SUBMARINERS' ADVOCACY GROUP

Submariners Helping Submariners©

The Unseen Burden:

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

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The Unseen Burden:

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

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VFW Department of Wisconsin

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Michael W. Schroeder
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Deceased Submarine Veterans
(Possibly Submarine-Related Issues)

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Albers, Guy MM2(SS)
Alexander, Kent C. RM1(SS)
Anderson, Brent MT2(SS)
Anstey, Ron L. EM2(SS)
Arruza, Dwayne FTB1(SS)
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Barber, John G MM2(SS)
Barber, Michael QM#(SS)
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Bellerud, Bryon LT
Bender, Eric LT
Bennett, Michael QMC(SS)
Benton, Melvin MTC(SS)
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Bloomquist, Robert FTCM(SS)
Boley, Patrick M. LT
Boutilier, Daniel CS1(SS)
Bright, Buzz QMCM(SS)
Brock, Rick ET1(SS)
Bullard, Alan ETCS(SS)
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Cain, Eugene T. YNC(SS)
Carman, Donald L.R. IC3(SS)
Carpenter, Randall D. MM1(SS)
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Conroy Danial MM1(SS)
Cook, Kraig MMCS(SS)
Cook, Michael MM1(SS)
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Crosier, Richard RMCM(SS)
Crowley, Paul STSC(SS)
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Sipes, Brannon MT1(SS)
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Slattery, John IC1(SS)
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Stohlman, Paul MTC(SS)
Strakos, Rodney MTC(SS)
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Tubo, Dennis SKCS(SS)
Turner, Thomas W. CAPT
Underwood, Calvin MT2(SS)
Upp, David A. MT3(SS)
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Willis, Michael K. SN(SS)
Wilson, Renzor TM2(SS)
Woodard, Chad A. FT2(SS)
Yeagar, Brian MM2(SS)
Yeager, Paul MM1(SS)
Zehe, George RM1(SS)
Zipay, Dennis MT1(SS)

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(Possible Submarine-Related Issues)

Abbey, David T. SK2(SS)

Adcock, Mark C. FT2(SS)

Aljets, Jason T. MM2(SS)

Allison, Charles MM2(SU)

Allison, Jay MM2(SS)

Alton, Michael H. SKC(SS)

Anderson, David STS2(SS)

Andrews, Justin MTCS(SS)

Armstrong, James B. EMC(SS)

Arbour, William ET1(SS)

Ashmore, Andrew M. STSCS(SS)

Atkins, Richard A. YNC(SS)

Augenstein, Mischell RM(SS)

Aumen, Stephen J. MS2(SS)

Baloaloa, Teofilo R. TM2(SS)

Basara, Brent E. STS1(SS)

Beglau, William M. LCDR, prior EMC(SS)

Bell, Brian E. EM1(SS)

Bell, Philip K. ET2(SS)

Bennett, Randall D. YNSN(SS)

Benson, Richard P. MM1(SS)

Benuska, Nick K. MM2(SS)

Biggs, E. Spencer Codee MTCS(SS)

Billingsley, Boyd MM3(SS)

Biscoe, Thomas R. HMCM(SS)

Bolger, William ETCS(SS)

Bond, James R. EM1(SS)

Bonnington, Thomas E. QMC(SS)

Bouch, David STS1(SS)

Boughton, Craig D. IC2(SS)

Broughton, Michael D. TM2(SS)

Bowles, Dale A. FT1(SS)

Bracha, Steven A. ET1(SS)

Brandenburger, Roger L. MMFN(SS)

Brashler, Richard L. MS3(SS)

Brewer, Jack STSCS(SS)

Brooks, George, J. TM3(SS)

Brooks, Kevin QM2(SS)

Brou, Patrick T. EMN2(SS)

Broughton, Randall L. MM3(SS)

Brown, Charles S, LT

Brummet, Bryan S. STSCS(SS)

Bryant, James K. EM2(SS)

Buffa, Scott M. MT2(SS)

Bugh, Michael W. MMFN/(S)

Butler Richard QM3(SS)

Byers, Charles H. MM1(SS)

Calabrese, Michael J, ETCS(SS)

Callahan, Timothy STS1(SS)

Calzadillas, Samuel J. ET2(SS)

Carreira, William A., Jr. ETCS(SS)

Cartwright, Gerald M. RMCM(SS)

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Dunbar, William STS1(SS)
Duncan, David IC2(SS)
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Gainey, Thomas MT2(SS)
Gale, Robert G. EM2(SS)
Galeani, John CS2(SS)
Gardner, Thomas G. EM1(SS)
Gates, Michael L. TMC(SS)
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Gilbert, James W. MM1(SS)
Giles, Raymond E. MMN2(SS)
Gillard, Roderick K. MM1(SS)
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Goodman, Robert E. MT2/IC1(SS)
Graves, Joseph MM2(SS)
Grundleger, Adam H. MM1(SS)
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Haas, Gary M. MMC(SS)
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Harrell, Mark MT2(SS)
Harris, Eugene J. MS2(SS)
Harris, Jefferson W. MT2(SS)
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Hillis, Jack D. ETCS(SS)
Hinderliter, John D. STSCS(SS)
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Hoffmann, Andrew MM3(SS)
Holland, Steven RM1(SS)
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Horne, Thomas E. STS1(SS)
House, Brian W. MM1(SS)
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Mortimer, Vaughn D. ET2(SS)
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Norman, Carl MM3(SS)
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Pattison, Glynn MM1(SS)
Pattison, Lynn MM1(SS)
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Tarnow, Raymond P. MT2(SS)

Taylor, Scott MM1(SS)

Terzigni, Frank C., Jr. MM3 (SS)

Tetsuwari, Christopher MMA2(SS)

Thomas, Wayne C. FTB1(SS)

Thomason, Brian K. STS2(SS)

Thompson, Clark S. MS1(SS)

Thurmon, Dennis L. MM2(SS)

Trowbridge, Charlie EM1(SS)

Tyrell, James P. MT2(SS)

VanNoy, Nathaniel K. MM1(SS)

Vegh, John E. STG1

Villiard, Ardi D. MTCS(SS)

Vo, Johnny, V. ITEC(SS)

Walker, Charles P. MS1(SS)

Walker, Roger D CTM2

Walton, Steven B. HMCM(SS)

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Warczynski, Leon M., III ET2(SS)

Waterman, Karl L. STS1(SS)

Weinstein, David HMC(SS)

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Introduction

8 November 2025

Dear Reader,

Thank you for taking the time to read this report, ***The Unseen Burden***, which addresses the diagnosed and undiagnosed medical struggles associated with service in the U.S. Navy's Submarine Force, also known as the Silent Service. The Submariners' Advocacy Group (SAG) developed this report to raise awareness of the many illnesses linked to life aboard submarines—a highly technical and inherently dangerous environment.

The names contained in the dedication represent only a small fraction of the approximately 300,000 living veterans who have served in the Submarine Force since 1947. Each of these Sailors was exposed to more than 150 hazardous chemicals, gases, and toxicants present in submarine atmospheres. Those named here, along with tens of thousands of others who continue to suffer, can rightfully be considered casualties of the Cold War.

Too often, these ailments lie dormant for years or decades before emerging with devastating impact. In many cases, illnesses strike suddenly and without mercy, leaving service members and their families to endure both unimaginable suffering and severe financial hardship.

The Sailors memorialized in this report represent a small portion of a much larger reality. Every Submariner who ever sailed beneath the ocean's surface accepted the risks of serving in a hostile environment, hundreds of feet underwater, for months at a time. They did so out of duty, patriotism, and an unwavering commitment to excellence. Yet, they could not have fully known the long-term consequences of exposure to toxic chemicals and ionizing radiation unique to submarine service.

This report is both a testament to their sacrifice and a call to honor their memory. It is also the driving force behind the Submariners' Advocacy Group's mission: to ensure that the suffering caused by this unseen burden is neither ignored nor forgotten, but addressed with the urgency, respect, and care it deserves.

Sincerely,

Stanley J. Martinez
Chairman and Chief Executive Officer
Submariners' Advocacy Group

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Glossary

Acute Exposure: Exposure by the oral, dermal, or inhalation route for 24 hours or less (*IRIS Glossary*, 2025).

Amine or MEA: Monoethanolamine, used in CO_2 Scrubbers.

Chlorate Candles: Provide a backup supply of oxygen. When the candle burns it releases oxygen, particulate contaminants, and some chlorine. (NRC, 1988, pg. 16)

Chronic Exposure: Repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species) (*IRIS Glossary*, 2025).

Generators: Oxygen Generators

Hematotoxicity: "Refers to the adverse effects of substances or agents on the blood and blood-forming organs" (Lee, 2025).

Hyperoxia: An excess of oxygen in the system resulting from exposure to high oxygen concentrations, especially at hyperbaric pressures of oxygen.

