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The Unseen Burden:

Toxic Exposures and Health Impacts on U.S. Navy Submariners

AMENDMENT 1 – JANUARY 2026

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Amendment 1:

Physiological Impact Analysis: Compromised Closed-Loop Atmosphere

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Physiological Impact Analysis: Compromised Closed-Loop Atmosphere

Environment: Normobaric (1 atm / Sea Level Pressure)
Base Composition: Nitrogen (≥ 80%), Oxygen (≤ 19%), Carbon Dioxide (0.8%–2%)
Contaminants: Carbon Monoxide (CO), Monoethanolamine (MEA), 2,6-di-tert-butyl-4-nitrophenol (DBNP), Benzene, Freon

Usage Definitions:

> **Contaminant** is used when discussing the air quality itself (e.g., "130 chemicals that could be submarine atmospheric contaminants").
>
> **Toxicant** is used when describing the biological burden on the crew (e.g., "increased inhalation of other toxicants" and "exposed to more than 150 hazardous chemicals, gases, and toxicants").

1. Executive Summary

This specific atmospheric profile presents a **"Cascading Failure"** scenario for human physiology. The base atmosphere creates a state of **hypoxic stress and hyperventilation** due to higher Carbon Dioxide (CO_2) and low Oxygen (O_2). The presence of toxicants turns this stress into a lethal trap. Because the subject is hyperventilating from higher CO_2, they absorb toxicants like **Carbon Monoxide** and **MEA** at a significantly accelerated rate. While CO disables oxygen transport, MEA begins to chemically erode the respiratory system and systematically shut down the body's filtration organs (liver and kidneys).

Critically, the lethality of this environment is obscured by a fundamental flaw in historical and current safety standards: **the reliance on research that isolates individual toxicants**. As detailed in the report ***The Unseen Burden***, past scientific studies have "narrowly focused on individual chemicals or gases," ignoring the complex reality of the submarine atmosphere (SAG, 2025, pp. 27-28).

- **The "Toxic Cocktail" Blind Spot:** There are between 130 and 200 known contaminants in submarine atmospheres. However, safety guidelines are typically derived from studying these chemicals in isolation, neglecting to consider the "submarine atmosphere as a holistic entity" (SAG, 2025, pp. 27-28).
- **Scientific Gaps:** The National Research Council (NRC) has explicitly acknowledged that they "did not address exposure to chemical mixtures," leaving the potential for antagonistic, additive, or synergistic interactions largely unexamined (NRC, 2008, p. 6).
- **Flawed Pressure Models (Hypobaric vs. Normobaric):** Safety standards for oxygen deficiency are frequently based on "hypobaric hypoxia" (high altitude) research rather than the "normobaric" (sea-level pressure) environment unique to submarines. Scientific evidence indicates that "hypobaric hypoxia induces

different physiological responses compared with normobaric hypoxia" (Millet et al., 2012). Therefore, research derived from high-altitude environments is "not representative" of the continuous, normobaric exposure faced by Submariners, rendering current safety assumptions potentially flawed (Guenette & Koehle, 2012; Hohenauer, et al., 2022; Rosales, et al., 2022; Saugy, 2016)
- **Real-World Consequence:** This failure to study the combined "toxic cocktail" effect means that safety thresholds (like those for CO_2 or MEA) may be dangerously high when applied to an environment where multiple stressors—hypoxia, hypercapnia, and toxicity—attack the body simultaneously.

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2. Base Atmospheric Physiology

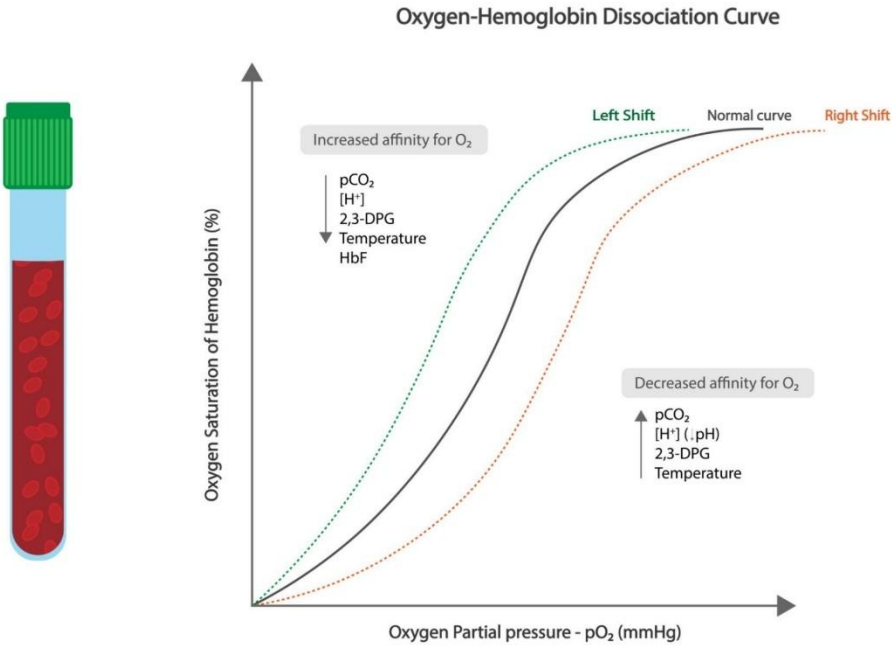
Nitrogen (N_2) at ≥ 80% (Inert Displacement)

