.344]), substance use and harms from substance use are complex. Substance use and substance use disorders are commonly cited reasons for housing loss, and people who are unhoused are likely to have experienced trauma, mental illness and/or incarceration, which contribute to a higher risk of substance use.[15-19] Being unhoused may increase use as a way of dealing with the difficulties and dangers of life without secure, private housing.[15,20-22] In addition, being unhoused directly and indirectly increases the harms associated with substance use, including acute toxicity events.[23] Due to stigmatization, as well as logistical barriers to service access, people who are unhoused may have worse access to treatment and harm reduction services.[24] They may also feel the need to conceal or rush substance use, use alone and use larger amounts to avoid drug possession charges.[25,26] Finally, being unhoused may put people in situations that are criminalized, and periods of incarceration may disrupt continuity of treatment and services as well as drug supply, contributing to increased risk of acute toxicity events upon release, in part because of reduced drug tolerance.[27-29]

The aim of this study is to describe the sociodemographic profiles, health history, substances involved and the circumstances of death due to accidental substance-related acute toxicity of people who were unhoused.

Methods

Ethics statement

This study was reviewed and approved by the Public Health Agency of Canada Research Ethics Board (REB 2018-027P), the University of Manitoba Health Research Ethics Board (HS22710) and the Newfoundland and Labrador Health Research Ethics Board (20200153).

Main data source

This study uses data from a national, retrospective chart review of substance-related acute toxicity deaths from coroner and medical examiner investigation files from between 1 January 2016 and 31 December 2017. Although death investigation procedures vary across Canada, the death investigation files generally contain some combination of a death certificate, coroner or medical examiner report, witness statements, medical records, police reports, toxicology reports and/or autopsy reports.

The case definition includes all individuals who died in Canada between 1 January 2016 and 31 December 2017 of accidental acute toxicity resulting from the direct effects of the administration of exogenous substance(s), where one or more of the substances was a drug or alcohol. Variables collected from the death investigation files by the data abstractors included sociodemographic risk factors, documented substance use and medical history, circumstances of death and toxicological findings. Because histories of mental and physical health conditions or symptoms were collected from medical records or witness statements in the death investigation files, these conditions and symptoms are not necessarily clinical diagnoses. Similarly, the study only captured what was available in the file, which might not have included the person's entire medical history or life experiences.

Abstractors received training and written guidance on what kinds of information to look for in death investigation files and how to code or describe this in the database. Potentially traumatic events might include: a friend's or family member's health problem; intimate partner problem (e.g. divorce, discord) or other relationship problem (e.g. family argument); job- or school-related problem; financial problem; recent death by suicide of a friend or family member; other death of a friend or family member; criminal legal problem (e.g. arrest, jail, court case) or other legal problem (e.g. custody dispute, civil law); interpersonal violence (as victim or perpetrator); child maltreatment experience; foster care experience; residential school experience; or experience of sexual abuse or physical abuse or assault. An abstractor might also have entered another event with an explanation for how it meets the definition of a potentially traumatic event. The chart review study protocol, database and definitions of variables are described in greater detail elsewhere. [30]

Definitions for housing status

To identify people who were unhoused, this study uses the Canadian Observatory on Homelessness' definition of homelessness, as "the situation of an individual … without stable, permanent, appropriate housing, or the immediate prospect, means and ability of acquiring it."[31,p.1] This includes people living unsheltered on the street, staying in emergency shelters and temporarily accommodated by couch surfing, staying with friends or family or trading informal employment or resources for housing. It also includes people at immediate risk of being unhoused because of job loss or eviction by a property owner, for example.

People who died of accidental acute toxicity in the national dataset were identified as "unhoused at the time of death" and/or "unhoused within 6 months of death" based on variables related to their living arrangements, any recent moves within 6 months of death, and open text comments abstractors made about the investigation file. A specific variable in the database indicates evidence in the death investigation file that the person experienced housing instability in their lifetime. We categorized those who had no documented evidence of being unhoused at any point in their lifetime (i.e. are not included in any of the variables for being unhoused) as "not identified as being unhoused." As coroner and medical examiner files are not a complete record of a person's life, some people categorized as "not identified as being unhoused" might be misclassified.

People who were hospitalized or in a correctional facility or other institution at the time of death were excluded from both "unhoused at time of death" and "not identified as being unhoused" categories, and are not included in comparisons between these two groups. Data on living arrangements were missing for 10.9% of all people; they too were excluded from further analysis. Comparisons of people missing data on their living arrangement with those not missing data revealed statistically significant differences in a subset of key variables that included age, sex, substance use history, history of substance use disorder (excluding alcohol) and contact with the health system in the year before death.

Statistical analyses

We selected variables for analysis based on hypothesized relationships with housing and substance use or as potential intervention points. As noted previously, death investigation protocols vary across

jurisdictions; as a result, many of the variables were not available in the source material for all jurisdictions, limiting descriptive analysis to the minimum numbers and proportions of people who died of acute toxicity who had information recorded for a given variable. We conducted Pearson chi-square tests to assess statistical differences between people who were unhoused at the time of their deaths and people not identified as unhoused ($p < 0.05$). As this study is descriptive and the variables were preselected based on hypothesized relationships, no adjustments were made for multiple comparisons. The substances and substance combinations most frequently contributing to the deaths of people in both subpopulations were identified using the ComplexUpset package.[32]

We also compared the demographic distribution of both populations who died of acute toxicity with the 2016 Coordinated Point-In-Time Count of Homelessness in Canadian Communities[20] and the 2016 Census.[33] Between 1 January 2016 and 30 April 2016, 32 communities across Canada participated in a coordinated count of people in shelters and on the streets within their community. Some of the counts also included people who were in health care or correctional facilities and had no place to go on discharge. The census provides a statistical overview of the population of Canada every 5 years. Individuals are counted at their usual place of residence, which can be a private or collective dwelling. While collective dwellings include shelters, the census is limited in its ability to capture people without housing.[12] Tests of statistical difference were not used in these comparisons because the populations from these three data sources are not independent.

To protect privacy, all counts from the chart review study data are randomly rounded to base 3, and proportions and rates are based on rounded counts.[30] All statistical analyses and random rounding were performed using R statistical software[34] and RStudio (version 2022.02.0).

Results

Comparison with the general population

Based on the available data for 7902 people in Canada who died of accidental acute toxicity in 2016 or 2017, at least 9.4% (n = 744) had been unhoused within 6 months of their death, and 8.9% (n = 702) were unhoused at the time of their death (Figure 1).

As 0.06% to 0.10% of the population of Canada was unhoused on a given day in 2016, with 0.67% unhoused at any time in 2016,[11,12,33] people who were unhoused were overrepresented among those who died of accidental acute toxicity.

Of the people who died of accidental acute toxicity, those who were unhoused tended to be younger (20–49 years; $p < 0.001$) and male ($p < 0.05$) (Table 1). Compared with the overall population[33] or unhoused population in Canada in 2016,[21] people who died of accidental acute toxicity and were unhoused were more commonly aged between 30 and 59 years and more often male (Table 1).

Encounters with the health care system by housing status

The minimum proportion of people who had contact with the health care system in the year before their death from accidental acute toxicity was higher for people not identified as unhoused than for those without housing (75% vs. 68%; $p < 0.001$). People without housing accessed outpatient and inpatient services more often than those not identified as unhoused (Table 2). They also sought care due to acute injury (8% vs. 4%; $p < 0.001$), a nonfatal acute toxicity event (14% vs. 7%; $p < 0.001$) or substance use and/or addictions (16% vs. 12%; $p < 0.05$) more often. However, the reason for seeking care was unknown for many people, irrespective of their housing status, and differences between the two groups may be related to reporting biases rather than true differences.

Histories of substance use, mental health and potentially traumatic life events

Of the people who died of accidental acute toxicity, those without housing had histories of substance use and of chronic substance use more often than those not identified as unhoused (92% vs. 83% and 63% vs. 54%, respectively; $p < 0.001$) (Table 3). The proportions of people with histories of substance use disorder or alcohol use disorder were similar for both populations. Depression or depressive symptoms were recorded more often for people not identified as unhoused than for those without housing (26% vs. 15%; $p < 0.001$). Mental health history was unknown for 29% of people without housing and 22% of those not identified as unhoused ($p < 0.001$).

Among the people who died of accidental acute toxicity, about half (53%) of those without housing had a history of at least one potentially traumatic event compared with about one-third (38%) for people not identified as unhoused, a difference of 17 percentage points ($p < 0.001$) (Table 3). People without housing experienced a potentially traumatic life event in the 2 weeks before their death more commonly than peers who were not identified as unhoused (6% vs. 4%; $p < 0.05$).

While criminal legal problems and intimate partner problems were the most frequently identified potentially traumatic life events for both populations, criminal legal problems were significantly more common for people who were unhoused (33% vs. 16%; $p < 0.001$).

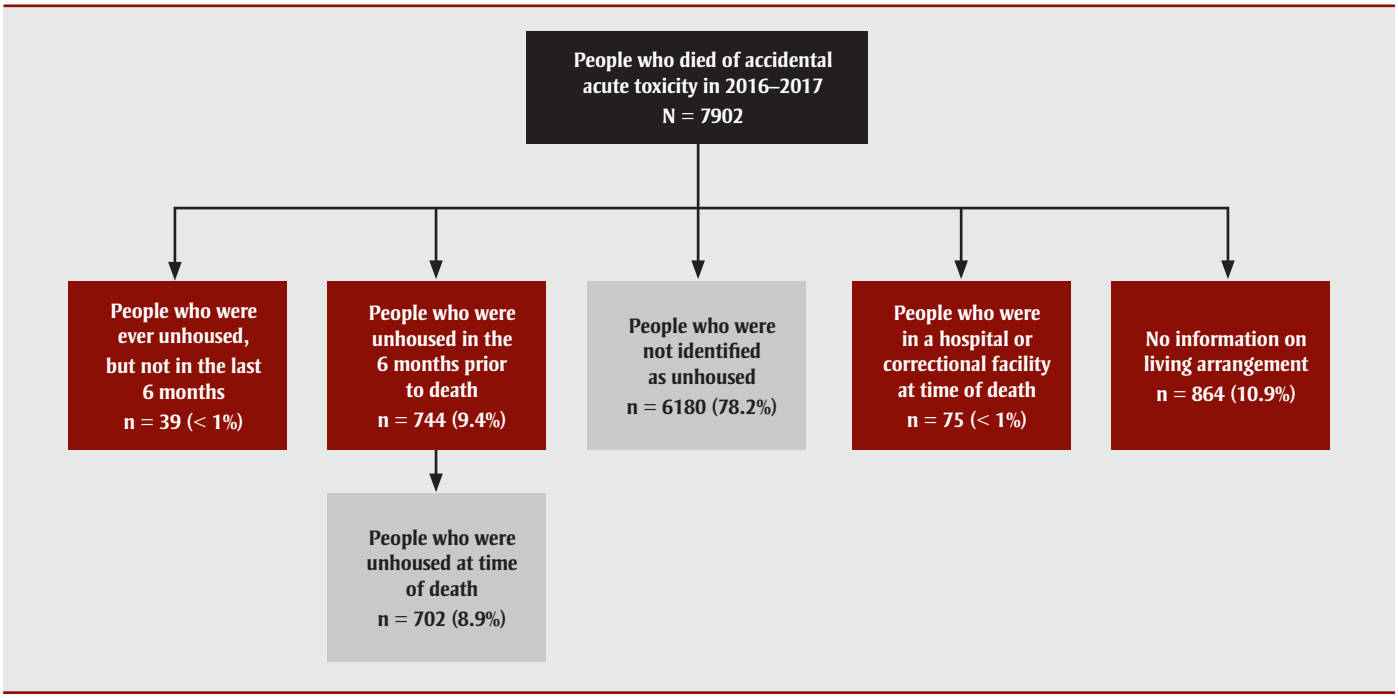
Recent institutionalization

At least 7% of people who died of accidental acute toxicity and were unhoused had been discharged from an institution up to one month before their death ($p < 0.001$). The proportion discharged from a correctional facility was higher for people without housing than those not identified as unhoused (3% vs. 1%; $p < 0.001$), but the proportions discharged from a hospital were similar (Table 3).

Circumstances of death

The locations of the acute toxicity events that led to accidental deaths differed significantly by housing status (Table 3). For people without housing, the acute toxicity events most often occurred in an outdoor public place, personal residence or home of another person (about 20%–23% of acute toxicity deaths for each location). For people not identified as unhoused, most fatal acute toxicity events occurred in a personal residence (74%). About one in four acute toxicity events experienced by people without housing occurred outdoors (for example, in outdoor public places, front or back yards of residences, sidewalks beside buildings), compared to only 3% for people not identified as unhoused ($p < 0.001$). People not identified as unhoused more commonly experienced

FIGURE 1
Housing status of people who died of accidental acute toxicity, Canada, 2016–2017



Note: In this study, we compare the groups in grey boxes. The groups in red boxes were excluded from these comparisons.

the acute toxicity event in or near a bed (29%; $p < 0.001$).

