Indication-stratified mortality risk of ibogaine treatment under contemporary safety protocols: a multisite analysis of 19,071 patients and updated systematic review of fatalities

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Additional Declarations: Yes there is potential Competing Interest. M.A. and K.S. report no conflicts of interest related to this manuscript. J.P.B. holds equity in Alvarius Pharmaceuticals, Beond, Brain Health Restoration Clinics, and Kernel, and serves as a consultant to Terragnosis, Sunstone Therapies, and Delphi Circle. To mitigate any potential influence of these interests on the study, data access and analysis were firewalled from J.P.B. M.A. was the sole investigator with access to the confidential, clinic-level data, which were provided under non-disclosure agreements held exclusively with M.A. M.A. conducted all statistical analyses of the clinical cohort and all screening, case adjudication, and de-duplication for the systematic review. J.P.B. contributed subject-matter expertise on ibogaine treatment and clinical safety practices but had no access to the underlying clinic data, no role in data analysis, and no influence over the selection, inclusion, or interpretation of fatality cases in the systematic review. The findings and their interpretation were determined independently of any commercial interest.

Abstract

Ibogaine has shown early therapeutic promise for substance use disorders (SUD) and post-traumatic stress disorder in veterans, but concerns about treatment-associated cardiac arrhythmia and death have constrained clinical development. Whether this risk is uniform across indications or concentrated in specific populations is unknown. We conducted a retrospective multisite study of 19,071 patients treated under established safety guidelines at 11 international clinics, with an updated systematic review of ibogaine-associated fatalities. Six deaths occurred within 72 hours, all among patients treated for opioid use disorder (6/10,382), with none among 8,689 non-SUD patients. This was mirrored in the review, where 41/44 fatalities with known indication involved SUD, predominantly opioid detoxification ($P=1.6\times10^{-9}$). The absolute rate represents a lower bound, given voluntary participation and non-adjudicated deaths; sensitivity analysis showed the conclusion robust to plausible unobserved deaths at non-participating clinics. These findings indicate that ibogaine-associated mortality is largely confined to opioid detoxification and rare in non-SUD indications.

Introduction

Ibogaine-assisted therapy has demonstrated early clinical efficacy in the treatment of substance use disorders (SUD), including opioid use disorder (OUD),[1] alcohol use disorder,[2,3] and cocaine dependence.[4] More recently, attention has shifted to non-SUD indications: in a prospective study of Special Operations Forces veterans with traumatic brain injury (TBI) and comorbid post-traumatic stress disorder (PTSD), depression and anxiety, magnesium–ibogaine treatment produced large, sustained symptom improvements with no serious cardiac events.[5] That work, conducted in a population without SUD, has become the principal scientific basis for an expanding policy and clinical interest in ibogaine in the United States. In June 2025, Texas allocated $50 million for Food and Drug Administration (FDA)-supervised ibogaine trials, subsequently matched by federal funding, and a 2026 federal initiative further prioritized psychedelic research for veterans. The FDA has cleared an investigational new drug application for noribogaine (ibogaine's principal metabolite) and established a pathway for eligible patients to access ibogaine compounds under the Right to Try Act. This marks a pivotal transition from observational and international use toward formally sanctioned clinical investigation, so it is essential to characterize ibogaine's safety profile and to determine which patients carry the highest risk.

Progress has been constrained by safety concerns, most notably ibogaine's potential adverse effects on the heart. These include proarrhythmic effects related to hERG potassium channel blockade, QTc prolongation, and risk of torsades de pointes (TdP).[6,7] A systematic review identified 19 ibogaine-associated deaths occurring 1.5 to 76 hours after administration between 1990 and 2008,[8] later updated to a total of 33 ibogaine-associated deaths until 2018.[9] Among cases with sufficient data, most involved pre-existing medical conditions, particularly cardiovascular disease or concurrent substance use, that likely contributed to fatal outcomes. Frequently cited historical fatality estimates of approximately 1 in

427 treatments (mortality rate: 0.23%; 2.34 in 1,000) were derived from data collected between 1989 and 2006,[9] preceding the implementation of standardized safety protocols.