At standard pressure, nitrogen is physiologically inert. Its primary danger here is **displacement**. By occupying over 80% (Knight et al., 1990) of the available volume, it mechanically forces oxygen levels down. Unlike diving scenarios, there is no risk of nitrogen narcosis; the risk is purely the reduction of oxygen availability.

Oxygen (O_2) at ≤ 19% (Normobaric Hypoxia)

Standard sea-level oxygen is 21%. A drop to 19% or lower initiates **mild hypoxia**.

- **Visual Impairment:** The retina is the most oxygen-sensitive peripheral tissue. Dark adaptation (night vision) drops significantly.
- **Cardiac Output:** The heart rate increases (tachycardia) to maintain oxygen delivery to tissues.



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Carbon Dioxide (CO_2) at 0.8%–2% (Hypercapnia)

This is the critical “driver” of the toxicity in this environment. 2% CO_2 is 50 times the normal atmospheric level.

- **The Accelerator:** CO_2 is the body’s primary signal to breathe. At levels above 0.8%, the brainstem signals for deep, rapid breathing (hyperpnea). This **increases the volume of toxicants inhaled per minute** (Cee & Branch of Inorganic Methods Development, 1990).
- **Acidosis:** CO_2 dissolves in the blood to form carbonic acid (H_2CO_3), lowering blood pH, causing confusion and throbbing vascular headaches.

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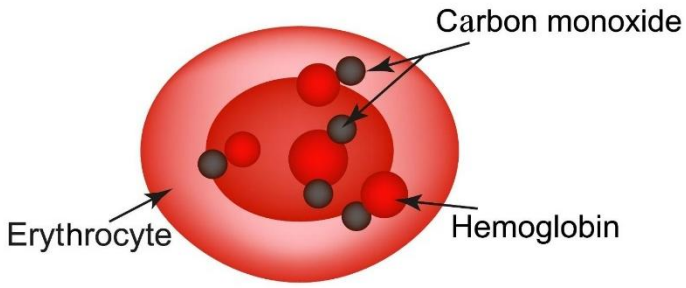
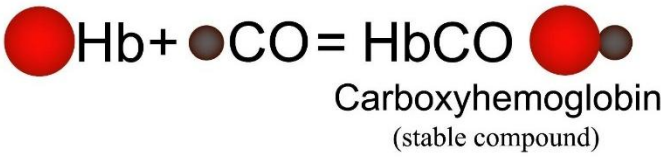
3. Analysis of Atmospheric Toxicants

Carbon Monoxide (CO): The “Hypoxia Multiplier”

In a standard atmosphere, low levels of CO are dangerous. In this **low-oxygen** atmosphere, they are rapidly lethal.

- **Accelerated Uptake:** Because the high CO_2 forces the subject to breathe 1.5x to 2x deeper and faster, CO is pulled into the bloodstream much faster than in a normal fire or leak scenario.
- **Hemoglobin Lockout:** CO binds to hemoglobin with **200–250 times** the affinity of oxygen, forming Carboxyhemoglobin (COHb). It effectively “evicts” the already scarce oxygen molecules.





Carboxyhemoglobin cannot carry oxygen and carbon dioxide

↓

Anoxaemia

↓

Suffocation

Mikrostoker (2016)

Monoethanolamine (MEA): The Systemic Toxicant

Source: Leakage from CO_2 scrubbing systems (amine beds).
Physiological Impact: MEA acts as both a local irritant and a systemic poison.

- **Respiratory Irritation:** MEA vapor is highly corrosive. It triggers **bronchospasm** (airway tightening). The subject is forced to hyperventilate (due to CO_2) through airways that are chemically burning and closing shut.
- **Hepatotoxicity (Liver Damage):** Once absorbed into the bloodstream, MEA accumulates in the liver. It causes **vacuolar degeneration** and necrosis of liver cells (hepatocytes), crippling the organ responsible for filtering the *other* toxicants (like Benzene) from the blood.
- **Nephrotoxicity (Kidney Damage):** The kidneys attempt to excrete MEA and its metabolites, leading to **renal tubular necrosis**. As kidney function fails, the body loses its ability to regulate the blood pH, which is already dangerously acidic due to the high CO_2 levels.

