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Original Research

Erectile dysfunction and subsequent substance abuse: a retrospective cohort study of US non-academic hospitals

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Abstract

Background: **Erectile dysfunction (ED) affects millions and is associated with cardiovascular disease, diabetes, and depression—conditions that share risk factors with substance use disorders.**

Aim: **This study investigates the temporal relationship between ED diagnosis and subsequent substance abuse/dependence diagnoses across different age groups.**

Methods: **This retrospective cohort study used the TriNetX database to identify adult males from US hospitals with ED diagnoses (ICD-10: N52) with at least 3 years of follow-up. Controls were propensity-matched 1:1 on age and race. Patients were stratified into three age groups (20-39, 40-64, and ≥ 65 years). Cox proportional hazards models assessed risk over a 3-year follow-up period.**

Outcomes: **Primary outcome was any substance abuse/dependence diagnosis (ICD-10: F10-F19 categories); secondary outcomes examined individual substance categories.**

Results: **The study included 323 838 patients (161 919 with ED, 161919 controls) across three age strata. In men ≥65 years ($n = 176\,960$), ED was associated with elevated risk for multiple substances. Sedative abuse/dependence showed the strongest association (HR 2.28, 95% CI 1.52-3.42, $P < .001$), followed by other psychoactive substances (HR 1.79, 95% CI 1.43-2.24), opioids (HR 1.58, 95% CI 1.27-1.95), cocaine (HR 1.60, 95% CI 1.26-2.04), and cannabis (HR 1.45, 95% CI 1.10-1.92) (all $P < .05$). The elderly cohort showed reduced overall risk (HR 0.86, 95% CI 0.82-0.89, $P < .001$), driven by protection against nicotine dependence (HR 0.71, 95% CI 0.68-0.75, $P < .001$). In men 40-64 years ($n = 133\,292$), ED was associated with increased risk for cannabis (HR 1.32, $P = .013$) and other psychoactive substances (HR 1.21, $P = .031$), with protective effects for nicotine (HR 0.90, $P < .001$). Men 20-39 years ($n = 13\,586$) showed protective associations for opioid (HR 0.53, $P = .020$) and stimulant abuse (HR 0.35, $P = .003$).**

Clinical Implications: **Clinicians should maintain heightened awareness for substance abuse risk, particularly sedative misuse, when evaluating and treating elderly men with ED. Age-specific screening and counseling may be warranted.**

Strengths and Limitations: **Strengths include large sample size and propensity matching. Limitations include potential selection bias, inability to assess treatment adherence, and database-specific coding variations.**

Conclusion: **Elderly men with ED demonstrate significantly elevated risk for sedative, opioid, and cocaine abuse in the 3 years following ED diagnosis, while younger men show protective associations. These findings suggest a bidirectional relationship that warrants further investigation.**

Keywords erectile dysfunction, substance abuse, opioids, sedatives, cocaine, elderly, age-stratified analysis.

Introduction

Erectile dysfunction (ED) refers to the consistent inability to achieve or maintain a penile erection for satisfactory sexual activity.[1] ED is a prevalent condition nationwide with an overall prevalence of 24.2% in men aged 18 years and older as measured by the International Index of Erectile Function.[2] There is a clear age gradient, with higher prevalence rates in older cohorts. For

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example, ED is present in 13.3% of men ages 25-34 and 52.2% of men over age 75. Age is one of the strongest predictive factors for the development of ED.

Apart from age, medical comorbidities contribute to risk of ED development.[3] Type 2 diabetes, chronic kidney disease, peripheral vascular disease, and higher body mass index are all associated with development of ED. Other chronic conditions, such as neurologic disorders (stroke or multiple sclerosis) and endocrine disorders (hyperprolactinemia) are implicated as well.[4,5] Lifestyle factors are independent risk factors, such as tobacco smoking, alcohol misuse, and use of certain recreational substances (opioids, cannabis, amphetamines).[6,7] The underlying etiologies of these comorbidities typically relate to vascular pathology, endothelial dysfunction, and/or disruption of neural pathways.[5,8]

