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Acute Substance-Induced Panic and Distress: A Harm-Reduction Framework for Immediate Stabilization

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Abstract

Recreational substance use can produce acute panic and distress even when no severe toxicological emergency is immediately evident. Existing literature commonly treats cannabis-related anxiety, stimulant intoxication, MDMA-related complications, cocaine-related cardiovascular risk, ketamine-related dissociation, and psychedelic challenging experiences as separate domains. This article conceptualizes acute substance-induced panic and distress as a harm-reduction stabilization problem located between informal user advice and emergency toxicology. It focuses on immediate stabilization rather than preparation, post-acute integration, durable psychological change, or general overdose management. The central model is an acute alarm loop in which bodily or perceptual change, threat interpretation, fear escalation, autonomic amplification, and further distress may reinforce one another. The model distinguishes analytically, rather than absolutely, between pharmacological effects and secondary escalation. The framework further distinguishes substance-specific acute risk modifiers, universal stabilization principles, research-mapped arousal-related exposures, and escalation boundaries. It does not claim pharmacological equivalence between substances and does not present any non-prescription substance as a treatment for intoxication. Rather, it offers a structured model for understanding and reducing mild-to-moderate acute distress during intoxication while preserving clear criteria for medical escalation.

Keywords: Acute distress, Acute intoxication, Harm reduction, Stabilization, Substance-induced panic.

1. Introduction

Recreational substance use can produce acute states of panic, fear, bodily alarm, derealization, dissociation, and perceived loss of control. Such reactions may occur with cannabis, stimulants, cocaine, MDMA, ketamine, and classic psychedelics. In severe cases, intoxication requires emergency medical management, particularly when chest pain, hyperthermia, seizure, collapse, severe confusion, respiratory compromise, neurological symptoms, or dangerous behavior is present. Yet many acute distress episodes occupy an intermediate zone: the person is frightened, physiologically activated, and subjectively destabilized, but remains responsive or orientable and does not show immediate red-flag signs of severe toxicological emergency.

This intermediate zone has not been adequately theorized as a distinct stabilization target. Clinical toxicology appropriately emphasizes severe intoxication, physiological instability, dangerous agitation, hyperthermia, seizures, cardiovascular complications, and overdose-specific treatment. Informal harm-reduction advice, by contrast, often consists of practical but uneven recommendations circulating in user communities, nightlife environments, cannabis contexts, psychedelic settings, and online forums. Between these domains lies a recurring problem: how to conceptualize and stabilize acute substance-induced panic and distress before it escalates, while preserving clear boundaries for medical escalation.

The need for such a framework is visible across several substance classes. Cannabis-related acute presentations frequently include anxiety, panic-like distress, derealization, paranoia, and cardiovascular sensations such as perceived heart racing [9,15,24]. Stimulant and sympathomimetic intoxication literature emphasizes autonomic activation, agitation, hyperthermia, hypertension, tachycardia, seizure risk, and toxidrome-based emergency assessment, but the lower-intensity zone of acute panic and distress is less systematically organized [1,3,12,27]. MDMA combines stimulant, empathogenic, and serotonergic features, with specific risks such as hyperthermia and hyponatremia in contexts involving heat, dancing, sweating, and excessive water consumption [10,11,14]. Cocaine is a stimulant with a distinct vasoconstrictive and cardiovascular risk profile, making chest pressure, palpitations, severe headache, and irregular heartbeat particularly important escalation signals [22,23]. Classic psychedelics and ketamine can produce distress through altered perception, self-boundary disruption, dissociation, derealization, and fear of losing control rather than through purely adrenergic mechanisms [6,13,19,26].

The present article focuses on immediate stabilization. It does not propose a universal treatment for intoxication and does not collapse pharmacologically distinct substances into a single mechanism. Its contribution is narrower: it conceptualizes acute substance-induced panic and distress as a harm-reduction stabilization target

and proposes a structured framework that separates shared acute distress dynamics, substance-specific risk modifiers, universal stabilization principles, research-mapped arousal-related exposure categories, and escalation boundaries.

The central claim is that different substances can produce distinct pharmacological states that nevertheless converge on a partially shared acute distress dynamic. A person may experience a racing heart after stimulants, derealization after cannabis, perceptual destabilization after psychedelics, emotional overwhelm after MDMA, chest fear after cocaine, or body detachment after ketamine. The pharmacology differs, but the subjective alarm process may share recognizable features: bodily or perceptual change becomes interpreted as danger; fear escalates; autonomic arousal intensifies; bodily sensations become more threatening; and the distress loop reinforces itself. This article terms that process the acute alarm loop.