The introduction of the *Clinical Guidelines for Ibogaine-Assisted Detoxification*[10] marked a shift toward structured risk mitigation, including cardiac screening, electrolyte management, medical monitoring, and exclusion of high-risk patients. Subsequent analyses have suggested that many reported fatalities were associated with preventable factors, such as excessive dosing, co-administration of QT-prolonging medications or CYP2D6 inhibitors, electrolyte abnormalities, and unmanaged comorbidities.[11,12] Yet two questions central to the current regulatory moment remain unanswered. First, it is unknown whether mortality risk is distributed uniformly across the patient populations now being considered for treatment, or whether it is concentrated in specific groups. Most plausibly, patients undergoing opioid detoxification are at higher risk due to withdrawal physiology, residual opioids, and polysubstance exposure. Second, systematic data on safety outcomes under contemporary, guideline-concordant care remain sparse, because the most influential mortality estimates predate the guidelines entirely.

We therefore addressed these questions in two complementary ways. Through collaboration with multiple international treatment centers, we assembled aggregated outcome data from 19,071 treatments delivered under established safety guidelines, allowing mortality to be stratified by treatment indication. In parallel, we extended prior fatality inventories[8,9] with cases reported since 2016 in the published and grey literature, enabling the distribution of fatalities across patient populations to be examined globally across more than three decades. Our primary interest was not the absolute mortality rate, which, as we discuss, cannot be precisely estimated from our limited, voluntarily contributed data. Rather, we determined which participants have the highest risk of fatality, which bears directly on patient selection for forthcoming trials.

Characterizing this risk matters because the conditions that ibogaine treats have high mortality rates. Untreated OUD carries an all-cause mortality of roughly 20.9 per 1,000 person-years,[13] and even established opioid agonist therapies carry appreciable mortality (11.3 and 4.3 per 1,000 person-years for methadone and buprenorphine, respectively).[14] PTSD is likewise associated with elevated all-cause mortality.[15,16] Although person-year estimates are not directly commensurable with acute, per-treatment risk, they nonetheless establish that any acute risk attributable to ibogaine must be weighed against the considerable mortality of the untreated condition.

In what follows we provide a contemporary, indication-stratified assessment of ibogaine-associated mortality from large-scale real-world data, together with an updated systematic review, intended to inform patient selection, cardiac risk mitigation, regulatory evaluation, and the design of prospective controlled trials.

Methods

Study Design

We conducted a retrospective observational study to characterize Ibogaine-associated mortality under contemporary clinical conditions with the primary aim of determining how fatality risk is distributed across treatment indications. The study combined (1) a multisite retrospective inventory of clinical outcomes and (2) a systematic review of reported fatalities. Seizure risk was evaluated as a secondary outcome (see Supplement).

No individual-level identifiable data were collected. All clinic data were aggregated and anonymized. Detailed methodology can also be found in the online supplement.

Data Sources

Multisite Clinical Data

A retrospective inventory study was conducted across international clinics administering ibogaine in accordance with the Clinical Guidelines for Ibogaine-Assisted Detoxification.[10]

Clinics were identified through professional networks and direct outreach to known treatment providers. Sixteen clinics were invited to participate; 11 contributed data. Participating clinics provided aggregated information under mutual non-disclosure agreements with author MA, to protect or proprietary or identifying clinical information, and no clinic-level identifiers are reported.

Published and Grey Literature

To contextualize clinical findings, we updated previously published fatality inventories.[8,9] Searches were conducted in PubMed using the terms “ibogaine,” “iboga,” “fatality,” and “death,” and structured searches of grey literature, including legal databases and media reports. Additional targeted searches were performed to identify non-English reports. The final search was conducted on April 9, 2026.

Identified cases were cross-referenced and de-duplicated against prior inventories and a structured ibogaine research database of case repositories (IbogaineVault[17]). Cases were included if death occurred within 72 hours after reported ibogaine exposure, irrespective of causal attribution.

Clinic Assessment and Data Collection

Participating clinics underwent structured safety evaluation using a standardized checklist administered via structured interview (IBO-SAFE; Appendix 2), assessing adherence to the published established guidelines, including pre-treatment screening, cardiac monitoring, and medical oversight.[10]

Clinics provided aggregated data for the period 2016–2026 (or longer when available), including:

- total number of patients treated (unique individuals),
- number of deaths within 72 hours after ibogaine administration,
- proportion of patients treated for substance use disorder (SUD) versus non-SUD indications,

- summary information on serious adverse events.