However, there also exist psychological contributors to ED which can occur in the absence of organic pathology. This can be due to pre-existing issues, such as prior traumatic experiences, inadequate sex education, or mental health conditions. Acute precipitating factors include relationship issues, familial or social pressures, and significant life changes, such as job loss.[9] Performance anxiety in regard to sexual activity is yet another important factor. Psychiatric conditions such as depression, generalized anxiety disorder, and schizophrenia are predisposing conditions. Depression is strongly associated with ED and the two exhibit a bidirectional relationship. Thus, the risk of developing depression is higher in men with ED, with rates increasing progressively in the years following diagnosis.[10] Furthermore, patients with various psychiatric conditions are at increased risk of various cardiometabolic conditions and substance use disorders, thereby further compounding risk of ED.[11,12]

The substances of particular concern in this context carry substantial individual and public health burdens. Opioid use disorder affected approximately 3.7% of US adults in 2022, contributing to a record 81 806 overdose deaths that year.[13] Sedative misuse, including benzodiazepines and other prescription anxiolytics, poses particular risks in elderly populations, where polypharmacy and psychiatric comorbidities compound vulnerability to dependence, cognitive impairment, and falls.[14] Cocaine use disorder, while less prevalent, similarly carries significant cardiovascular morbidity including elevated risks of myocardial infarction and stroke.[15]

While there is extensive evidence on the negative impacts of alcohol, cannabis, stimulants, opioids, and sedatives on erectile function, there is relatively limited evidence regarding a possible bidirectional association.[16] Specifically, the risk of developing substance use disorders after the onset of ED is not well understood. We therefore sought to answer the following question: does ED diagnosis increase the risk of subsequent substance abuse/dependence diagnoses, and does this risk differ across age groups?

Materials and methods

This retrospective cohort study used the TriNetX database, a global multi-center database with de-identified electronic medical record data, to identify adult males from US non-academic hospitals with ED diagnoses (ICD-10: N52) with at least 3 years of follow-up. Controls were propensity-matched 1:1 on age and race using the TriNetX platform's built-in algorithm, which employs logistic regression to generate propensity scores followed by greedy nearest neighbor matching. Index dates were defined by ED diagnosis for cases and by ambulatory visits for controls. Balance between cohorts was assessed using standardized mean differences, with values <0.1 indicating adequate balance. Exclusion criteria included prior diagnosis of substance abuse/dependence (ICD-10: F10-F19), schizophrenia spectrum disorders (F20-F29), bipolar disorder (F31), personal history of nicotine dependence (Z87.891), borderline personality disorder (F60.3), antisocial personality disorder (F60.2), and attention-deficit hyperactivity disorders (F90). Patients were stratified into three age groups (20-39, 40-64, and ≥ 65 years). Primary outcome was any substance abuse/dependence diagnosis (ICD-10: F10-F19 abuse and dependence categories); secondary outcomes examined individual substance categories. Substance categories were defined using ICD-10 codes as follows: alcohol (F10), opioids (F11, including heroin and prescription opioids), cannabis (F12), sedatives (F13, including benzodiazepines, barbiturates, and sleep medications), cocaine (F14), other stimulants (F15, including amphetamines, methamphetamine, and caffeine), hallucinogens (F16, including LSD, PCP, and ketamine), nicotine (F17), inhalants (F18), and other psychoactive substances (F19, including polysubstance use and substances not elsewhere classified). Chi-square tests were used to compare the prevalence of substance abuse and dependence diagnoses between ED and control cohorts within each age stratum. Cox proportional hazards models assessed risk over a 3-year follow-up period (1 day to 3 years post-index). All analyses were performed using the TriNetX platform (TriNetX LLC, Cambridge, MA, USA) with statistical significance defined as $P < .05$. This study utilized only deidentified, aggregated data from the TriNetX federated network without individual patient data export and was therefore exempt from Institutional Review Board review in accordance with federal regulations (45 CFR 46.104[d][4]).

Results

After applying exclusion criteria and propensity matching, 323 838 patients were identified (161 919 with ED, 161 919 controls). Baseline characteristics of the propensity-matched cohorts are presented in Table 1, with all standardized mean differences <0.1, indicating excellent balance across age, race, and demographic characteristics in all three age strata (20-39, 40-64, and ≥ 65 years).