By identifying this loop and distinguishing it from substance-specific acute risk modifiers, the framework aims to avoid two errors. The first is overgeneralization: treating all acute distress states as if they had the same cause and required the same response. The second is fragmentation: treating every substance-related panic episode as entirely unique, thereby missing common stabilization principles. The framework is transdiagnostic at the level of acute distress, not pharmacological mechanism.

For brevity, the framework is referred to as the Acute Substance-Induced Panic and Distress (ASIPD) framework.

2. Methodological Position

This article is a conceptual narrative review and framework paper. It does not attempt a systematic review, meta-analysis, clinical guideline, or treatment protocol. Sources were purposively selected to represent relevant domains: clinical toxicology, emergency medicine, cannabis-related acute presentations, stimulant and cocaine toxicity, MDMA-related complications, psychedelic harm-reduction literature, ketamine and dissociative-state research, panic and interoception models, and evidence on non-prescription arousal-related agents and exposures. The aim is theoretical organization and framework development rather than estimation of treatment efficacy.

The framework is intended as a research-oriented conceptual model. It identifies a neglected stabilization zone, proposes a functional mechanism of acute distress escalation, distinguishes substance-specific acute risk modifiers, and organizes potential stabilization strategies according to their role in interrupting the acute alarm loop. Its claims are deliberately limited: it does not establish clinical decision thresholds, does not validate specific interventions, and does not provide dosing or treatment recommendations.

3. Scope and Boundary Conditions

This article addresses mild-to-moderate acute distress states during recreational intoxication. The relevant state is temporary, acute, and distress-dominant. In this paper, distress refers to an acute subjective state of fear, perceived loss of control, perceptual destabilization, bodily threat appraisal, dissociation, derealization, or panic-like escalation during intoxication. It does not refer to general psychological suffering, long-term psychiatric disorder, or post-acute existential difficulty.

The term mild-to-moderate refers to the apparent acute distress state rather than to a definitive toxicological classification. It does not mean harmless and should not be read as a safety diagnosis. It denotes a conceptual working zone in which distress appears primary, the person remains responsive or orientable, and no severe physiological, neurological, or behavioral red flags are evident. Because early toxicity can resemble panic, uncertainty should shift interpretation toward caution rather than reassurance. The framework is designed to organize distress-reduction principles without delaying medical evaluation when physiological instability, severe confusion, hyperthermia, chest symptoms, neurological signs, or unsafe behavior emerges.

The included substance categories are cannabis and THC-containing products; stimulants and sympathomimetics, including amphetamine-type substances, methamphetamine, ephedrine-like agents, and synthetic cathinones; cocaine as a stimulant subtype with distinct vasoconstrictive risk logic; MDMA as a stimulant-empathogenic-serotonergic substance; classic psychedelics such as LSD, psilocybin, DMT, ayahuasca, 5-MeO-DMT, and mescaline; and ketamine or related dissociatives. These categories are included because each can produce acute panic or distress in recreational contexts, though through different mechanisms.

Several substance categories are not central to this model. Opioids, GHB/GBL, benzodiazepines, sedative-hypnotics, and alcohol-dominant intoxication require different primary frameworks because their dominant acute risk profiles often involve sedation, impaired consciousness, respiratory depression, blackout, aspiration, or overdose-specific management. These substances can be involved in panic, polysubstance risk, or confusion, but they are not best understood through a distress-first stabilization model.

Polysubstance use represents a major boundary condition for the framework. The substance-specific risk modifiers described below are clearest when a dominant substance class can be reasonably identified. In real-world recreational contexts, however, combined use of stimulants, cannabis, alcohol, sedatives, dissociatives, opioids, or serotonergic agents may mask, amplify, or alter expected presentations. In such cases, the framework should be applied more conservatively, with a lower threshold for escalation when the substance profile is uncertain, contradictory, or physiologically unstable.

The framework also does not attempt to explain or manage substance-induced psychosis, delirium, or sustained reality disorganization. Panic and distress may include fear, derealization, paranoia-like interpretation, or transient loss-of-control concerns, but persistent delusional conviction, severe disorganization, hallucination-driven unsafe behavior, or inability to reality-orient belongs outside the distress-first model. The framework ends when signs of medical instability, severe intoxication, delirium, unsafe psychosis, cardiovascular instability, neurological symptoms, hyperthermia, collapse, seizure, or respiratory compromise appear. These escalation boundaries are discussed in the section on escalation boundary.