Fatalities were reported by clinics based on internal records and case review. No independent adjudication of reported deaths was performed; fatality determination relied on clinic-reported records.

Outcomes

The primary outcome was mortality, defined as death occurring during or within 72 hours after ibogaine administration. SUD status was defined according to the primary clinical indication reported by the treating clinic and is subject to clinic-level classification variability.

Secondary outcomes (including seizures) are reported in the Supplementary Appendix.

Statistical Analysis

Mortality rates were calculated as the number of deaths divided by the number of treated patients. Comparisons between SUD and non-SUD groups were performed using Fisher's exact test. When zero counts were present, odds ratios were estimated using the Haldane–Anscombe correction.

For descriptive analyses of reported fatalities, cases from the literature and clinical dataset were combined to summarize temporal and geographic patterns. Annual fatality counts were compared with indices of public interest (Google Trends) using Spearman's rank correlation.

To explore changes over time, fatality rates were compared between a pre-guideline period (2004–2016) and a post-guideline period (2017–2026) using a one-tailed t-test, consistent with the a priori hypothesis of lower rates following guideline implementation.

All analyses were conducted using GraphPad Prism, version 11 (GraphPad Software).

Results

Multisite Clinical Cohort

Of 16 clinics contacted, 11 participated (69% response rate). Data were obtained through a combination of on-site visits/in-person visits (7 clinics) and structured remote assessments (4 clinics). Clinics demonstrated high adherence to safety guidelines, with IBO-SAFE scores ranging from 7.3 to 9.8 (mean, 8.8/10).

Across participating sites, 19,071 patients were treated with ibogaine between 1996 and 2026. The mean start year of available records was 2014 (SD, 9 years).

A total of 6 deaths occurred within 72 hours after ibogaine administration, corresponding to a mortality rate of 0.03% (0.3 per 1,000 patients).

All deaths occurred among patients treated for SUD, specifically OUD (6 of 10,382; 0.06%). Reported substances for which patients had sought treatment included fentanyl (*n*=3), heroin (*n*=1), methadone and oxycodone (*n*=1), and unknown opioids (*n*=1). All deaths occurred in males (age 45-49). Where available, ibogaine doses ranged from 14 to 15 mg per kilogram. Reported causes of death were consistent with cardiotoxicity.

No deaths were observed among patients treated for non-SUD indications (0 of 8,689). Mortality was significantly higher in the SUD group (Fisher's exact test, *P*=0.026). Because of the absence of deaths in the non-SUD group, the odds ratio was not directly estimable. After application of a Haldane–Anscombe correction, the estimated odds ratio was 10.9 (95% confidence interval, 0.61 to 193), indicating a substantially elevated but imprecisely estimated risk.

Reported Fatalities in the Literature and Grey Sources

Updating prior inventories,[8,9] we identified 16 additional cases, resulting in a total of 48 reported deaths between 1990 and 2026 (see Appendix 3 for full overview and details).

Fatalities were reported across 15 countries (Figure 1B), most frequently in Mexico (*n*=13), the United States (*n*=9), the United Kingdom (*n*=5), the Netherlands (*n*=4), and Costa Rica (*n*=4). Among cases with available data, the mean age was 39.4 years (range, 23 to 60), and most were male (34 of 46 with available data). Among cases with reported indications, 42 of 45 involved SUD. The majority of fatalities occurred in clinical settings (n=27), followed by home or unsupervised settings (n=9), underground or unregulated settings (n=7), and ceremonial or ritual contexts (n=3). Pre-treatment ECG screening was documented in only 10 cases, 7 of which occurred after 2016; among pre-2016 cases, ECG screening status was unknown or unreported in 33 of 36 fatalities. Where an official cause of death was available, the most frequently reported categories were cardiac or cardiovascular events (n=16) and drug intoxication or combined toxicity (n=12). Pre-existing cardiac disease was documented in 12 cases. Polysubstance use at or around the time of death was reported in 17 cases; among the 19 cases with positive post-mortem toxicology findings, opioids (morphine, heroin, fentanyl, methadone, oxycodone, or codeine) were the most frequently detected substances (n=12), followed by benzodiazepines (n=8) and cocaine (n=6), with several cases involving combinations of these. Concurrent alcohol or benzodiazepine withdrawal risk was present in *n*=7 participants. In *n*=2 cases, fatal accidents occurred following disorientation or behavioral disturbances during the ibogaine experience (one traffic fatality and one drowning).