Hypobaric: Pertaining to pressure of ambient gases below sea-level normal (<760 mmHg) (NRC, 2007, p. 254).

Hypoxia: A concentration of oxygen in arterial blood that is less than normal. (NRC, 2007, p. 255)

Normobaric: Denoting a barometric pressure equivalent to sea-level pressure (760 mmHg) (NRC, 2007, p. 254).

NRC: National Research Council

NRC COT: National Research Council's Committee on Toxicity

NSMRL: Naval Submarine Medical Research Laboratory

NUMI: Naval Undersea Medical Institute

OPSEC: Operational Security

PACT Act: The Sergeant First Class (SFC) Heath Robinson Honoring our Promise to Address Comprehensive Toxics

SAG: Submariners' Advocacy Group

Subacute Toxicity: Subacute toxicity refers to adverse effects that occur after repeated exposure to a substance for several weeks or months but less than 90 days (Sahu et al., 2017).

Subchronic Exposure: Repeated exposure by the oral, dermal, or inhalation route for more than 30 days, up to approximately 10% of the life span in humans (more than 30 days up to approximately 90 days in typically used laboratory animal species). [See also longer-term exposure.] (*IRIS Glossary*, 2025)

Executive Summary

The U.S. Navy's Submarine Force, comprising less than 7% of Navy personnel, operates approximately 22% of its combatant ships, embodying advanced technology and stealth crucial for national security. Despite their critical role and the inherent dangers of submarine duty, generations of Submariners are experiencing a broad spectrum of health issues, including rare diseases, cancers, blood disorders, immune system disorders, central nervous system conditions, and various respiratory system problems. A significant concern is the disproportionately high rate at which their claims under the U.S. Department of Veterans Affairs (VA) PACT Act are being declined compared to other veteran populations.

The core problem lies in the unknown number of Submarine Service veterans diagnosed with illnesses potentially linked to acute, subacute, and chronic exposure to toxic chemicals and biological contaminants within submarine atmospheres. This is exacerbated by a critical absence of consistent and accurate scientific data on submarine atmospheric toxicity, hindering the ability to definitively prove or disprove the link between exposure and illness. Furthermore, existing bureaucratic inefficiencies within the VA prolong claims processing, delaying essential healthcare and disability ratings for Submariners. This can lead to a Submariner's passing away without their rating ever being determined.

To address these pressing issues, this report proposes a multifaceted solution. First, PACT Act eligibility must be expanded to explicitly include Submariners who served on submarines from 1947 through the present day. Second, comprehensive, scientifically based studies on atmospheric contaminants are urgently needed across all classes of submarines, especially all operational classes of nuclear submarines. Third, VA claim processing efficiencies must be significantly improved by streamlining the application process, eliminating unnecessary complexities, and equipping Veteran Service Representatives (VSRs) with factual, data-driven evidence pertinent to submarine exposures.

This report serves as a direct call for swift and decisive leadership from the Secretary of War, the Department of the Navy, the U.S. Congress, and the Department of Veterans Affairs. It demands formal acknowledgment of the long-term health problems faced by Submariners, who repeatedly have been assured their work environments are safe. It emphasizes that current scientific studies are inconsistent and insufficient to characterize the hazards of exposure in this unique occupational setting. The report urges the immediate funding and commissioning of state-of-the-art scientific and medical studies to collect the necessary data to definitively assess the hazards associated with continuous exposure to submarine atmospheres.

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Preface: The Silent Service and Unseen Hazards

The United States Navy's submarines, affectionately known as "boats," represent the pinnacle of naval technology and stealth, safeguarding national interests and maintaining a silent global presence for over a century. Submarine duty is characterized by its arduous and challenging nature, undertaken solely by those who volunteer to undergo an extensive battery of physical and psychological tests, followed by up to two years of highly specialized technical training. Despite this rigorous preparation and the unwavering confidence placed in the Navy's assurances of safety, a profound question has emerged: "Are submarines safe for their crews, really safe?"

There are many unique factors to consider regarding the environment aboard a submerged submarine and how it differs from other industrial and military settings, including surface warships:

- **The submarine "world" shrinks.** Every space is confined, interconnected, and accessible to most of the crew while the submarine is deployed.
- **Crew movement is unrestricted.** With the exception of the Reactor Compartment, Sailors move throughout nearly all compartments for work, watch standing, or simply to pass time with shipmates.
- **Exposure is shared.** All hands are equally at risk of exposure to toxicants, hazardous chemicals, and radiation.
    - Many spaces for "non-nuclear" personnel are in close proximity to toxic, hazardous, and radiation exposure sources, i.e., crew berthing areas, workspaces, passageways, and other common areas.
    - Non-submarine-qualified sailors are required to become familiar with every space, system, and valve aboard as part of their qualification process.
    - All hands respond to casualties regardless of rank, rating, or assigned watch station.
- **The atmosphere is uniform.** A submarine's air is constantly recirculated by ventilation fans, creating an essentially homogenous environment under normal conditions. Only casualty events (real or training) disrupt this equilibrium.
- **Localized concentrations still exist.** Certain compartments present a higher risk due to the chemicals present:
    - Engine Room: 2190 lubricant oil and 2,6,-di-tert-butylphenol (DBP, an antioxidant). Submarine electrostatic precipitators nitrate DBP creating the toxicant, 2,6-Di-tert-butyl-4-nitrophenol or DBNP (Alexander et al., 2001, as cited by NRC, 2008, p. 88)
    - Auxiliary Machinery Spaces: Monoethanolamine (MEA) used in CO_2 Scrubbers.
    - Missile Compartment, Torpedo Room, and areas near the Diesel Fuel Oil Tank: Benzene exposure risk.

Submarine Periscope Operations

This critical inquiry was catalyzed by the Department of Veterans Affairs' press release on March 5, 2024, which expanded healthcare eligibility for all veterans exposed to toxicants during their service, whether at home or abroad (VA & Flynn, 2024). This announcement prompted a collective discourse among submarine veterans regarding the myriad toxic, hazardous, and radiation exposures they experienced while deployed in submerged environments. These discussions were fueled by countless anecdotal accounts of Submariners developing severe health issues, potentially as a direct consequence of their service. A striking, yet often overlooked, indicator of this pervasive exposure is the distinctive smell that clings to Submariners, permeating their uniforms and every porous material within the contained atmosphere of a submarine. This saturation of personal effects and shipboard surfaces by chemicals prevalent in the submarine's atmosphere raises fundamental questions about the long-term safety of such an environment, despite historical reassurances.

These discussions culminated in the establishment of the Submariners' Advocacy Group (SAG) in April 2024. Founded by 16 dedicated Submariners, SAG recognized the urgent need for collective action on behalf of the estimated 300,000 Submariners in the U.S. population, a demographic representing less than 0.1% of the population. SAG's core mission is to serve as a unified voice and to provide a comprehensive response to findings from studies and research conducted by the National Research Council's Committee on Toxicity, particularly concerning submarine atmospheric contaminants and hazards (NRC, 1988; NRC, 2007; NRC, 2008; NRC, 2009).

Historically, the "Silent Service" ethos, while vital for OPSEC, has inadvertently contributed to the marginalization of Submariners' health concerns. Their small numbers and the inherent secrecy surrounding their duties have resulted in significant under-representation when it comes to VA benefits, disability claims, and broader recognition of their service and the hazards they face. While landmark legislation such as the Agent Orange Act and the PACT Act have brought much-needed recognition and benefits to larger veteran populations exposed to hazards in other combat zones, the issue of submarine toxic exposures has remained largely unaddressed, with funding and emphasis disproportionately allocated elsewhere.

Although comprehensive in intent, this report is limited in scope to three gases, four chemical toxicants, asbestos, and radiation. In doing so, it only scratches the surface of the broader issue, as there are between 130 and 200 known contaminants present in submarine atmospheres (NRC, 1988, Table A-1, pp. 60–65). This reality underscores that the hazards faced by Submariners extend far beyond what is

captured in these pages. Accordingly, this report should be viewed as a catalyst—meant to spark conversations, guide future research, and inspire meaningful actions, policies, and legislation aimed at protecting those who serve beneath the sea.

This report is therefore intended to educate the public and initiate crucial conversations to demystify certain aspects of submarine life and the continuous, prolonged exposure to hazardous workplace environments experienced by Submariners. While acknowledging the necessity of OPSEC, all references provided herein are publicly available from reputable sources. The motivation for this report is deeply rooted in the unique bond shared among Submariners and a solemn commitment to the memory of shipmates who have suffered and died, possibly as a result of exposure to toxic chemicals, gases, bioaerosols, and radiation while serving on their beloved boats. Inspired by the Disabled American Veterans (DAV) and Military Officers Association of America (MOAA)'s report, "Ending the Wait for TOXIC-EXPOSED VETERANS," (DAV et al., 2024) this document aims to galvanize all veteran organizations to join SAG in advocating for congressional, VA, and administrative recognition of the profound contributions and unaddressed health needs of every Submariner.