4. The Acute Alarm Loop

The theoretical core of this framework is the acute alarm loop: a recursive process in which substance-related bodily or perceptual changes, threat appraisal, fear escalation, autonomic arousal, and intensified bodily or perceptual signals may reinforce one another. The model is consistent with panic theories emphasizing catastrophic misinterpretation of bodily sensations, interoceptive threat appraisal, and autonomic feedback processes [2,8,16,20].

The acute alarm loop should not be understood as a strictly sequential or purely cognitive model. In many intoxication states, especially stimulant-, cocaine-, MDMA-, or sympathomimetic-related states, autonomic arousal is not merely a secondary consequence of interpretation but part of the primary pharmacological effect. The model therefore distinguishes analytically, rather than ontologically, between substance-driven bodily change, threat interpretation, and secondary escalation. These elements may be tightly coupled in real time. The practical value of the model lies not in separating them absolutely, but in identifying modifiable points at which escalation may be reduced.

4.1. Mechanism of Recursive Escalation

The core of the ASIPD framework is the acute alarm loop, a recursive positive feedback process between substance-related bodily or perceptual alteration, threat appraisal, autonomic arousal, and intensified interoceptive or perceptual signals. This process can be analytically decomposed into four stages, although these stages may overlap in real time.

First, the cycle begins with substance-related somatic or perceptual input. Examples include stimulant-related tachycardia, tremor, heat, jaw tension, cannabis-related derealization or heightened heartbeat awareness, psychedelic perceptual destabilization, MDMA-related emotional and bodily intensification, or ketamine-related dissociation. In some cases, these changes may initially be recognized as transient substance effects.

Second, distress escalates when these bodily or perceptual changes are appraised as imminent threat. Palpitations may be interpreted as a heart attack, throat awareness as inability to breathe, derealization as permanent loss of reality, time dilation as an endless state, or depersonalization as irreversible psychological damage. This stage corresponds to catastrophic misinterpretation and interoceptive threat appraisal in established panic models.

Third, threat appraisal increases fear and autonomic arousal. Sympathetic activation may intensify heart rate, sweating, trembling, breathing irregularity, muscle tension, gastrointestinal sensations, dizziness, tingling, or heat perception. In stimulant-, cocaine-, MDMA-, or sympathomimetic-related states, this autonomic arousal may be both pharmacologically driven and secondarily amplified by fear. The framework therefore treats pharmacological activation and threat-based escalation as coupled rather than strictly separable.

Fourth, the secondary physiological stress response amplifies the initial bodily or perceptual disturbance. The intensified signals then appear to confirm the perceived threat, providing the person with apparent evidence that something dangerous is occurring. This closes the recursive loop and can maintain or intensify acute panic-like distress.

The ASIPD framework therefore suggests that escalation from moderate distress to more severe panic-like states is not always a simple pharmacological dose-response phenomenon. It may also involve state-dependent cognitive-somatic amplification. Stabilization should therefore target multiple points in the loop: sensory overload, bodily amplification, catastrophic appraisal, breathing-related escalation, and relational or environmental intensification. The aim is not to deny the pharmacological basis of the state, but to reduce secondary escalation that makes the state more frightening, unstable, or difficult to tolerate.

The acute alarm loop is often strengthened by environmental and social factors. Heat, crowding, loud music, bright lights, unfamiliar people, social pressure, physical exertion, isolation, and poorly calibrated responses from others can all intensify perceived threat. The model does not imply pharmacological equivalence. Cannabis, cocaine, MDMA, psychedelics, stimulants, and ketamine do not produce identical intoxication states. The acute alarm loop describes a convergent distress dynamic that may arise through different substance-specific pathways. It is a functional model of panic escalation, not a pharmacological model of intoxication.

The practical value of the acute alarm loop lies in identifying modifiable stabilization targets. If distress escalates through bodily signals, threat interpretation, autonomic arousal, and environmental amplification, then stabilization can target multiple layers: sensory load, physical exertion, breathing-related escalation, catastrophic appraisal, relational intensity, and further substance exposure. The aim is not to terminate intoxication, but to reduce secondary escalation that makes the state more frightening, unstable, or difficult to tolerate.

5. Substance-Specific Acute Risk Modifiers

A transdiagnostic model requires substance-specific boundaries. The same stabilization principle can have different implications depending on the substance. For this reason, the framework distinguishes a shared acute alarm loop from acute risk modifiers.