Regardless of housing status, the location of death was the same as the location of the acute toxicity event for most of the people who died, but people not identified as unhoused more often died at the same location and people without housing more often died in hospital after transport from another location.

Where data were available, the majority of people, irrespective of housing status, were using substances alone prior to the acute toxicity event, but people without housing were significantly more likely to be in the presence of other people (21%; $p < 0.001$) (Table 3).

People without housing who were found exhibiting one or more symptoms of opioid toxicity (i.e. snoring/gurgling, difficulty breathing, pinpoint pupils, unconscious/unresponsive or blue lips/fingernails/face) were more likely to have received naloxone than people not identified as unhoused (42% vs. 23%; $p < 0.001$). Among those who exhibited at least one symptom of opioid toxicity, people without housing were more likely to have received naloxone from EMS (28% vs. 14%), hospital staff (16% vs. 7%) and bystanders (9% vs. 3%), compared to people not identified as unhoused ($p < 0.001$). EMS, law enforcement and hospital staff were equally likely to attend the scene for people with either housing status, but fire service personnel were more likely to attend the scene for people without housing (20%; $p < 0.001$).

While the proportions of individuals known to still be alive when found were similar in both groups, a greater proportion of people not identified as unhoused were already dead when found (31% vs. 23%). For a greater proportion of people without housing, it was unclear or unknown whether they were still alive when found.

Substances contributing to death

Multiple substances contributed to most deaths, but deaths involving multiple substances were significantly more common for people without housing than for those not identified as unhoused (79% vs. 70%; $p < 0.001$). Where the specific substance or substances contributing to death were known, their origin was less often pharmaceutical in nature (25% vs. 37%; $p < 0.001$) and less often prescribed to the person who died (38% vs. 48%; $p < 0.05$) for people without housing (Table 3).

The substances most commonly contributing to accidental toxicity deaths were the same irrespective of housing status, but they differed by degree of contribution (Table 4). Fentanyl most frequently contributed to death for people with either housing status, but contributed significantly more frequently for people without housing than for those not identified as unhoused ($p < 0.001$). Cocaine contributed to a similar proportion of deaths for people with both housing statuses, but other stimulants like methamphetamine and amphetamine contributed to a greater proportion of deaths for people without housing than those with housing ($p < 0.001$).

While fentanyl was most often involved in the substance combinations that most frequently contributed to death for people with both housing statuses, the involvement of stimulants varied (Table 5). For people without housing, methamphetamine was more common among the highest-ranking substance combinations contributing to death, while cocaine was more common for people not identified as unhoused.

Discussion

People without housing are substantially overrepresented among those who died of

TABLE 1
Distribution by sex and age groups of the people who died of accidental acute toxicity, by housing status at time of death, 2016–2017, and for the total population, Canada, 2016

Age group, years	Unhoused N = 702 n (%)	Not identified as unhoused N = 6180 n (%)	Unhoused population (estimated proportions) N = 4266 n (%)	Total population N = 35 151 730 n (%)
Any sex				
≤19	Suppressed	123 (2.0)	1041 (24.4)	7 865 725 (22.4)
20–29	150 (21.4)	1095 (17.7)	776 (18.2)	4 528 685 (12.9)
30–39	222 (31.6)	1596 (25.8)	875 (20.5)	4 617 765 (13.1)
40–49	168 (23.9)	1323 (21.4)	538 (12.6)	4 615 095 (13.1)
50–59	138 (19.7)	1416 (22.9)	294 (6.9)	5 298 310 (15.1)
≥60	18 (2.6)	627 (10.2)	747 (17.5)	8 226 145 (23.4)
Male				
All ages	546 (77.8)	4581 (74.1)	2656 (62.3)	17 264 200 (49.1)
≤19	Suppressed	72 (1.2)	538 (12.6)	4 032 135 (11.5)
20–29	111 (15.8)	849 (13.7)	567 (13.3)	2 288 965 (6.5)
30–39	168 (23.9)	1272 (20.6)	691 (16.2)	2 266 925 (6.4)
40–49	138 (19.7)	978 (15.8)	380 (8.9)	2 262 200 (6.4)
50–59	111 (15.8)	999 (16.2)	137 (3.2)	2 603 935 (7.4)
≥60	15 (2.1)	411 (6.7)	346 (8.1)	3 810 035 (10.8)
Female				
All ages	156 (22.2)	1596 (25.8)	1610 (37.7)	17 887 530 (50.9)
≤19	Suppressed	51 (0.8)	503 (11.8)	3 833 590 (10.9)
20–29	36 (5.1)	249 (4.0)	209 (4.9)	2 239 720 (6.4)
30–39	54 (7.7)	321 (5.2)	183 (4.3)	2 350 840 (6.7)
40–49	33 (4.7)	345 (5.6)	158 (3.7)	2 352 905 (6.7)
50–59	27 (3.9)	417 (6.7)	158 (3.7)	2 694 375 (7.7)
≥60	Suppressed	216 (3.5)	401 (9.4)	4 416 115 (12.6)

Data sources: National chart review study of substance-related acute toxicity deaths in 2016 and 2017, Public Health Agency of Canada; 2016 Coordinated Point-In-Time Count of Homelessness in Canadian Communities[20]; 2016 Census Profile.[33]

Notes: Counts are randomly rounded to base 3. Cells with counts <10 are suppressed to protect privacy. Percentages are based on rounded counts.

TABLE 2
Distribution of health care system encounters for people who died of accidental acute toxicity, by housing status at time of death, Canada, 2016–2017 (N = 7902)

Variable	Unhoused, n (%)	Not identified as unhoused, n (%)	*p* value[a]
Total accidental deaths	**N = 702**	**N = 6180**	
Contact with the health care system in the year before death	477 (68)	4608 (75)	**<0.001**
Contact with the health care system in the year before death	**N = 477**	**N = 4608**	
Received outpatient treatment	342 (72)	2961 (64)	**<0.001**
Received inpatient treatment	138 (29)	927 (20)	**<0.001**
Unknown if treatment received was outpatient or inpatient	81 (17)	1323 (29)	**<0.001**
Sought care for acute injury	36 (8)	198 (4)	**<0.001**
Sought care for pain	105 (22)	1197 (26)	>0.05
Sought care for a nonfatal acute toxicity event	66 (14)	312 (7)	**<0.001**
Sought care for substance use and/or addictions	78 (16)	567 (12)	**<0.05**
Sought care for mental health	45 (9)	513 (11)	>0.05
Sought care for surgery	12 (3)	135 (3)	>0.05
Reason for seeking care was unknown	189 (40)	1932 (42)	>0.05

Notes: Percentages are based on counts that have been randomly rounded to base 3. Bolded *p* values indicate statistically significant data.
[a] *P* value for chi-square test. Exact *p* values are not shown to protect the random rounding.

TABLE 3
Distribution of health history, circumstances of death and substances contributing to death of people who died of accidental acute toxicity, by housing status at time of death, Canada, 2016–2017 (N = 7902)

Variable	Unhoused, n (%)	Not identified as unhoused, n (%)	*p* value [a]
Mental health	**N = 702**	**N = 6180**	–
Unknown mental health history	204 (29)	1374 (22)	**<0.001**
Depression or depressive symptoms	108 (15)	1581 (26)	**<0.001**
History of substance use disorder (excluding alcohol)	135 (19)	1221 (20)	>0.05
History of alcohol use disorder	57 (8)	564 (9)	>0.05
Substance use	**N = 702**	**N = 6180**	
History of substance use (excluding alcohol)	642 (92)	5106 (83)	**<0.001**
Mention of chronic substance use	444 (63)	3312 (54)	**<0.001**
Potentially traumatic life events [b]	**N = 702**	**N = 6180**	
History of potentially traumatic life events	372 (53)	2364 (38)	**<0.001**
Intimate partner problem (e.g. divorce, discord)	108 (15)	870 (14)	>0.05
Criminal legal problem (e.g. arrest, jail, court)	231 (33)	960 (16)	**<0.001**
Any potentially traumatic life event in the 2 weeks before death	42 (6)	231 (4)	**<0.05**
Release from an institution at least 1 month before death	**N = 702**	**N = 6180**	
Correctional facility [c]	24 (3)	66 (1)	**<0.001**
Hospital	24 (3)	129 (2)	>0.05
Any institution [d]	51 (7)	237 (4)	**<0.001**
Location of acute toxicity event leading to death	**N = 702**	**N = 6180**	**<0.001**
Outdoor public place	159 (22)	138 (2)	
Personal residence [e]	138 (20)	4572 (74)	
Home of another person	159 (23)	411 (7)	
Shelter	60 (9)	0	
Hotel or motel	33 (5)	246 (4)	
Public building	36 (5)	66 (1)	
Other or unknown	120 (17)	744 (12)	
Specific setting of acute toxicity event leading to death	**N = 702**	**N = 6180**	
Outdoors	171 (24)	210 (3)	**<0.001**
In vehicle	24 (3)	141 (2)	>0.05
In or near a bed	147 (21)	1791 (29)	**<0.001**
Location of death	**N = 702**	**N = 6180**	**<0.01**
Same as the location of the acute toxicity event	519 (74)	4764 (77)	
Hospital [f]	147 (21)	981 (16)	
Other	39 (6)	435 (7)	
Using substances in the presence of others before the acute toxicity event	**N = 702**	**N = 6180**	**<0.001**
Yes	147 (21)	1017 (16)	
Yes, alcohol only	21 (3)	246 (4)	
No	240 (34)	2505 (41)	
Unknown	297 (42)	2409 (39)	
Death was witnessed	**N = 702**	**N = 6180**	**<0.001**
Alive when found	75 (11)	645 (10)	
Deceased when found	162 (23)	1905 (31)	
Unclear if person was still alive when found	171 (24)	1293 (21)	
Unknown	294 (42)	2337 (38)	

Continued on the following page

TABLE 3 (continued)
Distribution of health history, circumstances of death and substances contributing to death of people who died of accidental acute toxicity, by housing status at time of death, Canada, 2016–2017 (N = 7902)

Variable	Unhoused, n (%)	Not identified as unhoused, n (%)	*p* value [a]
Attended the scene of acute toxicity event	**N = 702**	**N = 6180**	
EMS	378 (54)	3510 (57)	>0.05
Fire services	138 (20)	876 (14)	**<0.001**
Law enforcement	351 (50)	3180 (51)	>0.05
Hospital staff	15 (2)	183 (3)	>0.05
People who showed at least 1 symptom of opioid toxicity [g]	**N = 207**	**N = 1620**	
Received naloxone	87 (42)	375 (23)	**<0.001**
Naloxone administered by EMS	57 (28)	234 (14)	**<0.001**
Naloxone administered by hospital staff	33 (16)	120 (7)	**<0.001**
Naloxone administered by bystanders	18 (9)	45 (3)	**<0.001**
Multiple substances contributed to death	**N = 702**	**N = 6180**	
Yes	558 (79)	4311 (70)	**<0.001**
Substance types that contributed to death	**N = 702**	**N = 6180**	
Opioid contributed to death	579 (82)	4545 (74)	**<0.001**
Stimulant contributed to death	477 (68)	3051 (49)	**<0.001**
Both an opioid and a stimulant contributed to death	411 (59)	2250 (36)	**<0.001**
Specific substance(s) contributing to death are known	**N = 675**	**N = 5820**	
Nonpharmaceutical origin	573 (85)	4065 (70)	**<0.001**
Pharmaceutical origin	168 (25)	2175 (37)	**<0.001**
Substance contributing to death was of pharmaceutical origin	**N = 168**	**N = 2175**	
The substance was prescribed to the person who died	63 (38)	1038 (48)	**<0.05**

Note: Bolded chi-square test *p* values indicate statistically significant differences.

[a] Where categories are mutually exclusive the chi-square test compares the distribution across the categories for those who were unhoused and not identified as unhoused. Where categories were not mutually exclusive the chi-square test compares the presence and absence of that category for those who were unhoused and not identified as unhoused.

[b] Includes events identified for at least 10% of people in a group.

[c] Includes remand centres and young offender centres.

[d] Includes correctional facilities, remand centres, young offender centres, hospitals, mental health facilities, long-term residential health facilities (such as nursing homes), other health facilities or supervised residential facilities.

[e] A person experiencing homelessness may have a temporary, unsafe and/or inappropriate residence.[26]

[f]. Includes only the people who were transported from another location to a hospital. If the acute toxicity event occurred in the hospital where the person died, this would be categorized as "same as the location of the acute toxicity event."