USS LEWIS AND CLARK (SSBN 644)
41 For Freedom Fleet Ballistic Missile Submarine

USS DARTER (SS-576) snorkeling and filled with diesel exhaust.

The Submarine Atmosphere: A Unique and Challenging Environment

Duty aboard a submarine is inherently perilous, as these vessels are designed to operate within one of the most hostile environments known to humans: submerged in the world's oceans. Within their confines, submarines house a complex array of systems, explosives, and materials, all of which are capable of posing significant danger to the crew. This includes weapons such as torpedoes and missiles armed with immense explosive power, high-pressure air and hydraulic systems, intricate electrical and electronic systems, very large batteries, and flammable materials, such as hull and pipe insulation. All these components are contained within a pressure hull that provides a mere six-inch separation between the crew and the immense external sea pressure at operational depths.

From the moment a Submariner commences training and throughout their service, they are systematically conditioned to dismiss concerns about the boat's atmosphere. This conditioning is reinforced by assurances that the submarine's atmosphere is continuously monitored and maintained at levels deemed safe for human habitability. The Central Atmosphere Monitoring System (CAMS), in its various iterations (CAMS1, CAMS2, or CAMS2a), is presented as the "infallible guarantor" of atmospheric safety.

USS NAUTILUS (SSN 571)

The crew, known as Submariners, is expected to achieve expert proficiency in their respective fields. From the Commanding Officer, who bears ultimate responsibility for the vessel and its personnel, to the most junior Culinary Specialist, each crew member undergoes rigorous training to understand the function of every system and to respond effectively to any emergency. The "Submariner's Code" emphasizes mutual trust, as the survival of the submarine and its crew is fundamentally dependent on the collective expertise, proficiency, and unwavering trust among all members. The arduous qualification process, which can span up to 18 months and demands significant sacrifices in sleep and personal time, culminates in the awarding of the coveted Submarine Warfare Insignia, also known as "Dolphins," signifying entry into this exclusive fraternity.

The advent of nuclear power in 1955 irrevocably transformed submarine technology. This innovation provided an unlimited source of electricity and propulsion, allowing submarines to remain submerged for significantly longer periods than their diesel-electric predecessors. With this extended submerged capability, maintaining an acceptable atmosphere became paramount. This challenge was overcome by the

development of three crucial pieces of equipment: the Oxygen Generator, the CO_2 Scrubber (utilizing monoethanolamine, or MEA), and the CO-H_2 Burner. These systems enabled continuous atmospheric revitalization, removing the previous operational limitation of needing to surface or snorkel for air replenishment (Rigsbee, 1959).

A critical distinction in understanding potential health impacts faced by Submariners lies in the nature of their exposure. Unlike typical industrial settings where exposure to chemicals or other hazards is limited to an 8-hour workday, 5 days a week, Submariners are exposed to airborne toxic chemicals, biological agents, gases, and radiation 24 hours a day, 7 days a week, for the entire duration of their deployments, which can last for weeks or months (NSMRL, 1982, p. 2; Shea et al., 1984, p. 1). This continuous, prolonged exposure within a sealed environment fundamentally alters the toxicological profile and potential health outcomes compared to the intermittent exposures typically studied in civilian industrial contexts (Shea et al., 1984, p. 1). It is particularly concerning that the very factors used to establish atmospheric limits were, in some instances, “arbitrary” and dictated by the limitations of the atmosphere control equipment rather than solely by human physiological tolerance (Shea et al., 1984, p. 1). This implies that the health and safety of Submariners may have been secondary to technological feasibility or operational constraints from the outset of nuclear submarine operations, representing a foundational cause for the long-term health issues now observed in submarine veterans.

Submariner Conducting Torpedo Tube Maintenance

The “Silent Service” culture, while operationally beneficial for maintaining secrecy and mission effectiveness, has inadvertently created a systemic barrier to comprehensive health data collection and advocacy for Submariners. This report highlights that the small number of Submariners and their inherent silence have resulted in a failure to adequately account for them, leading to underrepresentation in data collection, VA benefits, and disability claims. This cultural emphasis on self-reliance, self-abnegation, and the extreme demands of submarine qualification likely discourages individuals from reporting their health concerns, preventing the aggregation of such concerns into a recognized public health issue. This highlights how the very strength of the submarine community’s operational culture inadvertently creates an unforeseen vulnerability regarding health monitoring and advocacy, making it imperative for groups like SAG to overcome this historical silence and ensure that the unique health challenges of Submariners are brought to light and addressed.

Critique of Existing Scientific Studies on Submarine Atmospheric Contaminants

The current body of scientific research concerning submarine atmospheric contaminants exhibits significant methodological flaws and critical data gaps, undermining its adequacy in addressing the unique exposure profiles of Submariners. These deficiencies form a central argument for the urgent need for new, comprehensive studies.

Inadequacy of Exposure Definitions

A primary criticism is the inappropriateness of the definitions for “acute exposure” (lasting 24 hours or less), “sub-chronic (“repeated exposure... (more than 30 days, up to approximately 90 days”), and “chronic exposure” (“repeated exposure... (more than approximately 90 days to 2 years)”) (*IRIS Glossary*, 2025) as applied to the submarine environment. These definitions, primarily derived from Auletta (1995) and typical industrial chemical exposure models, fail to capture the reality of submarine duty. Again, submarine crews are subjected to continuous exposure to contaminants 24 hours a day, 7 days a week, for the entire duration of a deployment, which can extend for “weeks or months”. This starkly contrasts with civilian industrial settings, where exposures are typically limited to an 8-hour workday, five days a week. Research projects based on shorter, intermittent exposures (i.e., 4, 6, or 8 hours with breaks) are therefore limited in providing meaningful data for the prolonged, continuous exposure experienced by Submariners. For example, the NRC COT subcommittee’s study on oxygen (NRC, 2007, pp. 252-277) was solely based on changes due to altitude, without considering the continuous nature of exposure to other atmospheric components.

Lack of Research on Chemical Mixtures

A critical toxicological oversight is the absence of comprehensive research on the effects of chemical mixtures within the submarine atmosphere. The National Research Council’s (NRC) own reports, including “Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 1” (2007), “Volume 2” (2008), and “Volume 3” (2009), explicitly acknowledge that:

> The committee did not address exposure to chemical mixtures. The potential for antagonistic, additive, or synergistic interactions between contaminants in the submarine environment is subject to substantial uncertainty, remains largely unexamined, and needs to be studied” (NRC, 2009, pp. 6-7).

Despite these direct recommendations, no detailed research or study of this complex chemical mixture is available in the publicly accessible record. Past studies have narrowly focused on individual chemicals or gases, neglecting to consider the submarine atmosphere as a holistic entity where multiple airborne chemicals and

gases could interact to produce entirely different and unknown byproducts through synergistic effects. The “Submarine Air Quality” report in Appendix A, specifically Table A-1, pages 60-65, lists 130 chemicals that could be submarine atmospheric contaminants (NRC, 1988). This means that the National Research Council (NRC) Committee on Toxicity's (COT) subcommittee, as directed by the DoD, focused only on 26 individual chemicals or gases, which is less than 20% of the known or possible contaminants present in submarine atmospheres.

In a sealed environment with continuous exposure to multiple chemicals and/or gases, the human body is subjected to a complex interplay of stressors. Individual chemicals, while having known effects, can combine to produce amplified, novel, or unpredictable toxicological outcomes. For instance, a hypoxic state (characterized by low oxygen levels) could alter metabolic pathways, making the body more susceptible to other toxicants. Another example is that elevated airborne concentrations of carbon dioxide can induce hyperventilation, increasing the intake of all airborne contaminants (NRC, 2007, p. 61). This suggests that the observed health issues in Submariners are likely not attributable to single agents but rather to a “toxic cocktail” effect, where the combined effect is greater than the sum of its individual parts. This necessitates a fundamental shift towards a holistic, systems-toxicology approach to studying the submarine atmosphere, recognizing the complex interplay of multiple stressors. Without understanding these interactions, effective prevention, diagnosis, and treatment for Submariners’ unique health conditions will remain elusive, and VA claims will continue to face an insurmountable burden of proof.

Flawed Assumptions: Normobaric vs. Hypobaric Hypoxia

A further question relates to the scientific validity of equating studies conducted in hypobaric hypoxia (HH) environments (i.e., higher altitudes with lower partial pressure of oxygen) with the normobaric hypoxic (NH) environment of a submarine. While a significant portion of the Earth’s human population lives at higher altitudes and is thus exposed to HH, the pressure experienced by a submarine crew is relatively constant and maintained roughly at sea-level pressure, classifying it as a normobaric environment. Although the NRC has suggested that HH studies “may be relevant” to understanding NH impacts on Submariners, there is a growing body of scientific evidence indicating that “hypobaric hypoxia induces different physiological responses compared with normobaric hypoxia” (Millet et al., 2012). Debevec and Millet emphasize that this notion cannot be directly translated to exposures of longer duration or generalized across a broad range of hypoxia/altitude applications, and that further strictly controlled studies comparing HH and NH during longer exposures are warranted (2014). Thus, research using HH environments with intermittent exposures is not representative of the continuous NH exposure in submarines and raises serious questions about whether the subcommittee made a flawed assumption by basing its recommendations on HH research for monitoring submarine atmospheres.