Table 1. Substance-specific acute risk modifiers.

Substance category	Acute modifier	Stabilization implication
Cannabis/THC	Delayed peak, derealization, paranoia, perceived heart racing	Explain time course, reduce stimulation, avoid further THC
Stimulants/sympathomimetics	Adrenergic load, heat, exertion, hypertension, tachycardia	Stop exertion, reduce heat, avoid added sympathomimetic load
Cocaine	Vasoconstriction, chest symptoms, palpitations, severe headache	Lower escalation threshold for cardiovascular symptoms
MDMA	Heat, sweating, serotonergic load, hyponatremia risk	Cooling, electrolyte-aware hydration, avoid excessive plain water and serotonergic add-ons
Classic psychedelics	Perceptual destabilization, altered self-boundaries, loss-of-control fear	Orientation, stable environment, non-confrontational support
Ketamine/dissociatives	Dissociation, depersonalization, impaired movement	Physical safety, reduced movement, safe body position

Cannabis/THC-related distress often involves altered perception of time, body, and reality. Edibles are relevant because delayed onset and prolonged duration can make intoxication feel unpredictable or unusually persistent. Immediate stabilization therefore emphasizes time-course explanation, stimulus reduction, and avoidance of further THC.

Stimulants and sympathomimetics include amphetamine-type substances, methamphetamine, ephedrine-like agents, and synthetic cathinones. Their acute risk modifier is adrenergic load: increased heart rate, blood pressure, heat, tremor, agitation, and physical drive. Stabilization therefore requires stopping exertion, reducing heat, avoiding additional sympathomimetic load, and monitoring whether the presentation remains within the mild-to-moderate distress zone.

Cocaine is pharmacologically a stimulant, but it should be treated as a distinct stimulant subtype because of its vasoconstrictive and cardiovascular risk profile. Cocaine-related panic may overlap with other stimulant states, but chest pressure, palpitations, severe headache, and irregular heartbeat require a lower threshold for escalation.

MDMA has stimulant, empathogenic, and serotonergic features. Its risk logic differs from standard stimulant panic because hyperthermia and hyponatremia are central concerns. Overhydration with plain water can be dangerous in MDMA contexts, especially when combined with heat, exertion, sweating, and impaired judgment. Stabilization therefore emphasizes cooling, rest, electrolyte-aware hydration, and avoidance of serotonergic add-ons such as 5-HTP or St. John's wort.

Classic psychedelics can produce distress through perceptual destabilization, altered time perception, self-boundary dissolution, symbolic overload, fear of losing control, or fear that the altered state will not end. They are grouped here only because acute distress may involve overlapping features such as perceptual destabilization, altered time perception, self-boundary disruption, and loss-of-control fear.

Ketamine and dissociatives require a different vocabulary. Ketamine-related distress should be understood primarily as dissociative distress rather than as panic in the narrow sense. The acute risk modifier is impaired orientation, altered body awareness, and reduced motor coordination. Stabilization therefore prioritizes physical safety, reduced movement, prevention of falls or injury, safe positioning, and calm reassurance about the temporary nature of dissociation.

6. Immediate Stabilization Framework

Immediate stabilization targets the acute alarm loop. It does not remove the substance from the body and does not replace medical care. Its purpose is to reduce secondary escalation by changing the sensory, bodily, cognitive, and relational conditions in which the distress is unfolding.

6.1. Environmental Downshifting

Environmental downshifting means reducing external inputs that amplify acute distress. Loud sound, intense light, visual complexity, crowding, heat, social pressure, and unnecessary movement can intensify fear and bodily vigilance. A first stabilization step is therefore environmental: move to a quieter space, reduce light, reduce noise, reduce the number of people involved, reduce heat exposure, and lower the pace of interaction.

This is especially relevant in nightlife, festival, and group settings. A distressed person may not be able to separate the substance effect from environmental overload. Reducing these inputs narrows the field of stimulation and makes orientation easier. Environmental downshifting should not be confused with abandonment. A distressed intoxicated person may need fewer stimuli, not isolation without support. The ideal environment is quiet, safe, minimally stimulating, and supported by a calm person who can orient without pressuring.

6.2. Interoceptive Stabilization

Interoceptive stabilization operationalizes the body-signal side of the acute alarm loop. During panic and intoxication, bodily signals may become intensified, unfamiliar, and threatening. Stabilization therefore seeks to reduce secondary bodily amplification without treating intoxication itself.