[g] Includes events identified for at least 5% of people in a group.

acute toxicity in 2016 and 2017 (8.9% of people who died were unhoused compared to < 1% of the overall population of Canada). The study findings suggest several opportunities for intervention and improving supports for people who are experiencing or at risk of being unhoused and are at risk of an acute toxicity death.

At least 7% of people without housing, which is about twice as many people not identified as unhoused, had been discharged from a health care or correctional institution in the month preceding their death. The stays in the institutions may or may not have been related to substance use. Risk of acute toxicity death is higher after a recent discharge for many reasons: a person's tolerance decreases after a period of not using substances; the stay could have interrupted access to and continuity of treatments and supports (e.g. opioid agonist therapies) and, depending on the length of stay, they may be experiencing withdrawal, leading to higher-risk use; or the available substances and their toxicity could have changed.[35-37]

Hospitals and correctional facilities could strengthen their transition planning prior to discharge and connect people experiencing or at risk of being unhoused with evidence-based harm reduction, treatment, health care and housing services to prevent acute toxicity events. A high proportion of people without housing had contact with the health care system more broadly prior to death (through outpatient or inpatient care); these encounters provide opportunities to connect individuals to necessary health and social services.

While people who were unhoused and died of acute toxicity had known histories of substance use more often than people not identified as unhoused, their histories of substance use disorders and of any mental health conditions were similar. This finding may be because of a lack of recorded history for people who were unhoused due to limited access to health services or limited information available during death investigations. One in every two people without housing who died had

TABLE 4
Origin and contribution of other substances for substances that contributed to at least 10% of accidental deaths, by housing status at time of death, Canada, 2016–2017 (N = 7902)

Substances contributing to more than 10% of deaths	Unhoused (N = 702)						Not identified as unhoused (N = 6180)					
	Rank	Contribution to deaths, % (n)	Nonpharmaceutical origin, % (n)	Pharmaceutical origin, % (n)	Unknown origin, % (n)	Contributed with other substances, % (n)	Rank	Contribution to deaths, % (n)	Nonpharmaceutical origin, % (n)	Pharmaceutical origin, % (n)	Unknown origin, % (n)	Contributed with other substances, % (n)
Fentanyl*	1	60 (423)	51 (216)	1 (6)	48 (201)	89 (375)	1	47 (2895)	46 (1329)	4 (108)	50 (1461)	81 (2343)
Methamphetamine*	2	48 (336)	100 (336)	n/a	n/a	96 (321)	4	19 (1179)	100 (1179)	n/a	n/a	90 (1056)
Cocaine	3	32 (225)	100 (225)	n/a	n/a	92 (207)	2	36 (2196)	100 (2196)	n/a	n/a	84 (1848)
Amphetamine[a,*]	4	26 (183)	16 (30)	0	84 (153)	100 (183)	6	11 (684)	19 (129)	1 (9)	79 (543)	100 (684)
Ethanol (alcohol)	5	21 (150)	n/a	n/a	n/a	94 (141)	3	22 (1347)	n/a	n/a	n/a	86 (1158)
Morphine[a]	6	15 (105)	17 (18)	6 (6)	14 (15)	97 (102)	5	14 (855)	16 (138)	13 (111)	71 (606)	94 (804)
Diacetylmorphine (heroin)*	6	15 (105)	100 (105)	n/a	n/a	94 (99)	7	10 (597)	100 (597)	n/a	n/a	97 (579)

Abbreviation: n/a, not applicable.

[a] Amphetamine is a metabolite of methamphetamine and morphine is a metabolite of diacetylmorphine (heroin). Their presence in toxicology testing could indicate that either they or their parent substance had been consumed.

* Significantly different by housing status, chi-square test, $p < 0.05$.

TABLE 5
Exclusive substances and substance combinations[a] contributing to most of the accidental acute toxicity deaths, by housing status at time of death, Canada, 2016–2017 (N = 7902)

Unhoused (N = 702)			Top substances and substance combinations[b]	Not identified as unhoused (N = 6180)		
%	n	Rank		Rank	n	%
8	54	1	Fentanyl, methamphetamine and amphetamine	6	144	2
7	48	2	Fentanyl	1	552	9
5	33	3	Fentanyl and cocaine	2	354	6
3	24	4	Fentanyl and methamphetamine	15	63	1
3	18	5	Cocaine	3	345	6
3	18	5	Fentanyl, methamphetamine, cocaine and amphetamine	15	63	1
2	15	7	Methamphetamine	8	120	2
2	15	7	Methamphetamine and carfentanil	>17	21	0
2	15	7	No toxicology information available	12	78	1
2	15	7	Multiple drug toxicity	4	282	5
2	15	7	Fentanyl, methamphetamine, amphetamine, morphine and diacetylmorphine (heroin)	>17	27	0
2	12	12	Fentanyl and ethanol (alcohol)	7	135	2
2	12	12	Methamphetamine and amphetamine	17	57	1
2	12	12	Ethanol (alcohol)	5	186	3
2	12	12	Fentanyl, methamphetamine and diacetylmorphine (heroin)	>17	24	0
2	12	12	Methadone	9	117	2
2	12	12	Fentanyl, cocaine and ethanol (alcohol)	13	72	1

Notes: Amphetamine and morphine are active metabolites of methamphetamine and diacetylmorphine (heroin), respectively, and their presence in toxicology may be due to their consumption or the consumption of the parent substance. It is not possible to discern from this data source whether these substances were consumed intentionally or unintentionally. Counts are randomly rounded to base 3 and those <10 are suppressed. Proportions are based on rounded counts.

[a] Exclusive substances or substance combinations that contributed to at least 10 deaths of people who were unhoused.

[b] Opioids: carfentanil, diacetylmorphine (heroin), fentanyl, morphine, methadone. Stimulants: amphetamine, cocaine, methamphetamine.

a documented history of at least one potentially traumatic event during their lifetime, which was significantly higher than for people with housing. This highlights the need for accessible, inclusive and trauma-informed services, consistent with Canadian clinical guidelines, for the complex health and social circumstances of people without housing.[38]

For people not identified as unhoused, the acute toxicity event occurred in a personal residence three times out of four. Among people without housing, one-quarter of the acute toxicity events occurred in an outdoor setting. Substance use in an outdoor setting might increase the odds of a witness noticing a medical emergency; however, it may also result in practices to conceal use (rushing use or using larger amounts) to avoid law enforcement encounters and drug possession charges, and use in outdoor or public places has been associated with increased risk of experiencing an acute toxicity event.[25,26,39]

While people with both housing statuses were more often using substances alone prior to the acute toxicity event, people without housing were more likely than those not identified as unhoused to be using substances in the presence of others, presenting a higher potential for intervention. Harm reduction programs, housing services and law enforcement policies could promote safer environments for both shelter and substance use, where medical assistance and naloxone are more readily accessible. Improving service integration and availability of wraparound services within existing supports (e.g. shelters/housing services, harm reduction and treatment programs) may also reduce harms and improve outcomes for people without housing, who can have complex and interrelated health and social service needs.

The pattern of substances involved in death differed by housing status. Stimulants and opioids are accessible street drugs and use of these substances to cope with trauma and situational stressors has been described previously.[40,41] Use of stimulants such as methamphetamines may help people stay awake and alert when they are unsheltered and unsafe.[42,43] The several-hour longer half life and generally lower price may explain why methamphetamines, rather than cocaine, more commonly contributed to the death of people experiencing homelessness; cocaine was more commonly a contributor to death of people with housing.[44] Co-use of opioids and stimulants has been reported to calm down a person after using a stimulant, to alleviate withdrawal symptoms or paranoia from a stimulant, to avoid feeling drowsy when using an opioid, to create a pattern of successive stimulation and sedation or to balance the effects of each substance.[45] Alternatively, the presence of both these substances may be unintentional and a result of contamination. Consulting with people who are or at risk of being unhoused about the substances they use and their patterns of use could inform health promotion and harm reduction

services, including safer supply options. Knowing what substances are causing harm can also help tailor training for first responders and bystanders responding to acute toxicity events.

Strengths and limitations

Both the national and chart review study estimates of unhoused people are based on point-in-time counts (for national estimates, it is the day of the count; for the chart review study, it is the day of death), and do not include all people who are unhoused in a community over a period of time. People often cycle in and out of being unhoused, and those who are temporarily staying with friends or family were less likely to be identified during the count.[46,47] People who are experiencing housing insecurity or who are at immediate risk of being unhoused were also less likely to be identified during the count.

The purpose of death investigations is to establish the cause and manner of death and, in some cases, to provide recommendations to prevent deaths of a similar nature in the future. Therefore, coroners and medical examiners are not seeking some of the variables of interest to this study. Death investigation protocols and methods of data collection and the availability of certain variables vary across the country. For example, binary sex was always available, but gender identity, rarely. In addition, less information may be collected during death investigations of people who were unhoused because it can be difficult to identify witnesses, friends, family members or service providers who can speak to the personal histories of those who died. In death investigation files, it is not always clear whether someone was living with a friend or family member because of housing insecurity. Being unhoused, histories of substance use, mental health conditions and symptoms, and potentially traumatic experiences are all likely underreported in the chart review study; thus, the data represent the minimum proportions of people who had these experiences. The differences between people who were unhoused at the time of their death and those not identified as being unhoused may be underestimated due to misclassification of people if information was absent from the death investigation.

Conclusion

This study identifies potential opportunities to reduce accidental acute toxicity deaths among people who are unhoused, including during contacts with health care services and through strengthening transition planning prior to release from institutions, taking into account the need for accessible, inclusive and trauma-informed services and improving service integration within existing supports, creating safer environments for shelter and substance use, and tailoring health promotion and harm reduction services to their specific needs.

Since the study period, the COVID-19 pandemic has contributed to an increase in the number of people who are unhoused and increased many barriers to services for them.[48] Research on the current relationships between housing status and substance-related harms and engagement with people with lived and living experience of being unhoused would be valuable to advance policies and programs to prevent further accidental acute toxicity deaths.

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Conflicts of interest

None to declare.

Authors' contributions and statement

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At-a-glance

A comparison of the characteristics of accidental substance-related acute toxicity deaths in Canada across life stages, 2016–2017

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Abstract

The acute toxicity (sometimes called "overdose" or "poisoning") crisis has affected Canadians across all stages of life, including youth, adults and older adults. Our biological risks and exposures to substances change as we age. Based on a national chart review study of coroner and medical examiner data on acute toxicity deaths in 2016 and 2017, this analysis compares the burden of deaths and circumstances of death, locations of acute toxicity event and death, health history and substances contributing to death of people, by sex and life stage.

Keywords: *substance use, acute toxicity deaths, youth, adults, older adults, Canada*

Highlights

- This analysis reveals key differences in the characteristics of acute toxicity deaths by sex and life stage, and suggests potential intervention points for each group.
- Many people across demographics were alone while using substances before the acute toxicity event, and many were alone when they died. Youth, particularly female youth, more often died in circumstances where someone might have been available to help by calling 911 or administering first aid and naloxone.
- For the people who were in contact with health care prior to their death, about one-quarter (24%–28%) of adults and older adults sought assistance for reasons related to pain. Youth more often sought assistance for a nonfatal acute toxicity event (13%–14%) or for mental health (particularly female youth, 21%) than people in other life stages.
- Multiple substances contributed to most deaths, and both pharmaceutical and nonpharmaceutical substances were common causes of death for all life stages and sexes. There are demographic differences in the specific substances contributing to death.

Introduction

The acute toxicity (sometimes called "overdose" or "poisoning") crisis has affected Canadians from all walks of life and of all ages—children, youth, adults including older adults have died. At the population level, our biological risks from substance use change as we age: our brains are not fully developed until our mid-20s;[1] over time we can accumulate more diseases and disorders;[2] and eventually our metabolism and ability to process substances slows down.[3] Our exposures to substances also evolve with age: first exposures to nonmedical substance use are often in our youth;[4] peer pressure to engage in nonmedical substance use changes over time;[4] and we are more likely to have multiple prescriptions in later life.[5]

In this analysis, we compare characteristics of acute toxicity deaths across life stages for youth, adults and older adults. This analysis serves as an important baseline at the beginning of the acute toxicity crisis that can be used to measure change. It is intended to bridge previously published in-depth reports on youth[6] and older adults,[7] and compares broader life stages rather than the 5- or 10-year age groupings other reports use based on the same dataset.[8,9]

Methods

This study was reviewed and approved by the Public Health Agency of Canada Research Ethics Board (REB 2018-027P), the University of Manitoba Health Research Ethics Board (HS22710) and the Newfoundland and Labrador Health Research Ethics Board (20200153).