Outdated Studies and Unheeded Recommendations

A consistent pattern of outdated research and unheeded calls for further investigation hinders the scientific understanding of submarine atmospheric contaminants. Documentation reveals that knowledge of chemical hazards in submarines dates back to at least 1958. However, comprehensive surveys recommended by the 1988 NRC report, “Submarine Air Quality,” were not performed by the time the NRC Committee on Toxicity (COT) subcommittee was established in 2002. This failure to conduct essential surveys precluded a more thorough understanding of submarine atmosphere toxicity, leading to potentially inaccurate or inappropriate prioritization of chemicals and gases for study between 2002 and 2009. This inaction has resulted in the continued exposure of thousands of submarine crew members to these chemicals.

> NOTE: SAG has learned that several detailed atmospheric surveys were carried out on active submarines during the 1990s and early 2000s. However, the results and reports have all been classified **SECRET** by the Navy, making them inaccessible to the NRC and the public. SAG has requested that these reports be declassified and released.

Furthermore, neurobehavioral studies on carbon dioxide, which inform exposure limits, are largely from the 1970s, with only small, more recent studies (Sun et al., 1996; Yang et al., 1997) suggesting “significantly lower acceptable concentrations” than previous findings. Despite these newer findings, the NRC subcommittee recommended raising the continuous exposure guidance level (CEGL) for carbon

Submarine Emergency Surfacing

dioxide without first validating these results (NRC, 2007, p. 60). This pattern of outdated studies, unheeded recommendations, and reliance on “arbitrary” exposure limits points to a systemic, long-standing failure within the scientific and regulatory bodies responsible for managing submarine atmospheres and Submariner health. This is not merely a data gap but a deliberate or negligent omission of critical research despite explicit warnings and recommendations over decades. The failure to conduct comprehensive surveys or validate newer, more conservative findings indicates a consistent de-prioritization of Submariner well-being. This pattern suggests that current health concerns among Submariners are a predictable consequence of historical scientific negligence, placing an unfair burden on veterans seeking benefits and strengthening the argument for immediate and retroactive policy changes.

Detailed Analysis of Key Atmospheric Contaminants and Associated Health Impacts

The unique, sealed environment of a submarine necessitates a rigorous examination of specific atmospheric contaminants and their documented health impacts on personnel.

Oxygen (O_2): The Hypoxic Environment

The primary engineering challenge for the Navy's nuclear submarines was maintaining adequate oxygen levels for extended submerged operations. Oxygen Generators, which produce oxygen through the electrolysis of purified water using potassium hydroxide (KOH) as a catalyst, eliminated the need for submarines to surface and replenish their atmosphere (Rigsbee, 1959). Older submarines stored oxygen in banks for controlled release, while newer designs release it directly into the atmosphere from an Integrated Low Pressure Electrolyzer (ILPE).

Normal atmospheric oxygen content is approximately 20.9%. Submarine atmospheres are intentionally maintained at oxygen levels of 19% or lower. This policy is explicitly designed to "decrease the risk of onboard fires" (NRC, 2007, p. 255). However, the Occupational Safety and Health Administration (OSHA) defines a "hypoxic" or "oxygen-deficient" atmosphere as "consisting of less than 19.5% oxygen. Further, this type of atmosphere is immediately dangerous to life and health (IDLH)" (Silverman, 2021).

An informal survey of 239 Submariners, collectively representing over 1,500 man-years of service from the 1960s to the early 2000s, indicated that 69% of respondents experienced monitored oxygen levels below 19% during normal operations. This corroborates earlier investigations by the Naval Research Laboratory (NRL) and Naval Submarine Medical Research Laboratory (NSMRL), suggesting that nuclear submarines could operate at ≤19% oxygen, provided further research was conducted on the long-term effects of such levels in atmospheres containing trace contaminants (Knight & NSMRL, 1986). However, no further research of this nature has been identified.

Data from Hagar (2003), cited in the NRC's 2007 report, indicated average oxygen partial pressures of 148-149 mmHg in nuclear submarines, with ranges of 118–188 mmHg (NRC, 2007). When converted to percentages, these ranges equate to approximately 16.5%–26.3% oxygen. The presence of hyperoxic levels (i.e., 25–26% O_2), which are highly combustible and exceed the 22.4% threshold for hyperoxia defined by Dean and Stavitzski (2022), raises questions about the accuracy of these readings, potentially suggesting measurements were taken near oxygen bleeds rather than representing the general submarine atmosphere. Despite evidence of these hyperoxic conditions, the NRC subcommittee chose not to investigate the associated risks, such as oxygen toxicity onboard submarines (NRC, 2007).

Integrated Low Pressure Electrolyzer (ILPE)

A concerning development is the design of modern submarines with only one ILPE and no oxygen banks, relying solely on Chlorate Candles for emergency oxygen. This design choice poses significant risks for prolonged hypoxic exposure if the ILPE fails and when operational requirements prevent surfacing or snorkeling to ventilate. One Submariner reported that the ILPE had been offline for three months at sea, resulting in extremely low oxygen levels, often lower than 17%. This departure from Admiral Hyman Rickover's insistence on redundant critical equipment for crew safety raises questions about whether this is a cost-saving initiative without adequate consideration for crew health.

Prolonged exposure to a hypoxic atmosphere has been associated with a wide range of health issues affecting multiple organ systems:

- **Respiratory:** Tracheobronchitis, pulmonary edema, sleep apnea, pulmonary hypertension.
- **Cardiovascular:** Cardiovascular problems, tachycardia.
- **Hematopoietic System:** Disorders of the blood-forming system.
- **Neurological/CNS:** Cerebral edema, headaches, confusion, restlessness, impaired thinking and coordination, poor judgment, slurred speech, vision changes, loss of consciousness, seizures, coma.
- **Ocular:** Retinal hemorrhage (NRC, 2007, p. 256).
- **Cancers:** Colon cancer, prostate cancer, brain cancer, cancers of the lip, buccal cavity, and pharynx (NRC, 2007, p. 265).
- **Reproductive:** Possibly reduced male fertility issues (NRC, 2007, p. 267).

The NRC report noted a lack of studies on the subchronic effects of mild hypoxia on mood or cognitive performance and recommended prospective studies to evaluate Submariners for symptoms like headaches and fatigue associated with the mild hypoxic environment (2007, p. 271).

> **NOTE**: Submarine submerged deployments result in the crew experiencing **chronic** exposures and not **subchronic** as defined above.

The following table compares the Emergency Exposure Guidance Levels (EEGL) and Continuous Exposure Guidance Levels (CEGL) for oxygen, illustrating the shifts in recommended atmospheric content over time:

Exposure Level	Current U.S. Navy Values (1988)	NRC Recommended Minimum Values (2007)
EEGL		
1-hour	19.6% - 30.9%	14.75%
24-hour	19.6% - ~22.5%	17.75%
CEGL		
90 days	19.6% - ~22.5%	19.6%

Table 1: Emergency and Continuous Exposure Guidance Levels for Oxygen (atmospheric content by percentage) (from Table 11-3, NRC, 2007, p. 270)

The data presented in Table 1, particularly when compared with the informal survey results mentioned above, is alarming. This is especially true given that OSHA defines anything below 19.5% as oxygen-deficient and immediately dangerous to life and health (Silverman, 2021). The NRC's decision not to investigate hyperoxic conditions, despite Hagar's data suggesting that these conditions may be present on submarines, further highlights a potential gap in understanding the full spectrum of oxygen-related risks.

Carbon Dioxide (CO_2): The Unacknowledged Burden

The second most critical concern for nuclear submarine engineers was the effective removal of carbon dioxide (CO_2) from the submerged atmosphere. "CO_2 is heavier than air, and that contributes to the development of toxic exposure situations in enclosed spaces." (NRC, 2007, p. 46) The solution emerged with the development of the CO_2 Scrubber, which utilizes the chemical properties of monoethanolamine (MEA or amine) to absorb CO_2 from the air. While highly efficient, a residual portion of CO_2 remains, resulting in atmospheric levels significantly higher than those found in normal air. Normal atmospheric CO_2 content is approximately 0.04%.