A stable body position is a first step. In cannabis-, stimulant-, MDMA-, cocaine-, or panic-dominant distress, an upright seated position may help reduce perceived breathlessness, improve respiratory comfort, and increase perceived control. In ketamine- or dissociation-dominant states, a safe seated, reclined, or side-lying position may be preferable because impaired coordination and altered body awareness increase the risk of falls or injury.

Respiratory down-regulation should be distinguished from intensive "breathwork." The relevant intervention is not hyperventilation, forced breathing, or emotionally activating breath practices, but slow, paced breathing with prolonged exhalation. In research or structured harm-reduction settings, this could be operationalized through simple exhalation-dominant patterns, for example approximately four seconds of inhalation followed by six seconds

of relaxed exhalation. Such timing should be understood as an illustrative operationalization rather than a universal prescription. Slow, exhalation-dominant breathing is included here as a mechanism-oriented stabilization category because it may reduce hyperventilation-related sensations such as tingling, dizziness, chest tightness, and perceived breathlessness [29]. In the acute alarm loop, these sensations can otherwise be misinterpreted as danger and further amplify panic.

Small sips of fluid may help with dry mouth, heat, or dehydration-related discomfort, while avoiding excessive water intake. Light carbohydrates may be useful when shakiness, fasting, or low intake contributes to bodily alarm. Temperature regulation also matters: cooling if overheated, warmth if chilled. Physical exertion should stop, especially in stimulant, cocaine, and MDMA contexts.

These measures reduce secondary physiological conditions that make intoxication feel more dangerous. In the acute alarm loop, the body becomes evidence. Interoceptive stabilization reduces the intensity and ambiguity of that evidence.

6.3. Cognitive Orientation

Cognitive orientation converts undefined threat into a time-limited state. The distressed person may not need complex explanation. They need simple, stable anchors: name, place, time, substance taken, current state, and expected temporariness. Orientation reduces uncertainty, and uncertainty fuels catastrophic interpretation.

Cognitive orientation should be brief. Overexplaining can become stimulation. Arguing with the person can become threat. Excessive questioning can increase self-monitoring. The best orientation language is simple, repetitive, and calm. Naming the state does not stop intoxication, but it gives the experience a frame. The person is not in an undefined catastrophe; they are in a substance-related state that can be observed and managed.

Examples of orientation language may include: "You are in a safe place," "This is an intoxication-related state," "The intensity can feel frightening, but it is time-limited," "We will reduce stimulation and stay still for now," and "We are watching for safety signs, and we will escalate if needed." In simpler peer-support contexts, this may be expressed as: "You are safe. This is the effect of the substance. It will pass." Such phrases should be brief, repetitive, and non-argumentative. Their purpose is not persuasion, interpretation, or reassurance at any cost, but reduction of uncertainty and catastrophic appraisal.

6.4. Relational De-Escalation

Relational de-escalation concerns the behavior of the support person. A distressed intoxicated person may be highly sensitive to tone, facial expression, pacing, and social pressure. A calm sober person can stabilize the social field. A panicked, mocking, argumentative, or overly intense person can worsen it.

Effective relational de-escalation uses few words, low verbal intensity, non-threatening posture, and predictable presence. It avoids ridicule, debate, forced insight, moral judgment, or confrontational correction. With psychedelic or paranoid content, the support person does not need to validate every belief, but aggressive contradiction can intensify fear. A better response is to orient toward safety and temporariness.

The relational goal is not persuasion. It is co-regulation. The support person lends stability until the acute alarm loop weakens.

7. Research Mapping of Arousal-Related Adjuncts and Exposures

This section is included as a research-mapping component rather than as an intervention recommendation. It does not propose, endorse, or recommend non-prescription substances for acute intoxication-related panic or distress. For most listed agents, no direct evidence exists in acute intoxication-related panic, and this absence of evidence is a central constraint rather than a minor limitation.

The purpose of this section is limited to three tasks: first, to classify commonly discussed non-prescription agents according to available indirect evidence; second, to distinguish potentially calming, neutral, activating, sedating, or interaction-prone exposures; and third, to identify areas where future research would be required before any applied recommendation could be justified. The categories below should therefore be understood as research-relevant arousal-modulation hypotheses and cautionary exposure classes, not as recommended acute interventions.

Table 2. Research mapping of arousal-related adjuncts and exposures.