For the purposes of this study, life stages are defined as youth (aged 12 to 24 years),

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adults (aged 25 to 59 years) and older adults (aged 60 plus years). Substances include alcohol, pharmaceutical and non-pharmaceutical drugs and chemicals not approved for human consumption (e.g. illegal drugs, nonpharmaceutical inhalants, industrial or household chemicals, or veterinary drugs). Based on a national retrospective chart review study of coroner and medical examiner data on all substance-related acute toxicity deaths from 1 January 2016 to 31 December 2017,[8,10] we calculated the burden of accidental substance-related acute toxicity deaths and characteristics of people who died by sex and life stage. Table 1 lists the variables used in the analysis and their descriptions.

Burden is based on the number of deaths, mortality rate and proportionate mortality ratio due to accidental substance-related acute toxicity. Mortality rates were calculated with population counts from the 2016 Census[11] as the denominator. To calculate the proportionate mortality ratios attributable to substance-related acute toxicity, we used data from Statistics Canada on all-cause accidental mortality counts by demographic group for the denominators. We included all-cause deaths with ICD-10 codes V01–V99 (transport accidents), W00–X59 (other external causes of accidental injury), Y85 (sequelae of transport accidents) and Y86 (sequelae of other accidents).

We also analyzed the circumstances of death, locations of the acute toxicity event and death, health history and substances contributing to death of people who died of accidental acute toxicity, by sex at birth and life stage, using the variables described in Table 1. We calculated the proportions of each group that had a given characteristic, and conducted Pearson chi-square tests to assess statistical differences across life stages and sex ($p < 0.05$). As information on the variables of interest are not always recorded in death investigation files, the results represent only the minimum proportions of people who had a given characteristic.

To protect privacy, all counts are randomly rounded to base 3 (i.e. values had different chances of being rounded to nearest multiples of 3) and counts less than 10 are suppressed.[10] Since table totals were also independently rounded to base 3, the sum of values do not always equal the total. Proportions and mortality rates are calculated with rounded counts.

Results

Each of these demographic groups has been affected by the acute toxicity crisis in different ways. Acute toxicity accounted for 41% to 60% of all accidental deaths for youth and adults (Table 2). The mortality rate due to accidental acute toxicity was much higher for male adults (30 deaths per 100 000 population) than the other demographic groups (2.8–9.5 deaths per 100 000 population). Among the people who died of accidental acute toxicity, contacts with health care, circumstances of death and substances contributing to death varied by life stage and sex.

Circumstances of accidental acute toxicity deaths

- Older adults were less often using substances in the presence of others prior to their death (12%–14% vs. 16%–28%).
- Older adults were more often already dead when found compared with youth and adults (38%–39% vs. 19%–29%).
- Many people were found in or near a bed (24% to 39%) where their acute toxicity event could have been misinterpreted as sleep. Females were more often found in or near a bed.
- Among people who were reported to show signs of opioid toxicity before death, naloxone was less often administered to older adults (counts and proportions suppressed due to small numbers).
- For all life stages, the most frequent location for the acute toxicity event leading to death was the individual's personal residence (59%–87%). Of those who had their acute toxicity event in their personal residence, older adults more commonly lived alone (31%–32% vs. 16% or less).
- Though less common across all life stages, youth and adults were more often at the home of another person compared to older adults (14%–16% and 9%–10%, respectively, vs. 5% or less).
- Most people died where the acute toxicity event happened (68%–84%). Female youth were most often transported to hospital before death (26%), and male older adults were least often transported to hospital (10%).

Health history and previous contacts with the health system of people who died of accidental acute toxicity

- Most people who died had a history of substance use (excluding alcohol). This was less common for female older adults (55%) than other demographic groups (71%–83%).
- Female older adults also had a history of substance use disorders (excluding alcohol) less frequently than other demographic groups (10% vs. 18%–22%).
- The frequency of alcohol use disorders increased with age (5%–6% among youth to 12%–15% among older adults).
- Male youth (42%) and female older adults (43%) had a history of chronic (daily) substance use less often than other demographic groups (48%–59%).
- Having a history of depression or depressive symptoms and of anxiety disorders was more frequent among females than males (29%–39% and 19%–22%, respectively, vs. 19%–22% and 11%–14%, respectively).
- Contact with health care services (inpatient or outpatient) in the year prior to death was more common with increased age (58%–61% for youth to 80%–91% for older adults).
- For the people who were in contact with health care prior to their death, there were no demographic differences in seeking care for substance use and/or addictions. About one-quarter (24%–28%) of adults and older adults sought assistance for reasons related to pain. Youth more often sought assistance for a nonfatal acute toxicity event (13%–14%) or for mental health (particularly female youth, 21%) than people in other life stages.

Substances causing accidental acute toxicity deaths

- A pharmaceutical substance contributed to death most often among female older adults (63% vs. 28%–46%).

TABLE 1
Variables used to describe the burden of substance-related acute toxicity deaths and the characteristics of people who died, by sex and life stage, Canada, 2016–2017

Variable	Description
Burden	
Number of accidental acute toxicity deaths	A count of accidental substance-related acute toxicity deaths.
Mortality rate due to accidental acute toxicity per 100 000 population	The number of deaths for every 100 000 people in that population. Controls for differences in the number of people that fall into each demographic category.
Proportionate mortality ratio due to accidental acute toxicity	The proportion of all-cause accidental mortality that is due to accidental acute toxicity.
Circumstances of death	
Was using substances in the presence of others	The person who died consumed substances in the presence of others prior to the fatal acute toxicity event, i.e. the substance use was witnessed.
Deceased when found	There was no known witness to the fatal acute toxicity event and no intervention was possible when the person who died was found.
Found in or near a bed	The person who died was in or near where they could have been thought to be sleeping. A perception that the person was sleeping, and not unconscious, could have delayed a response.
Had signs of opioid toxicity	A bystander or first responder who witnessed the fatal acute toxicity event observed one or more signs of opioid toxicity. These include snoring/gurgling, difficulty breathing, pinpoint pupils, unconsciousness or unresponsiveness, or blue lips/fingernails/face. As toxicology information is not immediately available, bystanders and first responders use toxidromes, or symptoms of toxicity, to determine the substance(s) causing the acute toxicity and how to respond.
Naloxone was given	The count as well as the proportion of those with signs of opioid toxicity who were given naloxone, an antidote for opioid toxicity. This includes naloxone given by bystanders, EMS, police, fire, hospital staff or others.
Place of acute toxicity event	The fatal acute toxicity event occurred in the home of the person who died, in the home of another person or in another location.
Lived alone	Of those whose fatal acute toxicity event occurred in their own home, those who lived alone. Those who lived alone may have been less likely to have someone nearby to help.
Place of death	Whether the person was transported from the location of the acute toxicity event to a hospital or another location before they died, or if they died in the same place as the acute toxicity event.
Health history [a]	
History of substance use (excluding alcohol)	The death investigation file includes information that the person had a history of substance use, excluding the use of alcohol or the use of pharmaceuticals as prescribed to them.
History of substance use disorder (excluding alcohol)	The death investigation file explicitly states that the person had a substance use disorder.
History of alcohol use disorder	The death investigation file explicitly states that the person had an alcohol use disorder.
History of chronic (daily) substance use	The death investigation file mentions that the person used substances chronically (i.e. daily).
Lifetime history of a nonfatal acute toxicity event	The death investigation file includes a record of a previous nonfatal acute toxicity event (overdose).
History of depression or depressive symptoms	The death investigation file includes information that the person had a depressive disorder or depressive symptoms.
History of anxiety disorder	The death investigation file includes information that the person had an anxiety disorder.
Contact with health care in the previous year	The person had contact with health care services in the year prior to their death. This could be outpatient or inpatient services.
Sought assistance for…	For those who had contact with health care services in the year prior to their death, the death investigation file describes a specific reason the person sought health care services, e.g. pain, a nonfatal acute toxicity event, substance use and/or addictions, or mental health.

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TABLE 1 (continued)
Variables used to describe the burden of substance-related acute toxicity deaths and the characteristics of people who died, by sex and life stage, Canada, 2016–2017

Variable	Description
Substances contributing to death	
Origin of substances contributing to death	Origins of substances are categorized into: • nonpharmaceutical ("street drugs" and substances not intended for human use, e.g. industrial or household chemicals or veterinary medications); • pharmaceutical (produced for human use by a regulated pharmaceutical manufacturer); • ethanol (originating in the alcoholic beverage industry or home-distilled alcohol and does not fall under the other origin categories), or • unknown (insufficient evidence to determine the origin of the substance). A substance can have multiple origins. Deaths due solely to prescribed substances or alcohol were not available from British Columbia.
At least one of the pharmaceutical drugs was prescribed	Of the people who had at least one pharmaceutical drug contribute to their death, those who had been prescribed at least one of these drugs.
Substances most often contributing to death	Specific substances that contributed to at least 10% of deaths for one of the demographic groups.
Multiple toxicity, no substances specified	Deaths with a cause of death describing multiple substances contributing to the death, but not listing the specific contributing substances.
Multiple substances contributed to death	Deaths where more than one substance was identified as a contributor to the death.

Abbreviation: EMS, emergency medical services.

[a] Abstractors included any information about health history in the file, including medical records or statements from family or friends. The conditions reported may not have been clinically diagnosed.

- A nonpharmaceutical substance contributed to death most often among youth and male adults (72%–74%).
- Youth had a prescription for pharmaceutical drugs that contributed to their deaths less often than other groups (16%–18% vs. 40%–66%).
- Multiple substances contributed to most deaths (55%–72%).
- The most common substances contributing to death in youth were similar for both sexes (fentanyl, cocaine, methamphetamine, ethanol [alcohol] and amphetamine), but there were sex differences for the other age groups. For example, fentanyl contributed to a greater proportion of male adult deaths (53%) than female adult deaths (36%) and to male older adult deaths (32%) than female older adult deaths (16%).
- Fentanyl was a cause of more than half of fatal acute toxicity events of youth (55%–57%) and male adults (53%).

Discussion

This analysis reveals key differences in the characteristics of acute toxicity deaths by sex and life stage and suggests potential intervention points for each group. Many people who died of acute toxicity had contact with health care in the year prior to their death. These encounters with the health care system provide earlier opportunities to identify and address the risk of a fatal acute toxicity event as well as unmet health and social needs that may contribute to substance use. About one in four adults and older adults were in contact with health care for reasons related to pain. Such contacts create an opportunity for discussions regarding pain management, including safe use of pain medications, seeking relief from other substances, and other available treatment options and services to help alleviate pain.

Youth, particularly female youth, more often died in circumstances where someone might have been available to help by calling 911 or administering first aid and naloxone (Table 2). It is important that potential witnesses to acute toxicity events be able to recognize and respond to the emergency, and have the right tools to help (e.g. a naloxone kit, a phone to call 911). Many people across demographics were alone while using substances before the acute toxicity event, and many were alone when they died. Removing the stigma of substance use is important so that those who are using substances alone can find greater safety with others. Supporting connections to laypeople trained in overdose prevention or formalized supervised consumption services could help prevent these deaths.

Multiple substances contributed to most deaths, and both pharmaceutical and nonpharmaceutical substances were common causes of death for all life stages and sexes (Table 2). When a pharmaceutical substance contributed to death, many people, and particularly older adults and female adults, had been prescribed the substance that caused their death. The involvement of multiple substances in an acute toxicity event is the norm, and the potential combined harms of substances are an important consideration for prescribing practices (e.g. management of multiple prescriptions), patient education, harm reduction programs, drug checking services and drug alerts.