Three of the deaths identified in the clinical cohort were also reported in grey literature sources and were included in this dataset after de-duplication.

Temporal Trends

Annual counts of reported fatalities (2004–2026) showed a moderate correlation with measures of public interest (Spearman r = 0.47, P = 0.026; Figure 1A). This association was most pronounced during

the pre-guideline period (2004–2016; r = 0.59, P = 0.036) and absent during the post-guideline period (2017–2026; r = 0.35, P = 0.32). Mean annual fatality counts were higher in the pre-guideline period than in the post-guideline period (2.15 vs. 1.1; P = 0.04), consistent with a reduction in reported fatalities following the introduction of standardized safety protocols.

Combined Analysis

Across the combined dataset (literature and clinical cohort), 41 of 44 deaths with known indication (93.2%) occurred in individuals treated for SUD, a distribution inconsistent with chance (binomial test against a null of 50% SUD prevalence, P=1.6×10^{-9}).

Notably, all three deaths in non-SUD contexts occurred before 2016 and involved non-clinical or ceremonial settings; two were associated with pre-existing cardiac conditions.

Figure 1: (A) (left): Annual ibogaine-associated fatality counts in red (2004–2026) overlaid with Google Trends search volume in blue (for "ibogaine" or "iboga"). A clear pattern can be seen between increased online searches and higher fatality rates in the pre-guideline period (2004–2016) relative to the post-guideline period. In addition, an overall lower fatality rate can be seen in the post-guideline relative to the pre-guideline period. (B) (right) Geographical distribution of reported ibogaine fatalities by country.

Sensitivity analysis

Given that in the Multisite Clinical Cohortdata could not be obtained from the five non-participating clinics, we conducted a tipping-point sensitivity analysis to quantify how many unobserved deaths at those sites would be required to alter the pooled mortality estimate. Holding the observed clinical cohort fixed (6 deaths among 19,071 patients), we solved for the number of additional deaths that non-participating clinics would need to have experienced for the combined population rate to reach the historical estimate of 0.23% (approximately 1 in 427).[9] Assumed patient volumes at missing sites were estimated as 2,000-6,000.

Under these assumptions, reaching the historical estimate would require approximately 42 to 52 unobserved deaths at the five non-participating sites, corresponding to mortality rates of roughly 0.9% to 2.1% — 30-70 times higher than the rate observed in the participating cohort and exceeding any rate reported across the periods covered by our systematic review. We additionally note that the systematic-review component of this study searched legal, media, and grey-literature sources without restriction on treatment setting; a death toll of this magnitude would be expected to generate detectable signals through these channels.

Discussion

In this study combining a multisite clinical cohort with an updated systematic review, ibogaine-associated mortality was strongly concentrated by treatment indication. Across 19,071 treatments at 11

international clinics with high adherence to established safety guidelines, six deaths occurred within 72 hours, all in patients treated for OUD; no deaths occurred among 8,689 patients treated for non-OUD indications. The same pattern was evident in the literature, where 41 of 44 fatalities with a known indication (93%) occurred in individuals with SUD, predominantly during opioid detoxification—a distribution far from what chance would produce (binomial $P=1.6\times10^{-9}$). Altogether, our findings indicate that fatality risk is low and clusters in the opioid-detoxification population.

The absolute rate observed in the clinical cohort (six deaths; 0.03%) should be read as a lower bound rather than a point estimate. Participation was voluntary, clinics contributed aggregated data under non-disclosure agreements, and fatalities were self-reported without independent adjudication. Each of these features possibly biases the observed rate downward: clinics that declined to participate are invisible to this analysis, and unwitnessed or contested deaths may be undercounted. The indication-stratification finding, by contrast, is robust to these same limitations.