Evidence category	Agent or exposure class	Research-relevant arousal domain or concern	Main limitation
A. Human evidence for stress/anxiety physiology, not intoxication-specific	L-theanine	Stress arousal, jitter, sympathetic tension	No direct evidence for intoxication-related panic
A. Human evidence for stress/anxiety physiology, not intoxication-specific	Magnesium	Muscle tension, tremor, stress-related arousal	Mixed evidence; not an acute antidote
A. Human anxiety evidence with cannabis relevance	CBD	THC-related anxiety plausibility	Product variability, delayed onset, THC contamination risk
B. Indirect physiological plausibility	Glycine	Settling, sleep pressure, inhibitory tone	Not specific to acute panic
B. Indirect physiological plausibility	Taurine	Arousal and cardiovascular plausibility	Limited acute intoxication data
B. Physiological context-specific plausibility	Electrolytes	Sweating, heat, MDMA hydration context	Not a panic treatment or treatment for hyponatremia
C. Traditional or limited	Passionflower	Calming/sedative	Sedation and interaction concerns

calming evidence		support	
C. Traditional or limited calming evidence	Valerian	Sleep pressure/sedation	Sedation; paradoxical response possible
C. Traditional or limited calming evidence	Lemon balm	Mild calming	Not intoxication-specific
D. Avoid/caution	Caffeine, nicotine, yohimbine, synephrine, ephedra	Increased sympathomimetic load	May worsen arousal, heat, tachycardia, panic
D. Avoid/caution	5-HTP, St. John's wort in MDMA contexts	Serotonergic load	May increase serotonergic risk
D. Avoid/caution	Alcohol or unsupervised sedative stacking	Sedation/confusion	May impair judgment and obscure deterioration

L-theanine is one of the more plausible research candidates for stimulant-like jitter or stress arousal because human studies suggest effects on stress response and relaxation without strong sedation [17]. However, this evidence is not intoxication-specific. Magnesium has mixed but relevant evidence in anxiety and stress-related symptoms and may be especially relevant when muscle tension, tremor, or bodily agitation dominate [5]. CBD is most relevant to THC-related distress as a research category, though it should not be described as a reliable antidote. Its onset, dose, formulation, and THC contamination risk matter [4].

Glycine is better understood as a settling or sleep-related research category than as an acute anti-panic tool [28]. Taurine has mechanistic plausibility through inhibitory and cardiometabolic pathways, but acute intoxication-specific data are limited [21]. Electrolyte-aware hydration is included as a harm-reduction research and context category in MDMA settings, not as a treatment for suspected hyponatremia or heat illness. Its relevance lies in hydration context, especially heat, sweating, dancing, and the need to avoid both dehydration and excessive plain water intake [10,11,14].

Passionflower, valerian, and lemon balm belong to a lower-evidence category: traditional or limited-evidence calming agents. Their possible relevance concerns sedation or subjective calming, but they raise concerns about additive sedation, interaction with alcohol or sedatives, and variable product potency [7,18,25].

A scientifically useful harm-reduction framework should also identify exposures likely to worsen the state. Rhodiola may be activating. Caffeine, nicotine, yohimbine, synephrine, ephedra, and similar sympathomimetic agents can increase arousal load. Niacin may worsen flushing or heat sensations. 5-HTP and St. John's wort are problematic in MDMA or serotonergic contexts because of serotonergic load. Alcohol and unsupervised sedative stacking can increase confusion, respiratory risk, falls, impaired judgment, and delayed recognition of deterioration.

The absence of intoxication-specific evidence is a central constraint of this section. No listed agent should be presented as effective for acute substance-induced panic or distress. The classification is intended only to support future study design and risk-sensitive harm-reduction discussion by separating arousal-related research hypotheses from exposure classes that may plausibly worsen stimulation, sedation, heat burden, serotonergic load, confusion, or delayed recognition of deterioration.

8. Escalation Boundary

The framework ends where severe intoxication, unstable physiology, delirium, unsafe psychosis, neurological symptoms, or cardiovascular instability begins. Escalation criteria are not external to the model; they define its boundary. Panic-like distress can mimic medical danger, but medical danger can also be misread as "only panic." A harm-reduction framework must therefore reduce unnecessary escalation while preventing dangerous underreaction.

Table 3. Escalation boundary.