The progression of carbon dioxide monitoring standards on submarines reveals inconsistencies over time. The "Proceedings of the Submarine Atmosphere Contaminant Workshop" recommended specific monitoring levels in parts per million (ppm): 25,000 ppm (2.5%) for 1 hour, 10,000 ppm (1%) for 24 hours, and 5,000 ppm (0.5%) for 90 days (Shea et al., 1984, pp. A4-12 – A4-14). By 1988, the NRC's "Submarine Air Quality" report listed CO_2 limits in percentages: 0.8% for 90-day, 4% for 24-hour, and 4% for 1-hour exposures (NRC, 1988, p. 5). However, the NRC's 2007 report, "Emergency and Continuous Exposure Guidance Levels for Selected

Submarine Contaminants: Volume 1," reverted to ppm for its recommendations, showing current U.S. Navy values and proposed/recommended levels.

The following table illustrates the evolution and inconsistencies in carbon dioxide exposure guidance levels:

Exposure Level	U.S. Navy Values (1984, 1988, 2007)	NRC Recommended Values (2007)
EEGL		
1-hour	2.5% (1984), 4% (1988), 4% (2007)	2.5%
24-hour	1% (1984), 4% (1988), 4% (2007)	2.5%
CEGL		
90 days	0.5% (1984), 0.8% (1988), 0.5% (2007)	0.8%

Table 2: Emergency and Continuous Exposure Guidance Levels for Carbon Dioxide (atmospheric content by percentage) (Shea et al., 1984, pp. A4-12 – A4-14; NRC, 1988, p. 5; NRC, 2007, p. 60)

Notably, the 90-day CEGL established in the 2007 NRC report—8,000 ppm (0.8%)—is approximately 20 times higher than the concentration of carbon dioxide in normal atmospheric air (0.04%). The rationale for raising the acceptable limit, rather than reducing it to more closely approximate natural levels, remains unclear. This inconsistency is especially concerning given the well-documented adverse health effects of prolonged CO_2 exposure, including cognitive impairment, respiratory distress, and potential long-term systemic impacts. These discrepancies in exposure standards underscore the urgent need for a comprehensive, evidence-based reassessment to ensure the health and safety of Sailors serving aboard nuclear submarines.

The NRC's report explicitly states that CO_2 concentrations between 7,000 and 300,000 ppm (Note: 0.7%–30%) can cause significant adverse effects in humans, including "tremor, headaches, chest pain, respiratory and cardiovascular effects, and visual and other central nervous system (CNS) effects" (2007, pp. 47-48). Furthermore, it identifies tremor, headache, hyperventilation, visual impairment, and CNS impairment as "key effects for setting EEGL and CEGL values" (2007, pp. 47-48). The report even notes that CO_2 exposures as low as 7,000 ppm can lower blood pH by up to 0.05 units (NRC, 2007, p. 51). Given these acknowledged risks

and the fact that effects can occur at 7,000 ppm (0.7%), the subcommittee's recommendation to establish the 90-day CEGL at 8,000 ppm (0.8%) appears contradictory to the data from the study and potentially exposes Submariners to unwarranted side effects.

The subcommittee's own statements further compound these concerns. In discussing the 90-day CEGL, the report highlights that the visual function findings from Sun et al. (1996) and Yang et al (1997) are "of greater concern," particularly because "there was no available 90-day study of neurobehavioral effects of CO_2 exposures" (NRC, 2007, p. 60). Despite this critical data gap and the acknowledgment that newer studies suggest "significantly lower acceptable concentrations," the subcommittee proceeded to recommend a CEGL above normal levels without the benefit of validated research.

Perhaps the most damning statement in the chapter on carbon dioxide is: "The possibility of increased inhalation of other toxicants as a result of CO_2-induced hyperventilation must be addressed" (NRC, 2007, p. 61). This directly implies that the elevated CO_2 levels, by inducing hyperventilation, could amplify the toxic effects of other contaminants present in the submarine atmosphere, compounding the health burden. If numerous toxicants are present, increasing the CO_2 CEGL without prior valid studies and research is a questionable scientific and medical decision.

While the 2007 NRC report did not identify specific long-term effects directly attributable to carbon dioxide content in submarine atmospheres, the potential for CO_2-induced hyperventilation to increase the inhalation of other toxicants suggests a risk for synergistic amplification of the toxic effects from other contaminants.

Monoethanolamine (MEA/Amine): The Pervasive Irritant

Submarine CO_2 Scrubber

Monoethanolamine (MEA), commonly referred to as amine, has been an integral component of submarine atmospheres for over 67 years, dating back to the commissioning of nuclear submarines equipped with CO_2 Scrubbers in 1957. These scrubbers, first utilized on vessels like the USS SEAWOLF (SSN-575), USS SKATE (SSN-578), and USS SKIPJACK (SSN-585), operate by absorbing carbon dioxide when MEA is cool and releasing it when MEA is hot, a process that relies on the chemical properties of MEA. As early as January 1959, the Navy was aware of MEA's potential toxicity and its "very low" tolerance level (Rigsbee, 1959). Despite this early knowledge and the expectation that alternative CO_2 removal systems would be

developed, MEA-based scrubbers remain in use nearly 68 years later, without a scientifically determined understanding of the risks associated with long-term chronic exposure.

The pervasive presence of MEA in the submarine environment is evident through anecdotal accounts: MEA vapor escapes from the scrubbers, permeating the entire submarine atmosphere, causing crew members' clothing to become saturated and leading to a distinctive smell. Over these six and a half decades, Submariners have reported experiencing a wide range of health issues potentially linked to MEA exposure, including:

- Sinusitis,
- Rhinitis,
- Adult-onset asthma,
- Lung issues,
- Kidney and liver issues,
- Prostate issues,
- And central nervous system disorders.

Despite these widespread reports, the Department of Veterans Affairs has not acknowledged MEA exposure as a service-connected toxic hazard, nor has it established a direct linkage to any health issue, resulting in a lack of disability benefits for submarine veterans. This situation is particularly egregious, given that, on average, it takes the VA 31.4 years to formally acknowledge a toxic exposure (DAV et al., 2024, p. 2); yet, Submariners have been waiting over twice this long for MEA-related recognition.

Studies and safety data sheets provide further details on the acute and chronic toxicity of MEA:

- **Acute Toxicity:** High concentrations of airborne MEA are known irritants to the skin, eyes, and respiratory tract in laboratory animals. Continuous exposure to high concentrations has induced lethargy in animals, and high oral doses have resulted in organ weight and histopathologic changes in the liver and kidneys, suggesting interference with lipid metabolism (NRC, 2007, pp. 196-197).
- **Chronic Health Effects (from Safety Data Sheet: Monoethanolamine):**
    - **Gastrointestinal Tract:** Swallowing MEA may lead to severe ulceration, inflammation, and possible perforation of the upper alimentary tract. (2015, p. 1)
    - **Kidneys and Liver:** Repeated overexposure can cause damage to these organs. (2015, p. 2)
    - **Respiratory System:** Inhalation may aggravate existing asthma and inflammatory or fibrotic pulmonary disease, leading to chronic bronchitis with cough and shortness of breath (2015, p. 2).
    - **Skin Contact:** Skin contact can aggravate existing dermatitis, and MEA can cause a skin allergy, leading to itching and rash even from very low future exposure. The safety data sheet advises avoiding breathing vapor and direct

skin contact, noting a “potential significant contribution to overall exposure by the cutaneous (skin) route, including mucous membranes and the eyes, either by contact with vapors or by direct skin contact with the substance” (2015, p. 2).

- **Nervous System:** Inhalation studies in laboratory animals suggest possible injury to the nervous system, with high exposure potentially affecting the central nervous system, causing lethargy, reduced alertness, and decreased activity levels (2015, p. 6).
- **Reproductive/Developmental Effects:** A laboratory study on rats given high doses of MEA by gavage showed increased embryofetal death, growth retardation, and some malformations, though its validity is questioned due to high doses and technical deficiencies (2015, p. 6).
- **Metabolic Disruption:** Exposure to MEA can interfere with lipid metabolism, potentially leading to altered triglyceride levels, which are associated with an increased risk of cardiovascular diseases (NRC, 2007, pp. 196-197).

A key study frequently cited by the NRC subcommittee, conducted by Weeks et al. in 1960, had significant limitations. The NRC report noted uncertainties in interpreting its findings, particularly regarding the extrapolation of animal data for developing exposure levels. It was observed that MEA vapor “apparently condensed on the inside of the inhalation chamber walls and other surfaces and was deposited in sufficient concentration…to make the hair and skin wet, greasy to the touch…” (NRC, 2007, p. 204). This deposition was associated with skin irritation. The subcommittee made a “conservative assumption” that reduced alertness and activity levels were primary effects for determining exposure guidance levels, despite no follow-up behavioral examinations being conducted. (NRC, 2007, pp. 197, 199, 200, 201, 204, 205)

Crucially, the NRC COT’s subcommittee report (NRC, 2007, Chapter 8) stated that “No atmospheric measurements of MEA on board submarines have been reported.” (NRC, 2007, p. 205) Furthermore, it noted a severe lack of data for several toxicity endpoints, including “chronic exposure effects, carcinogenicity, and male reproductive effects” (NRC, 2007, p. 196). This means that for 50 years, from 1957 to 2007, there were no quantitative measurements of MEA exposure or studies on its effects on submarine crews or veterans’ health. This represents a profound and unacceptable gap in scientific due diligence. While some industrial reports suggest “no systemic effects from industrial exposure” (Beard and Noe, 1981, as cited in NRC, 2007, p. 205), this cannot be compared to the continuous, 24/7 exposure experienced by submarine crews, which raises critical questions about cumulative effects from repeated and sustained exposures. The fact that MEA can be absorbed through inhalation, ingestion, and skin contact, and that a harmful contamination of the air can be reached rapidly on spraying or dispersing, further underscores the risk in a confined environment (*Ethanolamine (MEA)*, 2025).