The prevalence of substance abuse and dependence diagnoses across all categories and age groups is presented in Table 2, with ED patients demonstrating significantly higher prevalence than controls across most substance categories in the 40-64 and ≥ 65 age groups. Age-stratified hazard ratios for substance abuse/dependence diagnoses are shown in Table 3. In men aged 20-39 years ($n = 13\,586$), ED was associated with protective associations for opioid abuse (HR 0.53, 95% CI 0.31-0.91, $P = .020$) and stimulant abuse (HR 0.35, 95% CI 0.16-0.72, $P = .003$). In men aged 40-64 years ($n = 133\,292$), ED was associated with increased risk for cannabis (HR 1.32, 95% CI 1.06-1.63, $P = .013$) and other psychoactive substances (HR 1.21, 95% CI 1.02-1.43, $P = .031$). In elderly men (≥ 65 years; $n = 176\,960$), ED was associated with significantly elevated risk for multiple substances over 3-year follow-up. Sedative abuse/dependence showed the strongest association (HR 2.28, 95% CI 1.52-3.42, $P < .001$), followed by other psychoactive substances (HR 1.79, 95% CI 1.43-2.24, $P < .001$), opioids (HR 1.58, 95% CI 1.27-1.95, $P < .001$), cocaine (HR 1.60, 95% CI 1.26-2.04, $P < .001$), and cannabis (HR 1.45, 95% CI 1.10-1.92, $P = .009$). A sensitivity analysis excluding

Table 1 Baseline characteristics of propensity-matched cohorts by age group.

Characteristic	ED cohort	Control cohort
Age 20-39 years		
Total patients, *n*	6793	6793
Age, mean ± SD	33.4 ± 4.66	33.4 ± 4.66
Age at index, mean ± SD	26.9 ± 4.95	26.9 ± 4.95
Race, *n* (%)		
White	4372 (64.4%)	4372 (64.4%)
Black or African American	1101 (16.2%)	1101 (16.2%)
Asian	419 (6.2%)	419 (6.2%)
Unknown	579 (8.5%)	579 (8.5%)
Age 40-64 years		
Total patients, *n*	66 646	66 646
Age, mean ± SD	55.3 ± 6.51	55.3 ± 6.51
Age at index, mean ± SD	47.5 ± 7.15	47.5 ± 7.15
Race, *n* (%)		
White	44 081 (66.1%)	44 081 (66.1%)
Black or African American	13 413 (20.1%)	13 413 (20.1%)
Asian	3173 (4.8%)	3173 (4.8%)
Age ≥ 65 years		
Total patients, n	88 480	88 480
Age, mean ± SD	74.6 ± 6.83	74.6 ± 6.83
Age at index, mean ± SD	65.7 ± 7.44	65.7 ± 7.44
Race, *n* (%)		
White	64 631 (73.0%)	64 631 (73.0%)
Black or African American	13 974 (15.8%)	13 974 (15.8%)
Asian	4657 (5.3%)	4657 (5.3%)

Abbreviations: ED = erectile dysfunction; SD = standard deviation. All standardized mean differences <0.1, indicating balance after propensity score matching.

diagnoses within the first 90 days demonstrated an even stronger sedative association (HR 2.64, 95% CI 1.69-4.11, $P < .001$). Despite these substance-specific elevations, the elderly cohort showed reduced overall substance abuse risk (HR 0.86, 95% CI 0.82-0.89, $P < .001$). Hallucinogen abuse and dependence (F16) could not be reported across any age stratum due to insufficient sample sizes. Proportional hazards assumptions were violated for alcohol in all age groups and for overall substance use in ages 40-64 and ≥ 65 years, indicating that the hazard ratio between groups was not constant over the follow-up period and therefore a single HR may not fully characterize the temporal relationship for these outcomes (Table 3).

Nicotine dependence showed a distinct pattern across age groups. In men aged 20-39 years, no significant association was observed (HR 0.86, 95% CI 0.71-1.05, $P = .139$). In both middle-aged and elderly cohorts, ED was associated with reduced risk for nicotine dependence (ages 40-64: HR 0.90, 95% CI 0.85-0.95, $P < .001$; ages ≥65 years: HR 0.71, 95% CI 0.68-0.75, $P < .001$).

Discussion

The data reported within this study highlight significant age-related differences in substance misuse risk following ED diagnosis. Most notably, elderly men (≥65 years) with ED demonstrated significantly elevated risk for sedative (HR 2.28), opioid (HR 1.58), cocaine (HR 1.60), cannabis (HR 1.45), and other psychoactive substance abuse (HR 1.79) over the 3-year follow-up period. This contrasts sharply with younger men (20-39 years), who showed protective associations for opioid (HR 0.53) and stimulant abuse (HR 0.35). Men in the middle-aged cohort (40-64 years) demonstrated intermediate findings with modest elevations in cannabis (HR 1.32) and other psychoactive substances (HR 1.21).