In this study, we were unable to differentiate between whether the multiple substances involved were intentionally or unintentionally consumed. Initiatives to address the toxic drug supply would benefit all demographics, as would a harm reduction approach to prescribing that emphasizes patient education about the risks of their prescription drugs, their risks in combination with other substances and the risks of diversion.[12,13]

TABLE 2
Burden, circumstances of death, documented health history and substances contributing to the deaths of people who died of accidental acute toxicity, by sex and life stage, Canada, 2016–2017

	Youth (12–24 years)		Adults (25–59 years)		Older adults (≥60 years)	
	Female	Male	Female	Male	Female	Male
Total accidental acute toxicity deaths	**N = 207**	**N = 525**	**N = 1563**	**N = 4896**	**N = 246**	**N = 462**
Mortality rate due to accidental acute toxicity, per 100000 population	3.9	9.5	9.2	30	2.8	6.0
Proportionate mortality ratio, %	42	41	59	60	3.1	5.9
Circumstances of death, % (n)						
Was using substances in the presence of others*	28 (57)	19 (102)	20 (315)	16 (783)	12 (30)	14 (63)
Deceased when found*	19 (39)	24 (126)	28 (435)	29 (1419)	39 (96)	38 (177)
Found in or near a bed*	35 (72)	30 (156)	35 (543)	24 (1191)	39 (96)	24 (111)
Had signs of opioid toxicity[a]	36 (75)	30 (159)	33 (519)	26 (1263)	27 (66)	21 (96)
Naloxone was given*	40 (30)	34 (54)	22 (114)	28 (357)	Suppressed	Suppressed
Place of acute toxicity event*, % (n)						
Personal residence	59 (123)	61 (321)	70 (1092)	62 (3021)	87 (213)	77 (354)
Lived alone	Suppressed	7 (23)	16 (179)	15 (465)	32 (69)	31 (111)
Home of another person	16 (33)	14 (75)	10 (162)	9 (453)	Suppressed	5 (21)
Other	23 (48)	25 (129)	20 (309)	29 (1419)	10 (24)	18 (84)
Place of death*, % (n)						
Same as place of acute toxicity event	68 (141)	71 (375)	71 (1113)	76 (3735)	78 (192)	84 (390)
Hospital [b]	26 (54)	22 (114)	22 (339)	17 (825)	17 (42)	10 (48)
Other	6 (12)	7 (39)	7 (111)	7 (333)	5 (12)	5 (24)
Health history, % (n)						
History of substance use (excluding alcohol)*	83 (171)	81 (426)	78 (1215)	83 (4083)	55 (135)	71 (330)
History of substance use disorder (excluding alcohol)*	20 (42)	20 (105)	22 (339)	20 (966)	10 (24)	18 (81)
History of alcohol use disorder*	6 (12)	5 (27)	9 (135)	9 (426)	12 (30)	15 (69)
History of chronic (daily) substance use*	48 (99)	42 (219)	50 (783)	54 (2646)	43 (105)	59 (273)
Lifetime history of a nonfatal acute toxicity event	17 (36)	17 (87)	15 (234)	12 (585)	15 (36)	9 (42)
History of depression or depressive symptoms*	29 (60)	19 (99)	33 (516)	20 (963)	39 (96)	19 (90)
History of anxiety disorder*	22 (45)	14 (72)	19 (300)	11 (528)	21 (51)	11 (51)
Contact with health care in the previous year*, % (n)	**61 (126)**	**58 (306)**	**80 (1254)**	**66 (3249)**	**91 (225)**	**80 (369)**
Sought assistance for pain*	14 (18)	17 (51)	26 (321)	25 (810)	24 (54)	28 (105)
Sought assistance for a nonfatal acute toxicity event*	14 (18)	13 (39)	7 (90)	8 (249)	7 (15)	7 (24)
Sought assistance for substance use and/or addictions	19 (24)	17 (51)	13 (165)	14 (444)	9 (21)	9 (33)
Sought assistance for mental health*	21 (27)	14 (42)	13 (165)	11 (342)	11 (24)	6 (21)
Origin of substances contributing to death[c], % (n)						
At least 1 nonpharmaceutical*	72 (150)	73 (384)	55 (861)	74 (3624)	18 (45)	55 (255)
At least 1 pharmaceutical*	32 (66)	29 (150)	46 (720)	28 (1347)	63 (156)	40 (186)
At least 1 of the pharmaceutical drugs was prescribed*	18 (12)	16 (24)	56 (402)	40 (537)	66 (99)	56 (102)
Substances most often contributing to death, % (n)						
Fentanyl*	57 (117)	55 (291)	36 (555)	53 (2562)	16 (39)	32 (150)
Cocaine*	28 (57)	30 (156)	30 (474)	39 (1887)	10 (24)	36 (165)
Methamphetamine*	22 (45)	17 (87)	22 (339)	24 (1170)	5 (12)	11 (51)
Ethanol (alcohol)*	16 (33)	15 (81)	21 (330)	23 (1107)	18 (45)	26 (120)
Amphetamine*,[d]	13 (27)	11 (57)	7 (117)	14 (678)	Suppressed	6 (30)
Morphine[d]	12 (24)	14 (72)	14 (219)	14 (696)	11 (27)	17 (78)

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TABLE 2 (continued)
Burden, circumstances of death, documented health history and substances contributing to the deaths of people who died of accidental acute toxicity, by sex and life stage, Canada, 2016–2017

	Youth (12–24 years)		Adults (25–59 years)		Older adults (≥60 years)	
	Female	Male	Female	Male	Female	Male
Alprazolam*	10 (21)	10 (51)	2 (27)	2 (78)	Suppressed	Suppressed
Diacetylmorphine (heroin)*	6 (12)	12 (63)	6 (99)	12 (606)	Suppressed	6 (30)
Methadone*	6 (12)	7 (36)	11 (168)	8 (378)	5 (12)	8 (39)
Multiple toxicity, no substances specified*	Suppressed	3 (18)	8 (120)	4 (201)	15 (36)	6 (27)
Oxycodone*	Suppressed	6 (30)	7 (114)	5 (258)	12 (30)	6 (30)
Multiple substances contributed to death	68 (141)	64 (336)	72 (1122)	71 (3480)	55 (135)	64 (297)

Sources: National chart review study of substance-related acute toxicity deaths (2016 to 2017)[8,10]; 2016 Census.[11]

Notes: Deaths due solely to prescribed substances or alcohol were not available from British Columbia and all numbers in this table may be underestimates. The denominator for each group is from the 2016 Census.[11] The all-cause mortality counts by demographic group used to calculate the proportion of the mortality rate for all causes due to acute toxicity were provided by Statistics Canada. All accidental deaths include ICD-10 codes V01–V99, W00–W99, X00–X59, Y85 and Y86.

To protect privacy, counts from the national chart review study of substance-related acute toxicity deaths were randomly rounded to base 3, and proportions and rates were based on randomized counts. Counts <10 and the proportions and rates based on counts <10 are suppressed. Test statistics and exact *p* values are not shown to protect the random rounding.

[a] Signs of opioid toxicity include snoring/gurgling, difficulty breathing, pinpoint pupils, unconscious or unresponsive, or blue lips/fingernails/face.

[b] The "hospital" category includes only the people who were transported to hospital from another location. If an acute toxicity event leading to death occurred in a hospital and the person died in hospital it was categorized as "same as place of acute toxicity event." Less than 1% of fatal acute toxicity events occurred in a hospital. Notably, most of the fatal acute toxicity events that occurred in hospitals were among male adults (33 of 45 events, not shown).

[c] Origins of substances are categorized into: nonpharmaceutical ("street drugs" and substances not intended for human use, e.g. industrial or household chemicals or veterinary medications); pharmaceutical (produced for human use by a regulated pharmaceutical manufacturer); ethanol (originating in the alcoholic beverage industry or home-distilled alcohol, neither of which belong in the other origin categories); or unknown (insufficient evidence to determine the origin of the substance). A substance can have multiple origins.

[d] Amphetamine and morphine are active metabolites of other substances that may have contributed to death. Amphetamine is a metabolite of methamphetamine and morphine is a metabolite of heroin. The presence of these substances in toxicology testing may be because the parent substance was consumed rather than the substance itself.

* $p < 0.05$.

Strengths and limitations

A chart review of death investigation data allowed for more detailed analysis of patterns in substance-related acute toxicity deaths for different demographic groups. This is particularly true for the circumstances surrounding the death, as there is limited contextual information captured in other reporting systems.

Death investigation protocols vary across the country, and information about these variables is not always consistently available in death investigation files. Age, sex and manner of death were complete for all records, but for other characteristics, these are the minimum proportions of people who died of substance-related acute toxicity that had a given characteristic, and may underestimate the true number.

Conclusion

Acute toxicity is a major cause of accidental deaths among youth and adults in Canada, and entirely preventable. Contextual information from coroner and medical examiner files, even where some of the information we seek is missing, reveals patterns and potential opportunities to prevent further acute toxicity deaths for specific demographic groups, including through focused interventions across the life course. These patterns may have changed since the study period of 2016 to 2017, particularly during the COVID-19 pandemic, but these results serve as an important baseline to measure the impacts of interventions implemented in the intervening years.

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Conflicts of interest

The authors report no conflicts of interest.

Authors' contributions and statement

GC: Conceptualization, data curation, formal analysis, writing – original draft, writing – review & editing.

JH: Conceptualization, data curation, formal analysis, writing – original draft, writing – review & editing.

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Original quantitative research

Social media use and sleep health among adolescents in Canada

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Abstract

Introduction: Public health concerns over the impact of social media use (SMU) on adolescent health are growing. We investigated the relationship between SMU and sleep health in adolescents in Canada aged 11 to 17 years.

Methods: Data from the 2017–2018 Health Behaviour in School-aged Children study were available for 12 557 participants (55.2% female). SMU was categorized by frequency of use (non-active, active and intense) and the presence of addiction-like symptoms (problematic). Mixed effects logistic regression models identified associations between SMU and seven sleep health indicators (insomnia symptoms, daytime wakefulness problems, screen time before bed, meeting sleep duration recommendations, sleep variability and late bedtime on school and non-school days).

Results: Compared to active SMU, non-active SMU was associated with better sleep indicators, except for insomnia symptoms. Intense SMU was associated with greater odds of having poor sleep health indicators (adjusted odds ratio [aORs] from 1.09 to 2.24) and problematic SMU with the highest odds (aORs from 1.67 to 3.24). Associations with problematic SMU were greater among girls than boys, including having a later bedtime on school days (aOR = 3.74 vs. 1.84) and on non-school days (aOR = 4.13 vs. 2.18). Associations between SMU and sleep outcomes did not differ by age group.

Conclusion: Intense and problematic SMU were associated with greater odds of poor sleep health among adolescents in Canada, with stronger associations among girls than boys. Further research is needed to understand the mechanisms underlying associations between SMU and sleep to inform public health recommendations.

Keywords: *adolescents, social media use, sleep, insomnia, daytime wakefulness, sleep duration, sleep variability*

Highlights

- Intense and problematic social media use were both associated with worse sleep health compared to active social media use.
- The highest odds of having poor sleep health indicators were associated with problematic social media use (adjusted odds ratios from 1.67 to 3.24) assessed using the Social Media Disorder Scale.
- Non-active social media use was linked to better sleep health.
- Associations between poor sleep health indicators and social media use were stronger among girls than boys.
- Across social media use categories, odds ratios for having poor sleep health indicators did not differ by age group.

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Introduction

Social media use (SMU), defined as the time spent on social media platforms (e.g. Facebook, Twitter, TikTok, etc.) to connect with other users and exchange user-generated content, is an integral part of adolescents' lives around the world.[1,2] In the United States, the percentage of adolescents reporting intense SMU (i.e. being online almost constantly) increased from 25% to 45%.[3] One cross-national study estimated that 40% of adolescents aged 15 to 19 years increased their SMU during the COVID-19 pandemic.[4]

In the current literature, a distinction is made between intense and problematic SMU. Intense SMU is defined as spending a lot of time on social media, whereas problematic SMU implicates the presence of behavioural and psychological symptoms of addiction that affect daily functions.[2,5] While social media offers opportunities to strengthen friendships, promote social support and reduce social isolation, intense and problematic SMU may negatively impact youth health and well-being, including sleep.[2,5,6]

Sleep is essential to the health and development of adolescents and a contributor to their well-being through its influence on learning capacities, emotional regulation and memory processes.[7] Sleep health encompasses not just sleep duration but also sleep quality, regularity, satisfaction, appropriate sleep timing alertness during the day, and sleep-facilitating behaviours.[8,9] These components of sleep have been proposed in a sleep health framework called Peds B-SATED (Behaviour, Satisfaction/Quality, Alertness/Sleepiness, Timing, Efficiency and Duration).[9] In Canada, one in three children and adolescents do not meet sleep duration recommendations,[10,11] and at least 25% have symptoms of insomnia and daytime wakefulness problems[10]

Numerous studies have linked SMU with poor sleep health in adolescents.[12,13] SMU is hypothesized to impact sleep via four mechanisms: (1) exposure to blue light, which affects circadian timing; (2) psychophysiological activation due to the emotional content of social media; (3) the "never-ending" nature of SMU; and (4) the constant alerts that disturb sleep.[2,6,14] Studies linking SMU to sleep have generally focused on one aspect of sleep, most often sleep duration, but interest is growing in understanding the association between SMU and other aspects of adolescent sleep, such as sleep quality and sleep-facilitating behaviours.[8,9,15] Yet, according to a 2019 census on SMU in children and youth aged 8 to 18 years in the United States, only 14% reported that their parent monitored their time spent on social media.[16]

Associations between SMU and sleep may vary by gender and age. A study of adolescents in the United States and the United Kingdom found that the association between time spent on social media and lower well-being was greater among girls than boys.[17] Another study also identified significant differences in SMU by age group, with 11-year-olds reporting less intense SMU than 13- and 15-year-olds and significantly better mental health across most measures,[2] suggesting a need to look at age differences more closely. Studying gender and age differences in the associations between SMU and different sleep health indicators may offer a clearer picture of the factors in the relationship between SMU and sleep.