2,6-di-tert-butyl-4-nitrophenol (DBNP)

Source: Lubricating oil mists nitrated in electrostatic precipitators.
Physiological Impact: DBNP acts as a potent uncoupler of mitochondrial oxidative phosphorylation, forcing cells to consume oxygen and metabolic substrates without efficiently generating ATP manifesting as signs of catastrophic bioenergetic collapse rather than simple hypoxia (National Academies of Sciences, Engineering, and Medicine, 2008) .

- **Energy Blockade:** Because DBNP is slowly eliminated and distributes to multiple tissues, repeated low-level exposure in a closed submarine environment produces cumulative toxicity, so it functions both as an explicit toxicant and as an **amplifier** of the death spiral: low inspired oxygen and CO impair delivery, while DBNP prevents mitochondria from converting that limited oxygen into usable ATP, turning global hypoxia into true “cellular asphyxia” across heart, skeletal muscle, liver, kidney, and spleen (National Academies of Sciences, Engineering, and Medicine, 2008; Vesselinovitch et al., 1961).
- **Systemic cumulative toxicity and tissue deposition:**
  Because DBNP is lipophilic and slowly eliminated, repeated low-level exposure leads to body burden accumulation, visible yellow staining of skin and surfaces, and progressive multi-organ involvement even at doses below classical acute lethality thresholds, making long patrols uniquely hazardous (Alexander et al., 2001; Still et al., 2005; National Academies of Sciences, Engineering, and Medicine, 2008).
- **Hepatic and renal toxicity with reduced clearance capacity:**
  DBNP exposure causes fatty change in hepatocytes and zonal necrosis of renal tubular epithelium, along with increased blood urea nitrogen and decreased urinary concentrating capacity, eroding liver and kidney reserve needed to metabolize and excrete other contaminants in the toxic cocktail (Vesselinovitch et al., 1961;

National Academies of Sciences, Engineering, and Medicine, 2008).
- **Duplicitous Hazard:** CO prevents oxygen from *getting* to the cell; DBNP prevents the cell from *using* it.

Benzene: The Hematologic Time-Bomb

Sources:
- Missile Systems and Launch Tubes: A 2013 Navy directive confirmed missiles and missile tubes as sources of hazardous benzene. During a 2012 test onboard a submarine, benzene levels registered “in excess of OSHA-specified safe limits” inside these components. This critical finding exposed that these areas—previously unrecognized as hazardous confined spaces—required immediate mandatory benzene testing prior to personnel entry. Crucially, crew berthing is located within this same compartment, placing sleeping personnel in direct proximity to these toxic emissions (Dir. SSP, 2013).
- Volatile organic compounds (VOC).

Physiological Impact:
- **Central nervous system depression:** Benzene acts as a narcotic solvent causing headache, dizziness, confusion, tremors, and at higher concentrations, loss of consciousness and respiratory failure – effects that can be masked or misattributed in a CO_2-rich, low-oxygen environment (Agency for Toxic Substances and Disease Registry [ATSDR], 2007; ATSDR, 2023; U.S. Environmental Protection Agency [EPA], 2008)
- **Hematologic collapse:** Chronic inhalation of benzene disrupts hematopoiesis in bone marrow, causing decreased red blood cells, white blood cells, and platelets, which leads to anemia, immunosuppression, and bleeding risk in an already hypoxic, stressed crew (ATSDR, 2024; ATSDR, 2023; McHale, Zhang, & Smith, 2012)
- **Leukemogenesis and long-latency cancer risk:** Reactive benzene metabolites in marrow stem and progenitor cells cause DNA damage, chromosomal aberrations, and epigenetic changes, and clonal evolution toward acute myeloid leukemia and related hematologic cancers, converting a short-term atmospheric hazard into a decades-long oncologic burden (Hartley & Smith, 2010; McHale et al., 2012; Ross, 1996)
- **Multi-organ oxidative and inflammatory injury:** Biotransformation of benzene generates reactive oxygen species and protein/DNA adducts that damage liver, kidney, and other organs, amplifying systemic metabolic stress and reducing reserve capacity to cope with concurrent toxicants like CO, MEA, and DBNP (ATSDR, 2023; ATSDR, 2007; Chen et al., 2025)

Freon (Refrigerants)

Source: Cooling system leaks.
Physiological Impact:

- **Cardiac Sensitization:** Freon makes the heart muscle hypersensitive to adrenaline.
- **Sudden Death:** The stress (CO_2 anxiety + Hypoxia) floods the body with adrenaline. Freon can then trigger fatal ventricular fibrillation (Sabik et al., 2009).