This age-dependent pattern presents a notable paradox. Younger men with ED experience higher rates of mood disorders following diagnosis—with men under 45 years having a 40% risk of depressive symptoms compared with less than 20% in men over 65 years with similar ED severity.[17,18] Given the established association between mood disorders and substance use, one would expect younger cohorts to demonstrate elevated substance abuse risk.[19] However, our findings reveal the opposite pattern, suggesting that mechanisms beyond simple mood disorder pathways must be operative.

Younger men with ED may initially turn to opioids or stimulants in an attempt to enhance sexual performance or alleviate ED-related psychological distress. However, as these substances are well-documented to worsen erectile function,[20,21] their negative impact may create a deterrent effect, discouraging continued use. This negative feedback loop would likely be less operative in elderly men, whose greater average baseline ED severity may render the incremental worsening caused by substance use relatively less perceptible. Furthermore, elderly men with ED face distress not only from ED itself but also from its associated comorbidities, generating a heavier psychological burden that may increase

Table 2 Prevalence of substance abuse/dependence diagnoses in ED and control cohorts by age group.

Substance category	Age group	ED *n* (%)	Control *n* (%)	*P*-value
All substances				
	20-39	283 (4.63%)	237 (3.70%)	.010
	40-64	3687 (6.56%)	3099 (4.89%)	<.001
	≥65	3927 (4.90%)	3770 (4.41%)	<.001
Sedatives				
	20-39	—	—	—
	40-64	51 (0.08%)	31 (0.05%)	.036
	≥65	94 (0.11%)	31 (0.04%)	<.001
Other psychoactive substances				
	20-39	37 (0.55%)	30 (0.44%)	.440
	40-64	350 (0.53%)	214 (0.33%)	<.001
	≥65	255 (0.29%)	109 (0.12%)	<.001
Cocaine				
	20-39	—	—	—
	40-64	196 (0.30%)	113 (0.17%)	<.001
	≥65	174 (0.20%)	83 (0.09%)	<.001
Opioids				
	20-39	23 (0.34%)	31 (0.46%)	.353
	40-64	332 (0.50%)	217 (0.33%)	<.001
	≥65	308 (0.35%)	149 (0.17%)	<.001
Cannabis				
	20-39	34 (0.51%)	14 (0.21%)	.006
	40-64	220 (0.33%)	122 (0.18%)	<.001
	≥65	144 (0.16%)	76 (0.09%)	<.001
Inhalants				
	20-39	—	—	—
	40-64	44 (0.07%)	24 (0.04%)	.021
	≥65	46 (0.05%)	28 (0.03%)	.048
Other stimulants				
	20-39	11 (0.16%)	22 (0.32%)	.082
	40-64	137 (0.21%)	98 (0.15%)	.013
	≥65	65 (0.07%)	41 (0.05%)	.025
Alcohol				
	20-39	93 (1.40%)	53 (0.79%)	.001
	40-64	1094 (1.72%)	781 (1.19%)	<.001
	≥65	1204 (1.40%)	901 (1.03%)	<.001
Nicotine dependence				
	20-39	212 (3.34%)	181 (2.76%)	.062
	40-64	2753 (4.65%)	2430 (3.77%)	<.001
	≥65	2591 (3.13%)	2936 (3.40%)	.002

Abbreviations: ED = erectile dysfunction; *n* = number of patients with outcome; % = proportion of eligible patients (excluding those with outcome prior to index date). *P*-values calculated using chi-square test comparing ED vs. control cohorts within each age stratum. — = insufficient sample size.

vulnerability to substance abuse beyond what mood disorder pathways alone would predict.

Several hypotheses may explain the elevated substance abuse risk in older men with ED. Prescribing patterns represent one possible mechanism for the elevated sedative and opioid abuse risk in older men. Polypharmacy is common in elderly populations, and sedatives are frequently prescribed for anxiety and insomnia—conditions that may be exacerbated by ED-related distress.[22] Similarly, opioids are commonly prescribed for chronic pain conditions prevalent in this age group. This increased access to prescription medications with abuse potential may create opportunities for misuse in the setting of ED diagnosis. The strong association with sedatives (HR 2.28) and opioids (HR 1.58) supports this hypothesis, and the strengthened sedative association after excluding diagnoses within the first 90 days (HR 2.64) suggests true increased risk rather than mere detection bias.