Domain	Escalation signs
Cardiovascular	Chest pain, chest pressure, irregular heartbeat, collapse, severe hypertension, severe palpitations
Neurological	Seizure, focal neurological deficits, severe headache with confusion, syncope
Respiratory	Severe shortness of breath, cyanosis, respiratory compromise
Thermal	Hyperthermia, inability to cool down, heat illness signs
Cognitive/behavioral	Sustained severe confusion, delirium, dangerous behavior, unsafe psychosis, suicidality
Stimulant/cocaine-specific	Chest pressure, severe headache, irregular pulse, severe hypertension, neurological symptoms
MDMA-specific	Hyperthermia, confusion, collapse, seizure, severe headache, vomiting with confusion, suspected hyponatremia
Ketamine/dissociative-specific	Unresponsiveness, injury, respiratory compromise, sedative co-use
Psychedelic-specific	Dangerous behavior, persistent severe disorientation, psychosis-like state beyond the expected acute intoxication window

These escalation criteria operationalize the boundary of the framework. They do not imply that all red flags are equally likely across all substances. Rather, they indicate when the distress-centered model is no longer sufficient and medical evaluation becomes the appropriate frame.

Psychosis-like states are escalation-relevant when they involve sustained disorganization, dangerous behavior, inability to orient, or persistence beyond the expected acute intoxication window. Such states should not be reduced to panic or treated as simple alarm-loop escalation.

9. Research Agenda

The proposed framework is conceptual and requires empirical refinement. Several research directions follow.

First, observational studies in festival, nightlife, cannabis, and harm-reduction service settings could document acute substance-induced panic and distress presentations. These studies should record substance class, setting, subjective symptoms, stabilization strategies used, time to improvement, and escalation outcomes.

Second, cannabis-related acute anxiety deserves specific study because THC products, especially edibles and high-potency preparations, are common and can produce intense panic and derealization. Emergency department data already show that cannabis-induced anxiety is a meaningful presentation category [15]. A structured acute distress scale could help differentiate subjective panic, cardiovascular activation, derealization, paranoia, and red-flag features.

Third, analog studies could test individual components of stabilization without requiring intoxication. Paced breathing with prolonged exhalation, sensory reduction, orientation scripts, and relational de-escalation language could be tested in stress, panic, or interoceptive challenge models. This would provide a safer first step before intoxication-specific research.

Fourth, arousal-related agents and exposures discussed in the research-mapping section should be studied cautiously. Initial trials should focus on non-intoxicated stress models or cannabis-related anxiety contexts before broader claims are made. Outcomes should include subjective distress, heart-rate response, perceived control, time to settling, and adverse effects. Agents with activating, sedating, serotonergic, or interaction-prone profiles should be examined primarily as exposure risks before any applied use is considered.

Fifth, harm-reduction teams could develop standardized acute distress documentation tools. A brief checklist could include orientation, responsiveness, substance category, time since use, heat or exertion exposure, hydration context, chest symptoms, neurological symptoms, dangerous behavior, and stabilization response. Such tools would help bridge informal support and clinical escalation. A sample checklist is provided as Supplementary Appendix A.

10. Limitations

This article has several limitations. First, it is a conceptual narrative review and framework paper, not a systematic review, meta-analysis, clinical guideline, or treatment protocol. The framework organizes existing concepts and evidence domains but does not establish validated clinical thresholds or empirically tested intervention effects.

Second, the proposed acute alarm loop is a functional model rather than a validated psychological mechanism. It is intended to organize how bodily or perceptual change, threat interpretation, fear escalation, autonomic amplification, and environmental stressors may reinforce one another during acute intoxication-related distress. Future research would need to test the model against observational and experimental data.

Third, the substance categories included in the framework are heterogeneous. Cannabis, stimulants, cocaine, MDMA, classic psychedelics, and ketamine differ in pharmacology, duration, risk profile, and phenomenology. The framework therefore does not claim pharmacological equivalence; its value depends on maintaining the distinction between shared acute distress dynamics and substance-specific acute risk modifiers.

Fourth, real-world intoxication often involves polysubstance use, uncertain potency, adulteration, environmental stressors, sleep deprivation, dehydration, heat exposure, and incomplete information. These variables may substantially alter risk and reduce the usefulness of any simplified framework. Polysubstance use is especially important because combined substances may mask or potentiate expected risk profiles. The proposed model should therefore be understood as a starting point for structured harm-reduction research rather than as a complete decision system.

Fifth, the section on arousal-related agents and exposures is hypothesis-generating, classificatory, and non-prescriptive. The included agents are not presented as treatments for intoxication, and most evidence is indirect or not intoxication-specific. Product variability, unknown dosing, individual vulnerability, delayed onset, and interaction risk further limit any generalization.