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4. Synergistic Effects: The "Death Spiral"

The combination of these factors creates a feedback loop where each component makes the others more deadly:

Component A	Component B	Resulting Synergistic Failure
High CO_2	**Carbon Monoxide**	**Hyper-Absorption:** Heavy breathing from CO_2 draws CO into the blood significantly faster than normal.
MEA (Systemic)	**High CO_2 (Acidosis)**	**Metabolic Collapse:** High CO_2 makes the blood acidic; MEA destroys the kidneys, removing the body's only mechanism to fix that acidity.
Low O_2	**DBNP**	**Cellular Asphyxia:** CO stops oxygen transport; DBNP stops oxygen utilization. The cell starves from both ends (Still et al., 2002).
Benzene	**Low O_2**	**Anemic Hypoxia: Benzene disrupts bone marrow function,** causing anemia, which reduces the blood's capacity to carry the already limited oxygen supply (ATSDR, 2024; ATSDR, 2023; McHale, Zhang, & Smith, 2012).

Stress/Hypoxia	**Freon**	**Cardiac Arrest:** Hypoxia speeds up the heart; Stress releases adrenaline; Freon causes the heart to stop in response to that adrenaline (Sabik et al., 2009).

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5. Long-Term Health Consequences of Chronic "Toxic Cocktail" Exposure

The physiological impact of this environment extends far beyond the acute symptoms experienced during deployment. Unlike OSHA-regulated industrial settings—where exposure limits are based on intermittent, task-specific hazards within ventilated buildings using continuous fresh-air exchange via traditional HVAC systems—submariners operate within a sealed, fully enclosed atmosphere. Onboard air is regenerated and refreshed rather than replaced, allowing trace contaminants to persist and recirculate within the system. This results in continuous, 24/7 exposure for weeks or months at a time, fundamentally altering the toxicological profile when compared to standard industrial exposure models, which typically assume 8-hour workdays, defined recovery periods, and atmospheric dilution through outside air exchange.

The Latency Period and Systemic Accumulation

- **Dormancy:** Many of the illnesses associated with this environment lie dormant for years or decades before emerging with devastating impact.
- **Chemical Saturation:** A tangible indicator of this pervasive exposure is the distinctive "submarine smell" that permeates uniforms and porous materials, signaling that the crew is living in a chemically saturated environment. This suggests that chemicals like MEA and benzene are not just in the air but are being continuously absorbed through the skin
- **Medically Unexplained Chronic Multisystem Illness (MUCMI):** Similar to Gulf War Syndrome, the simultaneous exposure to multiple toxicants produces a "layered exposure scenario" where no single causal pathway explains the health outcomes.

Documented and Presumptive Long-Term Outcomes

Despite the lack of comprehensive studies on mixtures, the following long-term health issues have been identified in the Submariner population or linked to the specific toxicants present:

- **Oncological (Cancer):**
  - **Benzene:** Strongly associated with **Acute Myeloid Leukemia (AML)**, Non-Hodgkin lymphoma, and multiple myeloma.
  - **Asbestos:** Linked to **Mesothelioma** (latency 10–50 years) and bronchogenic lung cancer.
  - **Radiation Presumptions:** Colon, brain, bone, and stomach cancers are among the presumptive diseases linked to ionizing radiation exposure on nuclear vessels.
- **Respiratory and Systemic:**
  - **Chronic Respiratory Disease:** Including adult-onset asthma, chronic bronchitis, and potential pulmonary fibrosis from MEA and Ozone exposure.
  - **Organ Damage:** Chronic exposure to MEA and benzene metabolites is linked to long-term liver and kidney damage (hepatotoxicity and nephrotoxicity).
  - **Neurological:** Central nervous system conditions, tremors, and cognitive impairment resulting from chronic hypoxic and neurotoxicant (solvent) exposure.
- **Reproductive:**
  - Potential reduced male fertility and sperm motility issues, and higher rates of menstrual disorders and ovarian atrophy in females have been noted in association with benzene and hypoxic environments (SAG, 2025, p. 41).

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6. Conclusion

The physiological environment of a compromised submarine atmosphere is not merely a collection of isolated hazards, but a synergistic “toxic cocktail” that fundamentally challenges human biology. The interaction between **normobaric hypoxia**, **hypercapnia**, and **accelerated toxicant uptake** creates a systemic failure state that standard industrial safety models fail to predict. The reliance on hypobaric (high-altitude) research to justify safety limits for normobaric (submarine) environments represents a critical scientific gap. As evidenced by the “submarine smell” and the latency of severe illnesses, the body absorbs this toxic load continuously, leading to long-term oncological, respiratory, and organ-system damage that may not manifest until decades after service. Acknowledging this “Unseen Burden” requires shifting from isolated chemical analysis to a holistic understanding of chronic, combined physiological stress.

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