Gaps in mental health care could also contribute as older men are significantly less likely to receive treatment for mental health disorders compared with younger cohorts.[23] This treatment gap may leave elderly men with ED-related psychological distress without adequate psychiatric care, potentially increasing susceptibility to self-medication behaviors. Additionally, the higher

Table 3 Hazard ratios for substance abuse/dependence diagnoses in men with ED vs. controls, stratified by age.

Substance Category	Age 20-39 HR (95% CI)	Age 40-64 HR (95% CI)	Age ≥ 65 HR (95% CI)
All Substances	0.90 (0.76-1.07)	0.98 (0.96-1.03)[a]	**0.86 (0.82-0.89)[a]**
Sedative	—	1.19 (0.76-1.85)	**2.28 (1.52-3.42)[a]**
Other Psychoactive Substances	0.89 (0.56-1.43)	**1.21 (1.02-1.43)**	**1.79 (1.43-2.24)**
Cocaine	—	1.26 (1.00-1.66)	**1.60 (1.26-2.04)**
Opioid	**0.53 (0.31-0.91)**	1.12 (0.94-1.33)	**1.58 (1.27-1.95)**
Cannabis	1.74 (0.93-3.24)	**1.32 (1.06-1.63)**	**1.45 (1.10-1.92)**
Alcohol	1.25 (0.89-1.75)	1.06 (0.96-1.16)[a]	1.05 (0.96-1.12)[a]
Other stimulants	**0.35 (0.16-0.72)**	1.02 (0.78-1.32)	1.21 (0.82-1.79)
Nicotine dependence	0.86 (0.71-1.05)	**0.90 (0.85-0.95)[a]**	**0.71 (0.68-0.75)[a]**

Abbreviations: HR = Hazard Ratio; CI = Confidence Interval; — = Insufficient sample size. Bold indicates statistical significance ($P < .05$). [a]Proportional Hazards assumption violated.

prevalence of medical comorbidities in elderly populations may serve as diagnostic distractors, making psychiatric etiologies less apparent to clinicians focused on managing multiple chronic conditions.

Age-related differences in treatment response may play a role, as treatment success for ED generally increases with decreasing age, with younger men exhibiting improved response rates to phosphodiesterase-5 inhibitors (PDE5i).[24] As younger men achieve greater psychological relief through effective ED treatment, this may attenuate risks associated with mood disorders. Conversely, older men with less responsive ED may experience persistent psychological distress, potentially creating vulnerability to substance misuse.

Finally, it is important to consider the role of historical and cultural factors surrounding the role of substances in ED diagnosis. Older men grew up in an era with different attitudes toward discussing sexual health and seeking legitimate medical help, potentially making them more likely to explore alternative solutions. Substances including cocaine, cannabis, and opioids have historically been associated with myths regarding aphrodisiac properties, despite scientific evidence demonstrating their detrimental effects on sexual function.[20,21] Older generations may be more susceptible to these persistent cultural beliefs, leading to experimentation with illicit substances in hopes of improving sexual function, which may subsequently progress to dependence.

In contrast to the elevated risks for most substances, both middle-aged and elderly cohorts demonstrated protective associations for nicotine dependence (HR 0.90 and 0.71, respectively). Tobacco use demonstrates a well-established unidirectional, dose-dependent relationship with ED, with each additional 10 years of smoking raising ED risk by 15% and each additional 10 cigarettes per day raising risk by 14%.[25] Smoking cessation is frequently emphasized as part of standard counseling for newly diagnosed ED patients, and the protective effect observed suggests this counseling may be effective in motivating behavior change.[26–28]

Clinical implications

These findings have important implications for clinical practice. The particularly strong associations with sedatives and opioids suggest that iatrogenic pathways may contribute to substance abuse risk in this population. Clinicians evaluating elderly men with ED should therefore carefully assess both prescribed medications and illicit substance use, maintaining heightened awareness for abuse of sedatives, opioids, cocaine, and cannabis. Routine screening for substance use should be incorporated into comprehensive evaluation, with consideration for implementing validated screening instruments such as CAGE-AID or DAST-10. Despite the high prevalence of medical comorbidities in elderly populations that may organically contribute to ED, psychiatric evaluation should not be neglected. Mood disorders and psychological distress related to ED warrant specific attention and treatment, as untreated psychiatric comorbidities may increase vulnerability to substance misuse. Additionally, medication reconciliation and polypharmacy assessment are critical, with careful review of prescriptions for sedatives, anxiolytics, and opioids, considering both therapeutic necessity and abuse potential. For patients at higher risk, family members or caregivers may need to be involved in medication management.