2190 TEP and 2,6-di-tert-butyl-4-nitrophenol (DBNP)

The NRC has identified 2190 TEP (the primary lubricating oil for submarine machinery) and 2,6-di-tert-butyl-4-nitrophenol (DBNP) as toxic chemicals present in submarine atmospheres. (Still et al., 2002; NRC, 2008) DBNP is a contaminant formed by the nitration of an antioxidant found in turbine lubricating oil 2190 TEP. (Still et al., 2002; NRC, 2008) Its presence has been detected on submarine interior surfaces, eating utensils, dishes, and even on the skin of Submariners (Still et al., 2002; NRC, 2008).

DBNP is a potential health concern because it acts as an uncoupler of mitochondrial oxidative phosphorylation, a critical process for cellular energy production (Still et al., 2002; NRC, 2008). Studies involving adult male rats dosed orally with DBNP showed significant toxicity; 40% of rats receiving a high dose (40 mg/kg) died within 24 hours, and survivors exhibited severe symptoms, including:

- Prostration,
- Absence of auditory startle response,
- Reduced locomotor activity,
- Muscular rigidity for up to 8 days (Still et al., 2002).

Lower doses (15 mg/kg) resulted in elevated levels in various tissues 24 hours post-dosing, with particularly high concentrations in fat, followed by liver, kidneys, heart, lungs, brain, striated muscle, and spleen (Still et al., 2002; NRC, 2008). DBNP levels remained elevated in fat, liver, kidney, heart, and lungs for up to 144 hours, and in the liver for up to 240 hours (Still et al., 2002; NRC, 2008). These findings suggest that DBNP may accumulate in the body as a result of continuous or repeated exposures of short intervals (Still et al., 2002; NRC, 2008).

Beyond its direct toxicity, DBNP is related to 4-nitrophenol (4-NP), a known endocrine disruptor that can alter the central nervous system’s regulation of the reproductive system in females (Still et al., 2002; NRC, 2008). Studies in experimental and wild animals indicate that 4-NP can disrupt normal endocrine and neuroendocrine levels (Still et al., 2002).

While the provided information establishes DBNP as a known contaminant with concerning toxicological properties and bioaccumulation potential, detailed historical context regarding its specific use, comprehensive exposure levels, and a full spectrum of health conditions observed in Submariners directly linked to 2190 TEP or DBNP are not extensively documented in the provided material. This represents a gap that warrants further investigation.

Benzene: A Persistent Carcinogen

Benzene, a volatile organic compound (VOC) with well-documented health risks, has been a persistent contaminant in U.S. submarine environments since their inception, stemming from various historical and contemporary sources.

Historical Sources on U.S. Submarines: Historically, benzene was prevalent due to its widespread use in painting and cleaning activities. Naval personnel in the 1960s reportedly used benzene and benzene-containing products, often without protective gear, for tasks such as removing paint from hands. (Board of Veterans' Appeals, 1991) Painting duties frequently occurred below deck in enclosed spaces, significantly increasing the potential for exposure.

- The Bureau of Ships Technical Manual (November 1965) classified benzene as a “dangerous material” but still permitted its storage in paint and flammable liquid storerooms aboard ships (NRC, 2007, p. 45).
- By 1976, the Naval Ships’ Technical Manual was updated to explicitly list benzene as a material “not to be stored aboard ships and submarines” (NRC, 2007, p. 46).
- A 1982 letter from the Director of the Occupational and Preventative Medicine Division at the Department of the Navy indicated that benzene was “very likely” a constituent in paints, thinners, cleaners, polishes, or solvents used by naval personnel in the 1960s.
- Fleet-wide dissemination of safety standards requiring monitoring of confined spaces for benzene and carbon monoxide was not implemented until OPNAVINST 5100.23H in 2019, which defined confined spaces and clarified entry program requirements (U.S. Department of the Navy, 2019).

Submarine CAMS 2/2A

Present-Day Sources on U.S. Submarines: Despite improved monitoring systems like the CAMS 2/2a and additional regulations, benzene remains a concern. Following a 2007 recommendation from the National Research Council, the 90-day exposure limit for benzene air levels in U.S. Navy submarines was adjusted downward from 1.0 to 0.2 ppm in 2016 (U.S. EPA, 2016). While direct storage of benzene-based solvents is prohibited, paint and preservation activities in port continue to be a source of potential exposure through solvents like acetone. Although the U.S. Environmental Protection Agency (EPA) exempted acetone from the regulatory definition of volatile organic compounds (VOCs) in 1995 due to its low photochemical reactivity (U.S. EPA, 2024), this exemption disregards the inherent risk associated with benzene exposure.

Benzene in Tobacco Smoke: For decades, Submariners erroneously believed that onboard atmospheric equipment could effectively remove toxins from secondhand cigarette smoke. However, studies have revealed that non-smoking Submariners experienced significant involuntary benzene exposure from secondhand smoke, ultimately leading to comprehensive smoking bans (NRC, 2004; Sims et al., 1999).

Smoking policies evolved from unrestricted use in the 1970s to limited designated areas by 2000, but visual evidence, such as yellowing interior paint, indicated the ineffectiveness of air filtration systems in removing smoke. (NRC, 2004) A landmark study on nine submarines found that post-deployment cotinine levels in nonsmokers were 2.1 times higher than pre-deployment levels, confirming involuntary secondhand smoke exposure (Kassem et al., 2014). Air quality monitoring during temporary smoking bans demonstrated significant decreases in aerosol concentrations, directly implicating cigarette smoke as a primary contaminant (NRC, 2004). Empirical evidence of harm to nonsmokers ultimately led to the U.S. Navy's 2010 smoking ban, aimed at protecting nonsmokers (NRC, 2004).

Gender-Specific Metabolic Processing and Toxicokinetic Variability: Recent research has uncovered critical sex-based differences in benzene toxicity, with women exhibiting 23–26% higher metabolization rates than men under equivalent exposure conditions (Chen & Wang, 2023). While animal models suggest greater hematotoxicity in male rodents (Lee et al., 2021), human epidemiological data indicate that women face an elevated risk of benzene-induced blood dyscrasias (GHO, 2022) and hematopoietic malignancies (Martinez et al., 2020). These divergences are attributed to physiological factors, including variations in body composition and hormonal influences on metabolic enzymes (Kimura, 2019). Longitudinal data from 218,061 Chinese workers reveal gender-specific vulnerabilities, including a 14.2% abnormality rate in white blood cells for women compared to 8.7% in men (Liu et al., 2021) and a 9.5% reduction in the prevalence of platelets in women (GHO, 2022). Women also face a 1.92-fold increased risk of acute myeloid leukemia per ppm-years of exposure compared to 1.37-fold in men (Martinez et al., 2020), with X-chromosome inactivation patterns potentially modulating this risk (Fernández et al., 2021).

Health Conditions Directly Linked to Benzene Exposure: Benzene is associated with a wide array of acute and chronic health conditions, impacting hematological, immunological, carcinogenic, and systemic functions.

- **Blood Cell Abnormalities:** Chronic benzene exposure disrupts hematopoiesis, leading to reductions in red blood cells (anemia), white blood cells (leukopenia), and platelets (thrombocytopenia). Workers exposed to benzene levels as low as 1 ppm have shown 8–15% decreases in total white blood cell counts (Aksoy et al., 1987). Higher exposures (15–650 ppm) can cause severe pancytopenia, a simultaneous decline in all three blood cell types (Aksoy & Erdem, 1978).
- **Bone Marrow Suppression:** Benzene metabolites, such as hydroquinone and phenol, directly damage bone marrow stromal cells and hematopoietic stem cells, leading to hypocellular (underdeveloped) or aplastic (nonfunctional) bone marrow (National Library of Medicine, 1998). In extreme cases, myeloid metaplasia, where blood cells form in the liver or spleen, has been observed (U.S. EPA, 2016).
- **Immunosuppression:** Benzene exposure reduces CD4+ T-cell counts and disrupts the CD4+/CD8+ ratio, impairing cellular immunity (Aksoy et al., 1987) and may increase susceptibility to infections.