The aim of the current study is to investigate the association between SMU and sleep health indicators among adolescents in Canada and to examine any gender and age differences. We examined SMU with a previously developed scale[2] that combines intensity and problematic symptoms. We hypothesized that intense and problematic SMU would be associated with worse sleep health, compared to active SMU, and that associations would be stronger in girls than boys and in older than younger adolescents.

Methods

Data and participants

Data were from the 2017–2018 Health Behaviour in School-aged Children (HBSC) study, a cross-national research study and World Health Organization collaboration, that collects data every 4 years from a representative sample of students in Grades 6 to 10 in the school setting. The Canadian part of the survey used a random two-stage cluster sample of students from all provinces and two territories (Yukon and Northwest Territories). Data were collected between January and May of 2018. Participation was voluntary and anonymous. The Canadian HBSC study obtained student assent and active and/or passive parental consent, depending on school board requirements.

The General Research Ethics Board at Queen's University (GMISC-062-13) and the Health Canada–Public Health Agency of Canada Research Ethics Board provided ethics approval.

A total of 21 745 students from 287 schools participated in the survey. For this study, we excluded adolescents in Grade 5 (n = 40) and Grade 11 (n = 163) because the HBSC survey is representative of students in Grades 6 to 10. We excluded adolescents who responded to "neither term describes me" for gender (n = 325) because of the small sample size, and those with information missing on SMU (n = 6226) and on the variables in our analyses (n = 2434), resulting in a final sample of 12 557 students with complete data.

Measures

Social media use

To assess SMU intensity, the survey asked participants to identify how often they had online contact with the following four categories of people: close friends; friends from a larger friend group; friends they met through the Internet; and other people (such as classmates, siblings or teachers). Response options were: "never or almost never," "at least every week," "daily or almost daily," "several times each day" and "almost all the time throughout the day." The highest frequency reported across the four categories was used to establish three levels of SMU intensity: (1) non-active (never or at most weekly); (2) active (daily/several times a day); and (3) intense (almost all the time), as previously described by Boniel-Nissim et al.[2]

We assessed problematic SMU using the Social Media Disorder Scale.[18] The scale has previously demonstrated appropriate validity in a large international sample of adolescents.[19] The scale includes nine "yes/no" items that identify addiction-like symptoms of SMU during the past year (e.g. having conflict with family, lying about the amount of time spent on social media, feeling bad when cannot use social media, among others). Participants who responded yes to six or more items were classified as problematic users, regardless of their SMU intensity level.[2]

Participants were classified into one of four mutually exclusive categories: non-active SMU (non-active SMU and non-problematic use); active SMU (active SMU and nonproblematic use); intense SMU (intense SMU and nonproblematic use); and problematic SMU (problematic use regardless of SMU intensity).[2]

Sleep health

We investigated seven indicators of sleep health, based on availability in the dataset: insomnia symptoms, daytime wakefulness, screen time before bed, sleep duration, sleep variability, and sleep timing on school days and on non-school days (weekends and holidays). Many of these sleep health measures align with the Peds B-SATED framework, with four of the six domains included.[9] We were unable to investigate sleep satisfaction/quality and efficiency with the current data source.

Insomnia symptoms

Participants were asked how often they have trouble going to sleep or staying asleep. There were five response options: "never," "rarely," "sometimes," "most of the time" and "all the time." The variable was dichotomized as those with insomnia symptoms ("most of the time" and "all the time") and those without ("never," rarely" and "sometimes"), in line with previous work.[20]

Problems with daytime wakefulness

Participants were asked how often they have trouble staying awake during the daytime when they want to be awake. There were five response options: "never," "rarely," "sometimes," "most of the time" and "all the time." "Never" and "rarely" were grouped to create a dichotomous variable defined as no daytime wakefulness problems.

Screen time before bed

To assess sleep-facilitating behaviours, the survey asked participants how often they watched television or used a cellphone or computer/tablet in their bedroom in the last hour before going to sleep. There were five responses options: "never," "1 or 2 nights a week," "3 or 4 nights a week," "5 or 6 nights a week" and "every night." Participants who responded "never" or "1 or 2 nights a week" were categorized as using screens before bed less than 2 nights a week, and all the other participants were categorized as using screens before bed 3 or more nights a week.

Sleep duration

To assess sleep duration, the survey asked participants when they usually go to bed and when they usually wake up, on school days and non-school days (weekends and holidays), separately. Participants could answer within 15-minute increments. Sleep duration on school days and non-school days was calculated and used to determine average daily sleep duration, which was compared to sleep duration recommendations for adolescents. Sleep duration recommendations differ depending on age group: 9 to 11 hours per night for 11- to 13-year-olds and 8 to 10 hours per night for 14- to 17-year-olds.[21] Taking into account sleep duration recommendations by age, we separated participants into two categories, meeting the sleep duration recommendation or not meeting the sleep duration recommendation.

Sleep variability

To assess sleep variability/regularity, we calculated for each participant if there was more than a 2-hour difference between bedtime during the week and weekend nights.[22] Participants with less than a 2-hour difference between bedtime during the week and weekend nights were categorized as having little or no sleep variability.

Sleep timing

As wake times can largely depend on school start times, we used bedtimes as an indicator of sleep health. We calculated bedtime tertiles on school and non-school days for each age category (11–13 years and 14–17 years), given the shift towards a later bedtime during adolescence due to biological changes in circadian rhythms. We then categorized participants as having an early/moderate bedtime (first and second tertile) or a late bedtime (third tertile) on school days in comparison with peers. For those aged 11 to 13 years, late bedtimes on school and non-school days were after 10:30 p.m. and 12:00 a.m., respectively. For youth aged 14 to 17 years, late bedtimes on school and non-school days were after 12:00 a.m. and 1:00 a.m., respectively.

Sociodemographic variables

We included information on gender (boy/girl), cultural/ethnoracial background (categorized as White vs. non-White) and family affluence. Family affluence was measured using the Family Affluence Scale, a reliable and valid measure of socioeconomic status.[23] The FAS is a composite score based on household characteristics, including the number of cars, bathrooms, computers, having an unshared bedroom and the number of family holidays abroad during the past year. The responses were summed and categorized into three groups (0–6: "low affluence"; 7–10: "medium affluence"; and 11–13: "high affluence").[23]

Statistical analysis

We first conducted descriptive analyses of the sample across the four SMU categories. To examine the associations between the four SMU categories and sleep variables, we used mixed effects logistic regression models, with separate models for each of the seven sleep health outcomes. From the logistic regression models, we report both the odds ratio and 95% confidence interval (CI). All models were adjusted for gender, cultural/ethnoracial background, age and family affluence.

We then conducted additional exploratory analyses to examine gender and age differences by rerunning the models stratified by gender and age group. All models were controlled for clustering by schools using mixed effects models, and survey weights were applied to ensure results were representative of Grade 6 to 10 students in Canada. An alpha value of 0.05 was used to detect statistically significant results.

We conducted analyses in SAS Enterprise Guide version 7.1 (SAS Institute Inc., Cary, NC, US).

Results

Descriptive characteristics

The most common SMU category was active users (43.7%) followed by intense users (35.4%), non-active users (14.2%) and problematic users (6.7%). Problematic and intense users were generally more likely to be girls and non-White compared with active users. Conversely, non-active users were generally more likely to be boys and younger than the active SMU groups (Table 1).

At 9.0 (95% CI: 8.9–9.1) hours per night, 11- to 13-year-olds had a significantly longer mean sleep duration than 14- to 17-year-olds (8.1; 95% CI: 8.0–8.3 hours per night; $p < 0.001$) (data not shown). We also found more 11- to 13-year-olds

TABLE 1
Descriptive characteristics of the sample overall and by social media use category (n = 12 557)

Characteristic	Social media use, weighted % or mean (95% CI)				
	Total (n = 12 557)	Non-active user (n = 1787)	Active user (n = 5486)	Intense user (n = 4441)	Problematic user (n = 843)
Gender					
Boys	44.8 (42.8–46.8)	**56.5 (52.6–60.3)**	46.0 (43.5–48.5)	**39.6 (36.7–42.5)**	**35.9 (30.7–41.2)**
Girls	55.2 (53.2–57.2)	**43.5 (39.7–47.4)**	54.0 (51.5–56.5)	**60.4 (57.5–63.3)**	**64.1 (58.8–69.3)**
Age group, years					
11–13	49.4 (43.8–55.0)	**66.2 (59.9–72.4)**	49.2 (43.5–55.0)	**41.6 (35.5–47.7)**	42.1 (34.1–50.1)
14–17	50.6 (45.0–56.2)	**33.8 (27.6–40.1)**	50.8 (45.0–56.5)	**58.4 (52.3–64.5)**	57.9 (49.9–65.9)
Cultural/ethnoracial background					
White	71.7 (66.8–76.6)	**74.2 (68.8–79.7)**	74.1 (69.1–79.1)	**68.9 (63.8–74.0)**	**60.9 (53.9–68.0)**
Non-White	28.3 (23.4–33.2)	**25.8 (20.3–31.2)**	25.9 (20.9–30.9)	**31.1 (26.0–36.2)**	**39.1 (32.0–46.1)**
Relative family affluence					
Low	13.1 (11.6–14.5)	**14.9 (12.5–17.2)**	11.6 (9.9–13.3)	**13.4 (11.4–15.3)**	15.3 (11.6–19.1)
Medium	59.0 (56.8–61.2)	**62.7 (59.5–65.9)**	61.7 (59.3–64.2)	**53.5 (50.9–56.2)**	55.0 (50.1–59.9)
High	27.9 (25.2–30.6)	**22.4 (18.8–26.0)**	26.6 (23.7–29.6)	**33.1 (30.1–36.1)**	29.7 (24.3–35.0)
Insomnia symptoms					
No insomnia symptoms	76.0 (74.6–77.4)	78.4 (75.7–81.1)	77.1 (75.4–78.7)	75.2 (72.8–77.7)	**66.5 (61.9–71.1)**
Insomnia symptoms	24.0 (22.6–25.4)	21.6 (18.9–24.3)	22.9 (21.3–24.6)	24.8 (22.3–27.2)	**33.5 (28.9–38.1)**
Daytime wakefulness problems					
No	62.2 (60.2–64.3)	**73.1 (70.3–75.9)**	64.6 (62.2–66.9)	**59.5 (56.9–62.0)**	**38.7 (33.4–44.0)**
Yes	37.8 (35.7–39.8)	**26.9 (24.1–29.7)**	35.4 (33.1–37.8)	**40.5 (38.0–43.1)**	**61.3 (56.0–66.6)**
Screen time before bed, nights per week					
<2	15.3 (13.6–16.9)	**33.5 (29.4–37.7)**	15.8 (13.8–17.8)	**8.0 (6.7–9.2)**	**6.1 (4.0–8.2)**
≥3	84.7 (83.1–86.4)	**66.5 (62.3–70.6)**	84.2 (82.2–86.2)	**92.0 (90.8–93.3)**	**93.9 (91.8–96.0)**
Sleep duration recommendations					
Meeting recommendations	63.5 (60.4–66.7)	**75.0 (71.5–78.5)**	68.2 (64.9–71.4)	**55.4 (52.2–58.5)**	**45.5 (40.0–51.0)**
Not meeting recommendations	36.5 (33.3–39.6)	**25.0 (21.5–28.5)**	31.8 (28.6–35.1)	**44.6 (41.5–47.8)**	**54.5 (49.0–60.0)**
Sleep variability					
Little or no sleep variability	63.7 (62.0–65.5)	**74.2 (71.3–77.2)**	68.8 (66.7–70.8)	**55.0 (52.5–57.6)**	**49.7 (44.6–54.8)**
Sleep variability	36.3 (34.5–38.0)	**25.8 (22.8–28.7)**	31.2 (29.2–33.3)	**45.0 (42.4–47.5)**	**50.3 (45.2–55.4)**
Sleep timing on school days					
Early or moderate bedtime	80.1 (77.9–82.4)	**88.1 (85.8–90.4)**	83.4 (81.1–85.6)	**74.9 (71.9–77.9)**	**65.9 (59.5–72.4)**
Late bedtime	19.9 (17.6–22.1)	**11.9 (9.6–14.2)**	16.6 (14.4–18.9)	**25.1 (22.1–28.1)**	**34.1 (27.6–40.5)**
Sleep timing on weekends					
Early or moderate bedtime	74.0 (71.9–76.1)	**86.5 (84.0–89.0)**	79.9 (77.9–81.9)	**64.1 (61.6–66.6)**	**54.7 (49.6–59.9)**
Late bedtime	26.0 (23.9–28.1)	**13.5 (11.0–16.0)**	20.1 (18.1–22.1)	**35.9 (33.4–38.4)**	**45.3 (40.1–50.4)**
Sleep duration					
Average weekday	8.5 (8.4–8.6)	**9.0 (8.9–9.1)**	8.7 (8.6–8.8)	**8.3 (8.1–8.4)**	**8.0 (7.8–8.2)**
Average weekend	9.7 (9.6–9.7)	**10.0 (9.9–10.1)**	9.8 (9.7–9.8)	**9.6 (9.5–9.6)**	**9.3 (9.2–9.5)**

Note: Significantly different proportions ($p < 0.01$) compared to the reference category (active social media users) are bolded.

than 14- to 17-year-olds in the non-active SMU category (66.2% and 33.8%, respectively) compared with the active SMU category (49.2% and 50.8%, respectively) (Table 1).