Patient education remains essential in this population. Older men with ED should receive clear counseling that illicit substances and prescription medication misuse will not improve erectile function and may worsen it. Dispelling persistent myths about aphrodisiac properties of certain substances is particularly important in this demographic. The strong protective association observed for nicotine dependence suggests that targeted counseling about substance-specific risks may be effective, and similar educational approaches should be extended to other substances of concern, including sedatives, opioids, and cocaine.

Limitations

This study has several important limitations. As an observational cohort study, our findings demonstrate associations but cannot establish causality; we cannot definitively conclude that ED diagnosis causes subsequent substance abuse. Despite propensity score matching on age and race, residual confounding from unmeasured variables such as ED severity, baseline psychiatric symptom severity, socioeconomic status, and healthcare access patterns may influence the observed associations. The inability to assess ED severity, treatment type, or PDE5i use and adherence is a further limitation, as successful ED treatment may attenuate substance abuse risk. The exclusion of patients with prior nicotine

dependence, although necessary for confounding purposes, may also have selected a population with lower baseline addiction vulnerability. Additionally, substance abuse diagnoses in administrative databases are likely under-coded due to stigma, patient reluctance to disclose use, and variable screening practices across institutions, potentially leading to underestimation of true incidence rates. The accuracy of ICD-10 substance use disorder codes in administrative databases varies by substance but is often limited. For example, ICD-10 codes for opioid misuse have a reported positive predictive value of approximately 78% with a sensitivity below 60%, suggesting that the incidence estimates of this study likely underrepresent the true burden of substance use disorders in this population.[29]

Database-specific limitations also warrant consideration. The TriNetX platform aggregates data from multiple healthcare systems with varying coding practices, data filters, and mapping processes, which may introduce inconsistencies.[30] Treatment duration and medication adherence cannot be determined from claims data, limiting our ability to assess whether ED treatment success modified substance abuse risk. Selection bias is possible, as men with mild ED who do not seek medical attention would be excluded from both cohorts.[31] This analysis was restricted to US non-academic hospitals, and findings may not generalize to academic medical center populations, which may differ in patient demographics, coding practices, and screening protocols. Sensitivity analyses including academic centers were not performed due to platform limitations and represent a direction for future research. Finally, the TriNetX population is approximately 5 years older than the US average and contains a higher proportion of patients with unknown or unspecified race, which may limit generalizability of findings.[32]

Conclusion

Elderly men with ED demonstrate significantly elevated risk for sedative, opioid, cocaine, and cannabis abuse in the 3 years following ED diagnosis, with sedative abuse showing the strongest association (HR 2.28). This elevated risk may reflect prescribing patterns in elderly populations, mental health treatment gaps, age-related differences in treatment response, and historical attitudes toward seeking medical help. Paradoxically, younger men demonstrated protective associations for opioids and stimulants despite higher rates of mood disorders, a finding that remains unexplained and warrants further investigation. Clinicians should maintain heightened awareness for substance abuse risk, particularly involving sedatives and opioids, when evaluating and treating elderly men with ED. Age-specific screening and counseling may be warranted to address these distinct patterns of risk. Future research should investigate the mechanisms underlying age-specific differences in substance abuse risk and examine the effectiveness of targeted screening and intervention strategies in elderly men with ED.

Author contributions (CRediT statement)

Hossein A. Zolfaghari: Conceptualization, Methodology, Validation, Formal Analysis, Investigation, Writing—Original Draft, Writing—Review & Editing, Visualization; **Juan Miguel Riestra:** Writing—Review & Editing; **Aleksandar Popovic:** Writing—Review & Editing; **Melissa Nanotkar:** Methodology, Formal Analysis, Investigation, Writing—Original Draft, Writing—Review & Editing, Visualization; **Diba Atar:** Resources, Writing—Review & Editing; **Jake S. Shilan:** Writing—Review & Editing; **Meher Pandher:** Writing—Review & Editing; **Amjad Alwaal:** Writing—Review & Editing, Supervision, Project Administration.

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Conflicts of interest

The authors declare no conflicts of interest.

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