Sixth, the framework does not address substance-induced psychosis, delirium, withdrawal syndromes, chronic substance-related anxiety, or long-term psychiatric outcomes. Acute panic and distress may include transient fear, derealization, or loss-of-control concerns, but sustained disorganization, hallucination-driven unsafe behavior, or persistent inability to reality-orient require a different framework.

11. Conclusion

Acute substance-induced panic and distress should be treated as a distinct harm-reduction stabilization target. The proposed framework identifies an acute alarm loop in which bodily or perceptual change, threat interpretation, fear escalation, autonomic amplification, and further distress may reinforce one another. By separating this shared distress dynamic from substance-specific acute risk modifiers, the framework avoids both overgeneralization and fragmentation. Its practical structure distinguishes environmental downshifting, interoceptive stabilization, cognitive orientation, relational de-escalation, research-mapped arousal-related exposures, and escalation boundaries. Future research should test immediate stabilization strategies in real-world and controlled settings, refine evidence grading for arousal-related exposure categories, and develop standardized tools for documenting acute substance-induced panic and distress.

Abbreviations:
5-HTP: 5-Hydroxytryptophan
ASIPD: Acute Substance-Induced Panic and Distress
CBD: Cannabidiol
DMT: N,N-Dimethyltryptamine
GHB/GBL: Gamma-Hydroxybutyrate / Gamma-Butyrolactone
MDMA: 3,4-Methylenedioxymethamphetamine
THC: Δ9-Tetrahydrocannabinol

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Appendix A. Acute Distress Documentation Checklist for Harm-Reduction Settings.

This checklist is intended as a research and documentation aid for harm-reduction teams, event-based support services, or observational studies. It is not a diagnostic tool, treatment protocol, or substitute for medical assessment.

Basic Context

- Date, time, and setting
- Approximate time since substance use
- Setting type: nightlife, festival, private setting, cannabis setting, retreat, public space, other
- Alone or with others
- Presence of a sober support person

Substance Context

- Reported substance or substances
- Route of administration, if known
- Approximate dose, if known
- Time since last use
- Possible polysubstance use
- Unknown potency or suspected adulteration
- Alcohol, sedatives, opioids, stimulants, cannabis, dissociatives, or serotonergic agents involved

Observable State

- Responsive and orientable: yes / no / uncertain
- Able to state name, place, approximate time, and situation: yes / no / uncertain
- Main presentation: panic, fear, derealization, dissociation, agitation, confusion, overheating, chest symptoms, other
- Motor coordination: stable / impaired / unsafe
- Behavior: calm, anxious, agitated, disorganized, unsafe

Environmental and Physiological Context

- Heat exposure
- Dancing or exertion
- Crowding or sensory overload
- Dehydration concern
- Excessive water intake concern
- Fasting or low food intake
- Sleep deprivation
- Recent conflict, fear, or social stressor

Stabilization Measures Used

- Environmental downshifting
- Reduced light, sound, crowding, or movement
- Upright seated position
- Reclined or side-lying position
- Slow exhalation-dominant breathing
- Small sips of fluid
- Cooling or warming
- Light carbohydrate intake
- Cognitive orientation
- Calm sober support person
- Reduction of questioning or argument
- Avoidance of further substance use

Red-Flag Screening

Escalation should be considered when any of the following are present or uncertain:

- Chest pain or chest pressure
- Severe shortness of breath
- Cyanosis
- Collapse or syncope
- Seizure
- Severe headache with confusion
- Focal neurological signs
- Hyperthermia or inability to cool down
- Irregular heartbeat or severe palpitations
- Sustained severe confusion or delirium
- Dangerous behavior
- Unsafe psychosis
- Suicidality
- Unresponsiveness
- Suspected hyponatremia, especially with MDMA context
- Polysubstance use with uncertain or unstable presentation

Outcome Documentation

- Improved without escalation
- Required medical evaluation
- Required emergency services
- Time to visible settling
- Remaining symptoms after stabilization
- Notes on what appeared to reduce or worsen distress

Research Notes

- Which part of the acute alarm loop appeared most dominant?
- bodily sensation
- threat interpretation
- environmental overload
- autonomic amplification
- dissociation or perceptual destabilization
- relational/social escalation
- Which stabilization target appeared most relevant?
- environmental downshifting
- interoceptive stabilization
- cognitive orientation
- relational de-escalation
- escalation boundary recognition