With this bounded interpretation in mind, the data nonetheless are consistent with an association between adherence to structured safety protocols and reduced mortality. Historical estimates of ibogaine-related fatality (approximately 1 in 427 or 2.3 per 1,000 treatments [0.23%])[9] were derived from periods preceding the introduction of standardized screening, monitoring, and exclusion criteria. As shown in the sensitivity analysis, overturning the conclusion of lower current mortality would require an implausible concentration of unobserved deaths. In the present study, lower observed mortality coincided with high adherence to these protocols. This convergence is most informative within the clinical cohort, where protocol adherence was directly assessed. A parallel temporal pattern in the reported-fatality literature points in the same direction but must be interpreted more cautiously. Earlier increases in reported fatalities tracked with rising public interest in ibogaine, whereas this association was attenuated in the post-guideline period despite greater public interest in recent years; that is, increased public interest has not been accompanied by a proportional increase in reported fatalities. This is consistent with a protective effect of contemporary safety practices, though these observations do not establish causality.

Mortality was concentrated in patients with OUD. Several features of the opioid-detoxification setting offer a coherent, if provisional, explanation for the concentration of risk. All clinical-cohort deaths were attributed to cardiotoxic mechanisms, consistent with ibogaine's established potential for QTc prolongation and torsades de pointes.[6,7] Withdrawal-associated electrolyte disturbance, autonomic instability, and frequent polysubstance exposure may compound this liability. The proportion of fentanyl-involved deaths (three of six cases) raises the possibility that specific opioid exposures may confer additional risk, though the sample size precludes firm conclusions. One plausible mechanism involves fentanyl's extraordinary potency combined with its high lipophilicity, which promotes tissue sequestration and unpredictable redistribution into plasma during ibogaine administration.[18] Fentanyl has been detected in urine for up to 26 days in persons with OUD entering treatment, complicating pre-treatment washout verification[19] and motivates a concrete, testable precaution: extended washout and quantitative toxicology confirmation before dosing patients with recent fentanyl exposure. In contrast, no

deaths were observed among patients treated for non-SUD indications. Notably, all three non-SUD fatalities identified in the literature occurred before the introduction of the ibogaine safety guidelines in 2016, in non-clinical or ceremonial settings. Two involved pre-existing cardiac disease, consistent with the interpretation that careful, comprehensive screening and monitoring can reduce fatality risk.

OUD is itself a condition of high mortality, which may explain the higher rate of ibogaine-associated deaths in the OUD population. Among individuals with OUD receiving opioid agonist treatment all-cause mortality rates are 11.3 per 1,000 person-years for methadone and 4.3 per 1,000 person-years for buprenorphine.[14,20] In addition, mortality within rehabilitation settings has also been reported at 0.24% (SAMHSA, 2015). These comparisons are not directly equivalent, as they contrast per-treatment risk with person-year estimates (i.e., estimates of deaths among 1,000 patients each followed for one year), but they provide context for evaluating the magnitude of risk associated with ibogaine relative to existing treatment paradigms.

From a mechanistic perspective, ibogaine's known risk of QTc prolongation and torsades de pointes[6,7] provides a plausible pathway for observed fatalities. Widespread implementation of cardiac screening and monitoring, as well as the co-administration of magnesium to reduce QTc interval lenthening is therefore likely to be clinically relevant. Although approaches to magnesium prophylaxis varied across clinics, its use was consistent with established strategies to mitigate arrhythmia risk and may contribute to improved safety profiles.

Seizures were rare (6 of 19,071; 0.03%) and were attributed to benzodiazepine withdrawal or, in one case, pre-existing epilepsy rather than to ibogaine itself. Adjunctive administration of 5-MeO-DMT following ibogaine treatment was reported by most participating clinics and has been described in prior observational studies.[2,21,22] While this practice may contribute to therapeutic outcomes, its role in safety or efficacy remains uncertain and warrants further study.

Given recent IND clearance of noribogaine (the principal metabolite of ibogaine) by the FDA and the establishment of a pathway for eligible patients to access psychedelic drugs, including ibogaine compounds, under the Right to Try Act, more studies investigating ibogaine are expected. Therefore, these new safety data can be important in designing and evaluating such studies. The structured safety assessment instrument developed and reported in this study (IBO-SAFE: Appendix 2), which is based on the Ibogaine Safety Guidelines,[10] may provide a practical framework for Institutional Review Boards and clinical investigators to evaluate and standardize safety procedures in forthcoming trials.