Association between SMU and sleep health indicators

Non-active SMU was associated with significantly lower odds of problematic sleep health indicators compared with active SMU (aORs from 0.42 to 0.78), except for insomnia symptoms, where the association was not significant. Intense SMU was associated with significantly worse sleep for all sleep health indicators except insomnia symptoms (aORs from 1.14 to 2.24). Finally, problematic SMU had the highest odds of having poor sleep health indicators (aORs from 1.67 to 3.24). All of the aORs for problematic SMU and insomnia symptoms, were consistently greater than the aORs for intense SMU, although some not significantly (Table 2).

Stratified analyses

The odds of having poor sleep health indicators were greater for problematic users who were girls than for problematic users who were boys, compared to their active user peers; these indicators included insomnia symptoms (aOR = 4.13 and 2.18, respectively), having daytime wakefulness problems (aOR = 3.09 and 2.11, respectively), using screens 3 or more nights a week (aOR = 3.23 and 2.21, respectively), not meeting sleep duration recommendations (aOR = 2.83 and 1.86, respectively), sleep variability (aOR = 2.71 and 1.65, respectively), having a later bedtime on school days (aOR = 3.74 and 1.84, respectively) and having a later bedtime on non-school days (aOR = 4.13 and 2.18, respectively) (Figure 1).

A similar relationship was found for girl intense users and boy intense users, compared to their active user peers, including insomnia symptoms (aOR = 2.33 and 2.19, respectively), daytime wakefulness problems (aOR = 1.20 and 1.05, respectively), not meeting sleep duration recommendations (aOR = 1.77 and 1.62, respectively), sleep variability (aOR = 1.99 and 1.66, respectively), and having a later bedtime on school days (aOR = 2.06 and 1.72, respectively) and non-school days (aOR = 2.33 and 2.19, respectively). Odds ratios of having poor sleep health indicators did not differ significantly between girl non-active users and boy non-active users compared to their active user peers.

Overall, odds ratios for having poor sleep health indicators did not differ significantly between the 11- to 13-year-olds and the 14- to 17-year-olds across SMU categories (Figure 2).

Discussion

In this study, we examined associations between SMU and seven sleep health indicators in a nationally representative sample of adolescents in Canada. Our results show that both problematic and intense SMU were associated with worse sleep health across a range of indicators, compared to active SMU, while non-active SMU was associated with better sleep health. The presence of insomnia symptoms was the only indicator not associated with SMU. Associations were stronger for girls than for boys, but did not differ by age group. The associations for problematic SMU were generally stronger than for intense SMU with all the sleep health indicators.

Compared to previous research exploring the relationship between SMU and sleep,[12,24-26] our study offers a comprehensive examination of the relationship between SMU and different indicators of sleep health. Few studies have made a distinction between intense and problematic SMU and their associations with sleep. Our results point to both the intensity and problematic nature of SMU affecting many aspects of healthy sleep. A few explanations for these associations have been proposed. First, electronic devices (e.g. cellphones, tablets, computer screens) emit blue light, which affects the production of melatonin, a hormone that regulates circadian rhythms and sleep.[6,7] Second, social media activities can lead to psychophysiological arousal, in part because of the emotional content of social media, which can lead to difficulties falling asleep.[14,27] Third, the "never-ending" nature of social media can make it difficult to stop use at night, particularly for adolescents, who are still developing their capacity to self-regulate.[14,24] Fourth, the constant alerts may disrupt the sleep of the large number of adolescents who keep their phones in their bedrooms at night.[14,24] Research has found that 15% of those in France report disturbance of sleep because of text messages alerts.[28] Further, "fear of missing out," a general state of anxiety at missing out on experiences, may prevent young people from turning off their phones at night and disengaging from social media at bedtime.[15] This fear may contribute to psychophysiological activation before bedtime and to delayed bedtimes.[15]

Previous findings on the link between SMU and various health outcomes (e.g.

TABLE 2
Adjusted odds ratios of sleep health indicators by social media use category (n = 12 557)

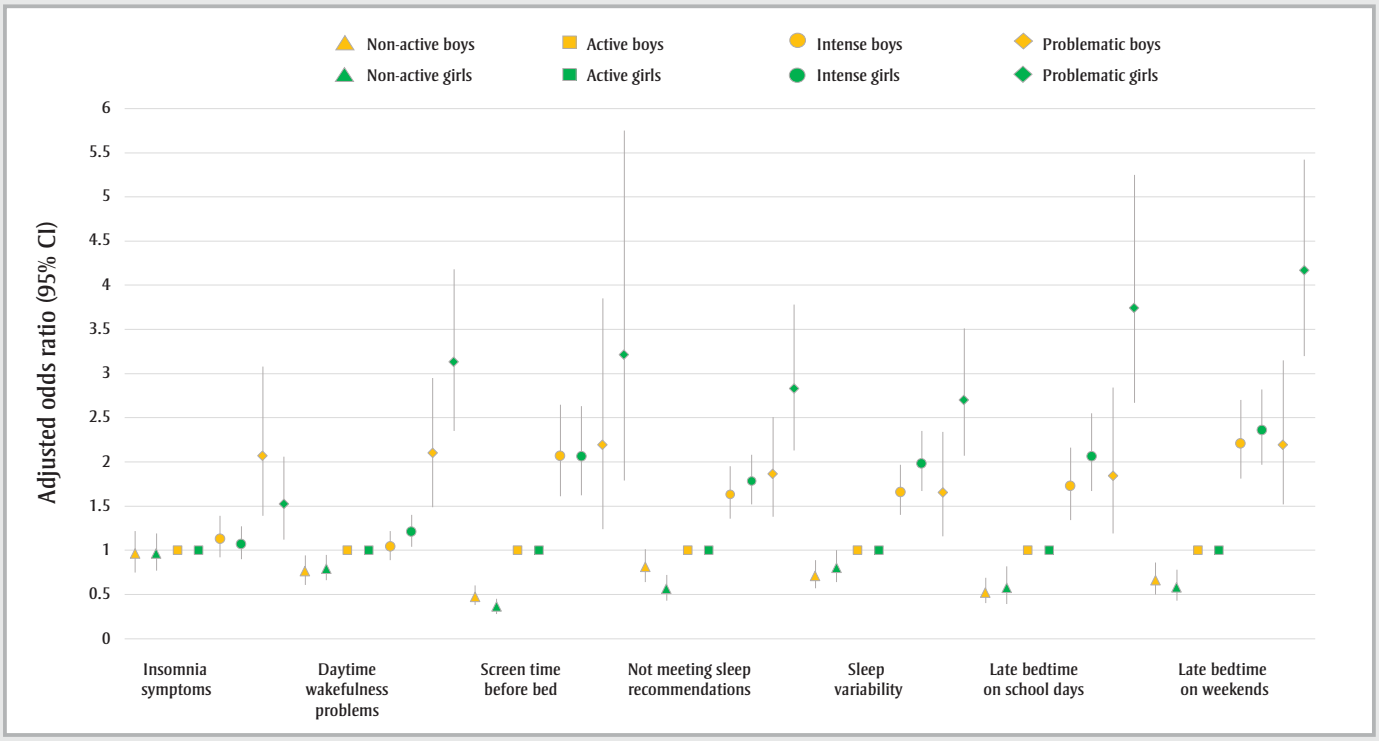
Social media use intensity	Sleep health indicators, aOR (95% CI)						
	Insomnia symptoms	Daytime wakefulness problems	Screen time before bed	Not meeting sleep duration recommendations	Sleep variability	Late bedtime on school days	Late bedtime on weekends
Non-active	0.98 (0.83–1.14)	**0.78 (0.68–0.90)**	**0.42 (0.34–0.50)**	**0.68 (0.58–0.81)**	**0.73 (0.62–0.87)**	**0.53 (0.42–0.67)**	**0.58 (0.48–0.72)**
Active	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Intense	1.09 (0.94–1.27)	**1.14 (1.03–1.26)**	**2.07 (1.73–2.48)**	**1.70 (1.50–1.93)**	**1.82 (1.60–2.07)**	**1.89 (1.63–2.18)**	**2.24 (1.97–2.55)**
Problematic	**1.67 (1.31–2.12)**	**2.67 (2.15–3.31)**	**2.76 (1.86–4.08)**	**2.43 (2.01–2.93)**	**2.23 (1.79–2.77)**	**2.89 (2.20–3.79)**	**3.24 (2.61–4.02)**

Abbreviations: aOR, adjusted odds ratio; CI, confidence interval.

Notes: All regression models are adjusted for gender, age, cultural/ethnoracial background and relative family affluence categories, and weighted using survey weights.

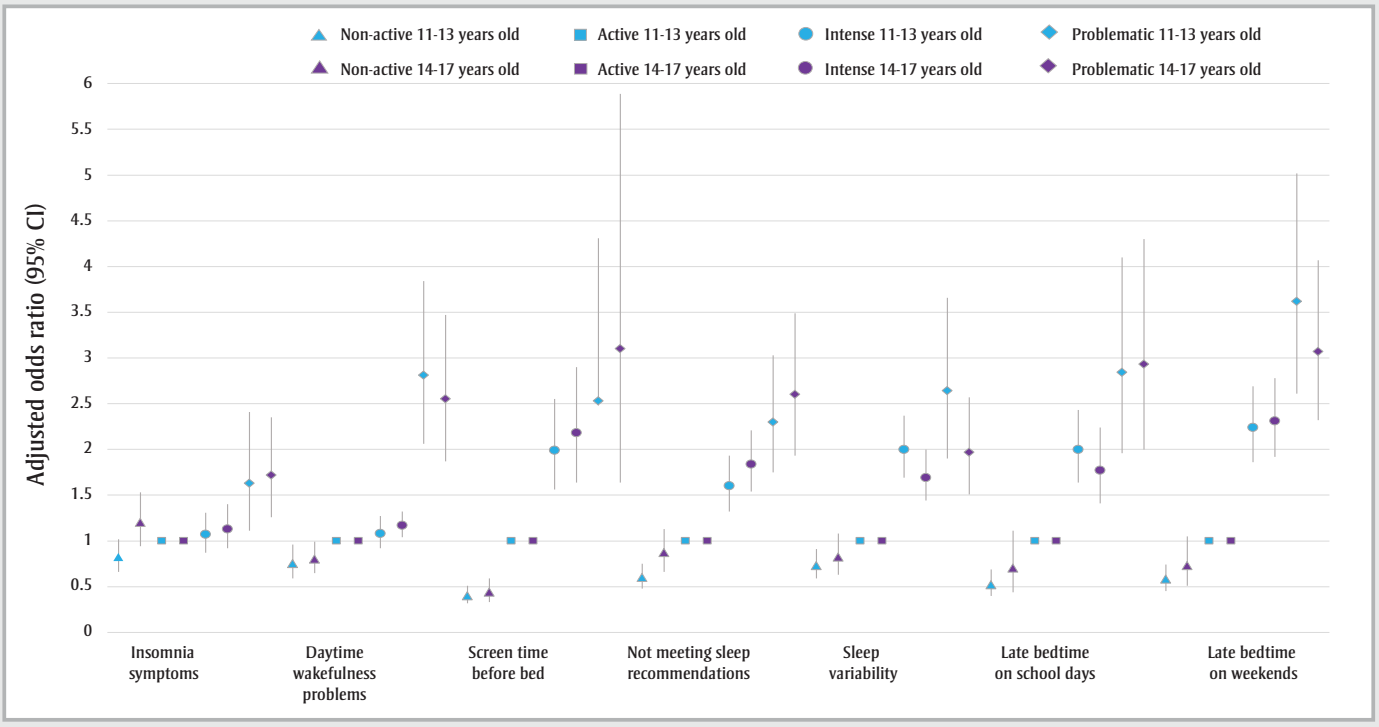
Significantly different proportions ($p < 0.01$) compared to the reference category (active social media users) are bolded.