- **Carcinogenic Effects - Leukemia:** Benzene is classified as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC). Acute myeloid leukemia (AML) is the most strongly associated cancer, with studies showing a 2–3 fold increased risk in benzene-exposed workers (American Cancer Society, 2023). Preleukemic conditions like myelodysplastic syndrome (MDS) often precede AML (U.S. EPA, 2012).
- **Other Hematologic Cancers:** Non-Hodgkin lymphoma (NHL) and multiple myeloma have also been linked to benzene, though with less consistent evidence than for AML (U.S. EPA, 2016).
- **Central Nervous System (CNS) Effects:** Acute exposure to high benzene concentrations (≥500 ppm) causes dizziness, headaches, and tremors due to its narcotic effects (National Library of Medicine, 1998).
- **Dermatological Effects:** Skin contact with liquid benzene results in irritation, erythema, and blistering, while prolonged exposure can lead to dermatitis (U.S. EPA, 2016).
- **Fertility Issues:** Female workers exposed to benzene show higher rates of menstrual disorders and ovarian atrophy, while male workers exhibit reduced sperm motility and count (U.S. EPA, 2012).
- **Fetal Toxicity:** Transplacental exposure in pregnant women is associated with low birth weight, congenital anomalies, and childhood leukemia (American Cancer Society, 2023).
- **Cardiovascular Toxicity:** Chronic exposure correlates with arrhythmias and cardiomyopathy, likely due to benzene's disruption of cardiac ion channels (American Cancer Society, 2023).
- **Hepatic and Renal Damage:** Benzene metabolites accumulate in the liver and kidneys, causing oxidative stress and fibrosis (National Library of Medicine, 1998).

Ozone

Ozone is a chemical that can be formed in electrical devices (Persson et al., 2002). In the context of a submarine, where air pollutants can accumulate due to prolonged submerged periods without proper venting, it becomes necessary to monitor compounds like ozone that might not typically be found in volatile concentrations in other environments (Persson et al., 2002). While nuclear submarines have long faced the challenge of air purification, and solutions developed for them may be adapted for other submarine types, the specific details regarding ozone's sources and historical context within the U.S. Navy submarine fleet are not comprehensively provided in the available material (Persson et al., 2002).

Despite the limited specific information on ozone in submarines, the general health effects of ozone exposure in enclosed environments are well-documented. Ozone is a powerful oxidant that can irritate the airways and cause health problems even at relatively low levels (U.S. EPA, 2025). When inhaled, ozone can cause damage to the lungs, resulting in chest pain, coughing, shortness of breath, and throat irritation (U.S. EPA, 2025). It can also exacerbate chronic respiratory diseases, such as asthma, emphysema, and chronic bronchitis, and compromise the body's ability to

fight respiratory infections (U.S. EPA, 2025). Higher exposures can lead to a build-up of fluid in the lungs (pulmonary edema), a medical emergency (NJ Dept of H&SS, 2003).

The effects of ozone on lung function include reductions in forced expiratory volume in one second (FEV1) and airway inflammation (U.S. EPA, 2025b). At the cellular level, ozone and its reactive intermediates can injure airway epithelial cells, triggering a cascade of inflammatory responses (U.S. EPA, 2025b). Other documented effects include increased small airway obstruction, decreased integrity of the airway epithelium, and increased nonspecific airway reactivity (U.S. EPA, 2025b). Individuals vary in their susceptibility, but those who are active outdoors, children, older adults, and people with pre-existing lung conditions are at a greater risk (U.S. EPA, 2025). Exercise during exposure increases the amount of ozone inhaled and the risk of harmful respiratory effects (U.S. EPA, 2025c). While recovery from short-term, low-level exposure can occur, health effects may become more damaging, and recovery may be less certain at higher levels or from longer exposures (U.S. EPA, 2025c).

The available information suggests that while ozone is a recognized atmospheric contaminant in submarines, detailed research on its long-term health effects on Submariners in this unique enclosed environment is not extensively provided. This represents a gap in the understanding of its full impact on this population.

Asbestos

Asbestos, a toxic, naturally occurring mineral, was extensively used in the construction of U.S. Navy submarines prior to the 1980s, with nearly 400 vessels containing the material (Danzinger, 2025). Its durability, heat resistance, and fireproofing qualities made it a popular choice for insulation around pipes, boilers, and engines, as well as in flooring, walls, and ceilings (Danzinger, 2025). The tightly sealed and poorly ventilated environment of submarines meant that microscopic asbestos fibers, once disturbed, could easily become airborne and recirculated throughout every compartment, leading to widespread exposure for crew members (Danzinger, 2025).

Submariners unknowingly inhaled these asbestos fibers during their time onboard. These fibers, once lodged in the lungs, abdomen, or heart, can lead to life-threatening diseases decades later (Danzinger, 2025). The latency period for asbestos-related cancers, particularly mesothelioma, can be 10 to 50 years or even longer, making early detection difficult and often leaving individuals unaware of their condition until it is advanced (Danzinger, 2025). Even short-term exposure can lead to long-term health issues (Danzinger, 2025).

Jobs with a high risk of asbestos exposure on submarines included engine room workers, electricians, insulators, machinists, pipefitters, plumbers, and welders (Dryfoos, 2025). Asbestos was concentrated in critical areas such as the control center, torpedo room, reactor compartment, sonar areas, and weapon and oxygen

storage areas (Veterans Guide, 2025). Friable asbestos, found in pipe lagging, sound insulation, and sheet gaskets, can easily release fibers, whereas non-friable asbestos in components such as engine gaskets and deck tiles can become friable through drilling, puncturing, or normal wear (Veterans Guide, 2025).

The most serious health condition directly linked to asbestos exposure is mesothelioma, a rare and aggressive cancer typically affecting the lining of the lungs (pleura), but also capable of impacting the abdominal or heart linings (Danzinger, 2025). Common symptoms include persistent cough, chest pain, shortness of breath, unexplained weight loss, and fatigue (Danzinger, 2025). Mesothelioma is considered a 100% disability by the VA, entitling affected veterans to significant monthly disability benefits (Wright, 2025).

Beyond mesothelioma, asbestos exposure can also lead to other serious conditions, such as asbestosis (lung scarring) and primary lung (bronchogenic) cancer (Veterans Guide, 2025). Families of Submariners were also at risk of secondhand asbestos exposure from fibers brought home on uniforms, tools, or equipment (Wright, 2025).

Although asbestos use has been regulated since the 1980s, and the military phased out its use during the 1970s, many veterans and former crew members still face the long-term health consequences (Danzinger, 2025). Some submarines commissioned after 1980, and still active today, such as the USS OHIO and USS MICHIGAN, have also been confirmed to have harbored asbestos (Veterans Guide, 2025).

Ionizing Radiation Exposure

Trident1 (C4) Handling Container

In March 2024, the VA expanded PACT Act eligibility under a classification called TERA, which stands for Toxic Exposure Risk Activity. It also established four separate cohorts for TERA eligibility (VA, 2024). Cohort 1 includes those who have been exposed to “Radiation… served on nuclear submarines and other nuclear ships or in shipyards” (VA, 2024). However, the specific details about radiation exposure levels or health effects within the VA documentation are limited and often unclear beyond general mentions. It’s important to recognize this as a separate toxic exposure pathway for submariners (VA, 2024).

In the early 1980s, the Navy established the “Man REM Reduction Program.” Among other things, it established Missile Compartment Upper-Level (MCUL) as a radiation-restricted

area. This limited access and work times in MCUL. This has been confirmed by many veteran Submariners who served during that time and afterwards. The program mentioned that radiation exposures were above the normal.

Nuclear-powered submarines inherently involve exposure to ionizing radiation due to the presence of nuclear-powered propulsion plants and the possibility of carrying nuclear weapons. The VA recognizes that military service can expose individuals to ionizing radiation, potentially leading to long-term health effects. Presumptive diseases linked to ionizing radiation exposure include a range of cancers, such as:

- Bile duct cancer
- Bone cancer
- Brain cancer
- Breast cancer
- Colon cancer
- Esophageal cancer
- Gallbladder cancer
- Stomach cancer
- Leukemia (excluding chronic lymphocytic leukemia)
- Lymphomas (excluding Hodgkin's disease)
- Multiple myeloma
- Pancreatic cancer
- Pharynx cancer
- Ovarian cancer
- Prostate cancer
- Rectal cancer
- Respiratory tract cancer (including lung cancer)
- Salivary gland cancer
- Small intestine cancer
- Urinary tract cancer (kidney, renal pelvis, ureter, and urinary bladder).

Non-cancerous conditions linked to radiation exposure include:

- Posterior subcapsular cataracts
- Non-malignant thyroid nodular disease
- Parathyroid adenoma

The long-term health implications of this exposure require diligent monitoring and recognition for all submarine veterans, regardless of NEC/MOS designation (VA, VHA, n.d.).