Several limitations qualify these conclusions. The retrospective design and reliance on aggregated, self-reported clinic data introduce reporting and selection biases whose direction, as noted above, possibly understates the absolute mortality rate. Fatalities occurring outside participating clinics or in unregulated settings may be incompletely captured. Apparent temporal declines in reported fatalities should be interpreted cautiously, as recent deaths are systematically less likely to have entered the published or grey literature. The inclusion of grey literature introduces heterogeneity in data quality but

was necessary to provide a comprehensive overview of reported cases. Outcomes were limited to the acute 72-hour period, and longer-term risks were not assessed. In addition, variability in ibogaine formulation and dosing could not be systematically evaluated.

Against these limitations, the study's strengths are its scale, its standardized assessment of protocol adherence, the firewalling of data access and analysis from any commercial interest, and the convergence of two independent data sources—clinical and literature-based—on the same indication-stratified pattern across more than three decades.

In conclusion, ibogaine-associated mortality in these data is concentrated in patients undergoing opioid detoxification and is rare in the non-SUD indications now prioritized for clinical investigation. The absolute rate observed under contemporary, guideline-concordant care is low but should be regarded as a lower bound; the more secure and more actionable finding is the location of risk. These results support careful screening of patients with OUD and cardiac risk mitigation in the design of prospective controlled trials, and they provide the population-level safety context that small prospective studies[5] cannot supply on their own.

Declarations

Acknowledgement

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Disclosures

M.A. and K.S. report no conflicts of interest related to this manuscript. J.P.B. holds equity in Alvarius Pharmaceuticals, Beond, Brain Health Restoration Clinics, and Kernel, and serves as a consultant to Terragnosis, Sunstone Therapies, and Delphi Circle.

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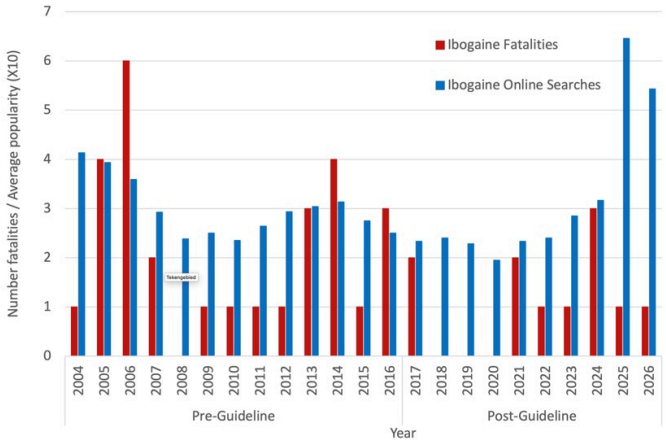
Figures



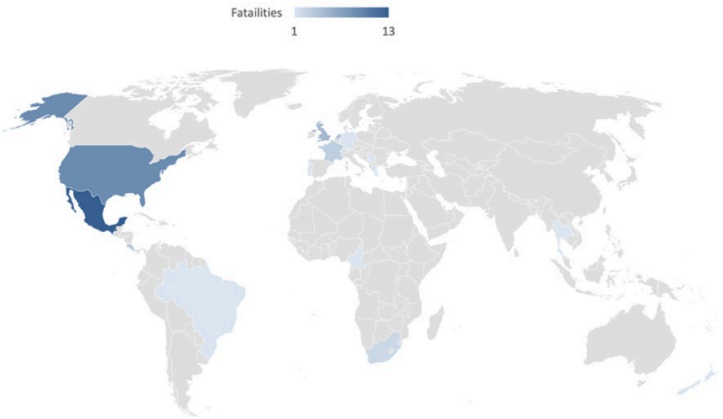


Figure 1

(A) (left): Annual ibogaine-associated fatality counts in red (2004–2026) overlaid with Google Trends search volume in blue (for “ibogaine” or “iboga”). A clear pattern can be seen between increased online searches and higher fatality rates in the pre-guideline period (2004–2016) relative to the post-guideline period. In addition, an overall lower fatality rate can be seen in the post-guideline relative to the pre-guideline period. (B) (right) Geographical distribution of reported ibogaine fatalities by country.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Appendix12.docx
- ArnsetalIbogaineFatalityDatabaseApr2026.xlsx