FIGURE 1
Adjusted odds ratios stratified by gender for seven sleep health indicators, by social media use category (n = 12 557)



Abbreviation: CI, confidence interval.
Note: All regression models are adjusted for age, cultural/ethnoracial background and relative family affluence categories and weighted using survey weights.

FIGURE 2
Adjusted odds ratios stratified by age for seven sleep health indicators, by social media use category (n = 12 557)



Abbreviation: CI, confidence interval.
Note: All regression models are adjusted for gender, cultural/ethnoracial background and relative family affluence categories and weighted using survey weights.

mental health, physical activity) suggest a curvilinear relationship where both non-active and problematic use are associated with health risks relative to active use; this is referred to as the Goldilocks hypothesis.[29] However, we found that non-active use was associated with better sleep indicators than active use, suggesting a monotonic relationship between SMU and poor sleep health. Notably, intense users had worse sleep health outcomes than non-active and active users, even if their use was not problematic.

We found a stronger association between problematic SMU and sleep indicators among girls than boys. Previous work has shown that, even when using screens for less than 2 hours per day, girls were more likely to experience insufficient sleep than boys.[30] However, to our knowledge, no study has examined gender differences in intense and problematic SMU and sleep. There is evidence that girls and boys use social media differently and that girls may be more susceptible to the negative impacts of SMU.[14] For example, girls are more likely to engage in social comparison and seek feedback on social media, which may influence their body image concerns and possibly explains why their sleep is affected to a greater extent.[17,31] They may also be more susceptible to the psychophysiological arousal effects of social media; they report greater emotional investment and increased stress linked to SMU.[26] A longitudinal study of adolescents in the Netherlands found that social media stress was associated with greater daytime sleepiness among girls but not boys.[26] Finally, research also suggests a difference between active (e.g. posting or commenting) and passive (e.g. viewing posts, scrolling) SMU. Passive SMU is associated with lower well-being, with a stronger effect in girls,[32] that might translate into girls spending more time on social media at night and more sleep difficulties.

Overall, we did not find statistically significant differences between 11- to 13-year-olds and 14- to 17-year-olds in associations between SMU and sleep indicators. To the best of our knowledge, our study is among the first to examine age differences in the association between SMU categories and sleep health indicators. Sleep health tends to change with age, with older adolescents sleeping less and going to bed later, partly due to biological changes.[7,11] Time spent on social media also increases as adolescents age, which could explain why the associations between SMU categories and sleep health remain similar with age.

Strengths and limitations

There are several strengths to this study. First, it uses nationally representative data, with data collection following a standardized protocol. The study also included a distinction between intense and problematic SMU, using a validated scale for problematic SMU, and examined seven sleep health indicators that offer a broad picture of sleep health.

Some limitations need to be acknowledged. The use of cross-sectional data does not allow for causal inferences. There may have been unmeasured confounding that we were not able to adjust for in our analysis. In addition, the data were self-reported. Some studies suggest that self-reported sleep duration correlates moderately with actigraphy-measured sleep but that self-reports often overestimate sleep duration, which may have introduced some measurement error.[33] Only 58% of the Canadian HBSC sample had complete data and were included in our analyses. When comparing the characteristics of our included sample against the excluded samples, we found significant differences in gender, age group, culture/ethnoracial background, SMU and screen time before bed, which may indicate a risk of sampling bias in our study impacting the generalizability of our results (data available from the authors on request). Many of the measures used in this study have not been validated, representing an important area of future research. Our measure of SMU does not specify if the use was active (e.g. communicating with friends and creating content) or passive (e.g. scrolling through feeds), and our study could not distinguish between SMU and exposure to screens in general. We also did not have information on the type of device (i.e. phone, tablet, computer) that may play a moderating role in the associations with sleep health. To more precisely understand how SMU affects sleep, further studies should distinguish between active and passive SMU and account for other types of screen use.

Conclusion

In our study, intense and problematic SMU were associated with worse sleep health compared to active SMU, whereas non-active SMU was linked to better sleep health. These associations were stronger among girls than boys.

Further research is needed to understand the underlying mechanisms between SMU and sleep and to investigate potentially important gender differences. To guide public health recommendations, further studies could collect data on specific social media activities and use objective measures of sleep and SMU (such as time spent on social media apps). During the COVID-19 pandemic, adolescents increased their time spent on social media.[4] Considering our findings, it will be important to examine how changes in youth SMU due to the pandemic have impacted sleep health in adolescents.

Acknowledgements

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Conflicts of interest

None to declare.

Justin J. Lang is one of this journal's Associate Scientific Editors, but has recused himself from the review process for this article.

Authors' contributions and statement

FLP: Conceptualization, Formal analysis, Data curation, Writing – Original draft, Writing – Review & editing

JJL: Formal analysis, Data curation, Writing – Original draft, Writing – Review and editing

BM: Data curation, Writing – Review and editing

IS: Data curation, Writing – Review and editing

KCR: Data curation, Writing – Review and editing

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JPC: Data curation, Writing – Review and editing

IJ: Data curation, Writing – Review and editing

MBN: Data curation, Writing – Review and editing

GG: Conceptualization, Data curation, Formal analysis, Writing – Original draft, Writing – Review and editing

All authors approved the final manuscript.

The content and views expressed in this article are those of the authors and do not necessarily reflect those of the Government of Canada.

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https://doi.org/10.24095/hpcdp.44.7/8.06

Letter to the Editor

Re: Indigenous people's experiences of primary health care in Canada: a qualitative systematic review

Chandrakant P. Shah, MD, FRCPC, SM (Hyg), DrSc(Hon), OOnt (1,2)

Dear Editor,

I read the article by G. Barbo and S. Alam titled "Indigenous people's experiences of primary health care in Canada: a qualitative systematic review," which was published in the April issue of your journal.[1] As someone who has been involved in providing primary care to the Indigenous communities of Northwest Ontario and Anishnawbe Health Toronto, I found the article to be insightful. It reaffirmed commonly known facts about the issues facing Indigenous peoples in health care, such as privacy concerns, racism, discrimination and lack of culturally safe care.[2] Although organizations such as the Indigenous Physicians Association of Canada have developed needed core competency for health care professionals[3] and the Provincial Health Services Authority of British Columbia has developed courses to provide culturally safe care, the pace of change remains slow, and we continue to read stories about racism and discrimination against Indigenous people to this day.

What is the root cause of these issues? In the late 1980s, when I taught Indigenous health to health sciences students at the University of Toronto, I asked my class to describe Indigenous people and one other racial group, such as Italians or Japanese in Canada. I was dismayed to find that nearly 90% of the adjectives cited for Indigenous people were stereotypically negative, compared to only 10% for the other racial group. Most students had no encounter with Indigenous people and based their opinions on media encounters.[4] I realized that the students harboured "unconscious bias," and to remedy this, they needed Indigenous cultural safety courses provided by Indigenous teachers who had "lived experiences." I also conducted an environment scan to assess the teaching of Indigenous health courses across health sciences programs in Ontario's colleges and universities and found a lack of such courses in most of them due to a lack of Indigenous teachers.[5] With the help of Indigenous professionals, we developed an Indigenous cultural safety course and trained Indigenous preceptors across Ontario to deliver such courses in the health sciences programs. We found positive changes in students' attitudes towards Indigenous people.[6]

What is needed is the education of all Canadians, young and old, as well as new and naturalized Canadians, about Indigenous history and the impact of colonization, residential schools, the Sixties Scoop and our postcolonial policies such as the *Indian Act* on the health and well-being of Indigenous peoples. To start with, I recommend reading the *Honouring the Truth, Reconciling for the Future* by the Truth and Reconciliation Commission of Canada.[7]

Thank you for bringing this issue to light.

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Release notice

Perinatal Health Indicators (PHI) Data Tool

Stephanie Metcalfe, MSc; Jennifer Lye, MPH; Hongbo Liang, PhD; Holly Arscott, MScPH; Chantal Nelson, PhD; Wei Luo, MSc

The Maternal and Infant Health Section of the Public Health Agency of Canada (PHAC) is pleased to announce an update to the Perinatal Health Indicators (PHI) Data Tool.

The interactive Data Tool on the PHAC Infobase website presents statistics on maternal, fetal and infant health in Canada based on data from the Canadian Institute for Health Information's (CIHI) Discharge Abstract Database (DAD), the Canadian Community Health Survey (CCHS), and the Canadian Vital Statistics (birth, stillbirth and death databases).

The data include 20 indicators grouped into four key health domains: health behaviours and practices, health services, maternal outcomes, and infant outcomes. For this update, five new indicators were added and three existing ones were modified.

To access the latest Perinatal Health Indicators Data Tool, visit https://health-infobase.canada.ca/phi/.

Author reference:
Center for Surveillance and Applied Research, Health Promotion and Chronic Disease Prevention Branch, Public Health Agency of Canada, Ottawa, Ontario, Canada

https://doi.org/10.24095/hpcdp.44.7/8.08

Call for papers: Generating stronger evidence to inform policy and practice: natural experiments on built environments, health behaviours and chronic diseases

Guest editors: Dr. Stephanie Prince Ware (Public Health Agency of Canada), Dr. Gavin McCormack (University of Calgary)

HPCDP Journal Editors: Robert Geneau and Margaret de Groh (Public Health Agency of Canada)

Where we work, learn, play, eat and live has important implications for health. The built environment has been associated with the development of chronic disease, and with health behaviours often seen as critical pathways for this relationship.[1,2] Built environments refer to components of the physical environment that are human-made or human-modified and include structures and buildings, recreation facilities, green spaces and parks, transportation systems and community design.

Natural experiments are interventions that occur without a researcher's ability to manipulate the intervention or exposure to the intervention.[3,4] Natural experiments offer the opportunity to evaluate the effects of "naturally occurring" interventions such as changes to the built environment (e.g. creation of a new bike path, park improvements, infrastructure changes to schools or workplaces, construction of a new recreation facility or grocery store) on health behaviours and chronic disease risk. Natural experiments are often more practical for investigating the health impacts of environmental interventions when compared to traditional experimental studies (e.g. randomized controlled trials). Compared to cross-sectional studies, natural experiments provide a means to generate rigorous evidence to better establish causality, as well as to understand the implementation of interventions in "real-world" scenarios.

This special issue answers the 2017 Canadian Public Health Officer annual report's call to further evaluate the health impacts of community design features in Canada.[5] This special issue resonates with the expanding scholarly and policy-oriented interest in the utility of natural experiments as a critical tool in advancing the body of evidence and for informing interventions to improve public and population health.[6,7] Specifically, the objective of this special issue on natural experiments is to provide timely evidence to further understand the effectiveness of built environment interventions on health behaviours and chronic disease prevention in a Canadian context.

Health Promotion and Chronic Disease Prevention in Canada: Research, Policy and Practice is seeking relevant topical research articles that present new findings or synthesize/review existing evidence on natural experiments of the built environment (or related policies) that influence health behaviours with implications for chronic disease prevention in Canada.

Relevant topic areas include, but are not limited to:

- Built environments, including community or neighbourhoods, workplaces, schools, transportation infrastructure, home environments, recreation environments, parks, playgrounds, green spaces, public open spaces, natural environments and seniors' residences.
- All health-related behaviours, including physical activity, sedentary behaviour, sleep, food consumption, smoking and substance use.
- Chronic diseases and health-related outcomes, including body mass index, fitness, blood pressure, blood lipids, blood sugar, injuries, falls, mental health, stress, depression, anxiety, Alzheimer's disease, dementia, obesity, metabolic syndrome, cardiovascular disease, cancer, diabetes and lung disease.

International submissions will be considered if they include Canadian data, results (e.g. as part of multi-country studies or global comparisons) and/or evidence-based discussion of implications for community or population health in Canada.

Consult the Journal's website for information on article types and detailed submission guidelines for authors. Kindly refer to this call for papers in your cover letter.

All manuscripts should be submitted using the Journal's ScholarOne Manuscripts online system. Pre-submission inquiries and questions about suitability or scope can be directed to HPCDP.Journal-Revue.PSPMC@phac-aspc.gc.ca.

Submission deadline: November 30, 2024

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Other PHAC publications

Researchers from the Public Health Agency of Canada also contribute to work published in other journals and books. Look for the following articles published in 2024:

Apelian H, Aho J, Wong E, Cox J. The impact of the COVID-19 pandemic on social determinants of health, mental health, and substance use among key populations affected by sexually transmitted and blood-borne infections in Canada. Can J Public Health. 2024;115(3):432-42. https://doi.org/10.17269/s41997-024-00888-4

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