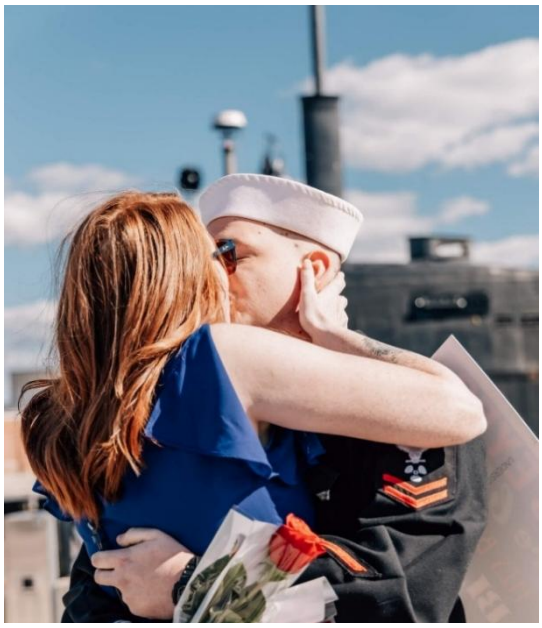
Data Gaps for Other Contaminants

While the provided material identifies 2190 TEP, DBNP, Ozone, and Asbestos as chemicals for examination, it is important to note that detailed information on their specific sources, historical context, high-risk occupational groups and job duties, and

comprehensive health conditions directly linked to their exposure within the submarine environment is not consistently available. This highlights a broad data gap in the existing research, emphasizing the need for more targeted and thorough investigations into all potential 130 to 200 Navy-identified contaminants (NRC, 1988, pp. 60-65). The NRC's own reports have repeatedly called for a "full analysis of the submarine atmosphere" and acknowledged that "the submarine atmosphere does not appear to be well characterized" (NRC, 2007, p. 5). This lack of comprehensive, scientifically based, and peer-reviewed surveys of the entire submarine atmosphere and exposed populations, particularly concerning the synergistic interactions of multiple airborne chemicals and gases, remains a critical deficiency.

The primary difficulty lies in attempting to correlate a discrete disease with a discrete toxic chemical or gas. Submarine service involves **simultaneous, chronic exposure to multiple contaminants**, producing conditions where interactive or cumulative effects are both likely and largely unstudied. This layered exposure scenario is analogous to Gulf War Syndrome, categorized as a medically unexplained chronic multisystem illness (MUCMI), in which no single causal pathway adequately explains the health outcomes observed. The question of why some Sailors develop debilitating diseases while others remain unaffected cannot be answered within the current research framework. Without a systematic investigation into the **cumulative, synergistic, and long-term effects of combined exposures**, this remains a critical and unresolved data gap with direct implications for force health protection.

The Human Cost: Submariners' Suffering and Disproportionate Denials



The human cost of unaddressed toxic exposures in the submarine environment is borne by generations of Submariners now suffering from a wide array of debilitating health issues, ranging from rare cancers, blood disorders, cardiovascular issues, chronic respiratory problems, and sleep apnea. These conditions often manifest decades after service, leaving veterans in a prolonged and often isolated struggle for recognition and care. The personal accounts, such as a Submariner who experienced prolonged low oxygen exposure at 14% for three months at sea, underscore the severity of these unacknowledged incidents and the lack of proper medical record-keeping.

A significant challenge for these veterans is the disproportionately high rate at which their claims, filed under the PACT Act, are being denied by the VA. While the PACT Act was designed to expand healthcare and benefits for veterans exposed to various toxic substances, its application to Submariners appears to be hindered (VA, 2025). The VA's typical timeframe for formally acknowledging an exposure is 31.4 years from the first incidence. However, for Submariners, the wait for recognition of exposures like MEA has been over twice as long, with no VA linkage or service-connected disabilities conceded for MEA exposure despite 67 years of documented use. This prolonged delay and denial of benefits for a population that was assured of their safety represents a profound breach of trust.

The lack of consistent and accurate scientific data on submarine atmospheric toxicity and ionizing radiation exposure directly contributes to these denials, as it creates an insurmountable burden of proof for individual veterans to demonstrate a direct service connection for their illnesses. The existing bureaucratic processes within the VA further extend claims processing times, exacerbating the suffering of Submariners seeking healthcare and disability ratings. This situation highlights a systemic failure to account for Submariners and their unique exposures, leading to their under-representation in benefit and disability recognition.

Submariner Homecoming – Welcome Home Daddy!

Policy and Legislative Imperatives

Addressing the profound health crisis among U.S. Navy Submariners necessitates immediate and comprehensive policy and legislative action. The current framework, while expanded by the PACT Act, still falls short in adequately recognizing and compensating Submariners for their unique toxic exposures.

Current Limitations of the PACT Act for Submariners

The Sergeant First Class Heath Robinson Honoring Our Promise to Address Comprehensive Toxics (PACT) Act of 2022 represents a significant expansion of VA health care and benefits for veterans exposed to toxic substances, including burn pits, Agent Orange, and radiation (VA, 2025). It has added numerous presumptive conditions and expanded eligible locations for exposure (VA, 2025).

However, the PACT Act does not explicitly list submarine service as a presumptive exposure location or specific submarine-related illnesses as presumptive conditions, outside of general radiation exposure for nuclear technicians or those involved in nuclear weapons handling (VA, 2025). This omission results in Submariners, despite their continuous exposure to a unique cocktail of chemicals in a sealed environment, facing disproportionate denial rates for their PACT Act claims. The burden of proof remains on individual Submariners to establish a direct service connection for conditions that are likely consequences of their unique operational environment.

The Role of Veteran Organizations

Veteran organizations such as the Disabled American Veterans (DAV) and the Military Officers Association of America (MOAA) have been instrumental in advocating for toxic-exposed veterans, notably through their “Ending the Wait for TOXIC-EXPOSED VETERANS” report, which served as a direct motivation for this report. These organizations collectively advocate for systemic changes to ensure that all veterans receive the care and benefits they have earned. However, all major veterans’ organizations overlook or have forgotten about submarine veterans.

SAG was founded specifically to be the voice of the Silent Service, recognizing that their small numbers and historical secrecy have led to a significant under-representation in veteran benefits and disability recognition. This report is part of SAG’s efforts to shed light on the exposures and lifelong health impacts experienced by Submariners.

Call for Amended PACT Act Eligibility for Submariners

To rectify the current inequities, PACT Act eligibility must be amended to explicitly include Submariners who served on all submarines from 1947 to the present day, especially those on nuclear-powered submarines. This amendment would recognize the inherent and continuous toxic exposures unique to the submarine environment,

establishing a presumptive link between service and the wide array of health conditions observed in this population. Such a policy change would alleviate the immense burden of proof currently placed on individual veterans and streamline access to critical healthcare and disability compensation.

Demand for Scientifically Based Studies on Submarine Atmosphere Contaminants

Submariners Standing Watch Underway

A fundamental requirement for long-term solutions is the commissioning of comprehensive, scientifically based studies on atmospheric contaminants across all previous and operational classes of nuclear submarines currently in the Navy's warship inventory. Existing research has been criticized for its methodological flaws, including the use of inappropriate definitions of exposure duration, a critical lack of investigation into chemical mixtures and synergistic interactions, and flawed assumptions in equating hypobaric hypoxia research with the normobaric hypoxic environment of submarines. These new studies must employ advanced atmosphere monitoring and diagnostic equipment onboard deployed submarines to collect accurate, real-time data on all atmospheric components, and studies to explore their interactions and the health effects of combined exposures. The results must be published for peer review to ensure scientific rigor and transparency.

Demand for Scientifically Based Studies on Historic Submarine Atmosphere Contaminants Linked to Studies on Health Outcomes in Historic and Current Submariners

All atmospheric and radiation survey records from 1960 to 2000 should be declassified and made public. Studies should then assess the impact of the findings on the health of the veteran submarine crew members. Older Submariners deserve retroactive VA recognition and appropriate disability ratings for related health issues. Further, while there are a limited number of epidemiological studies on mortality of Navy veterans who served on submarines, there have been few studies on occupational risks or adverse health outcomes (particularly respiratory, cardiovascular, oncological, and neurologic morbidity) linked to specific exposures in

these unique environments. A formal health registry (similar to the Airborne Hazards and Open Burn Pit Registry) linked to exposure information for Submariners must be established by the VA.

Need for Improved VA Claim Processing Efficiencies

Beyond legislative changes, immediate improvements are needed in VA claim processing efficiencies for Submariners. These include:

- Removing unnecessary layers of complexity in the application process
- Updating claim forms to reflect the unique exposures of Submariners
- And providing Veteran Service Representatives (VSRs) with factual and data-driven evidence specifically tailored to determining disability eligibility for this population.

The current system's inefficiencies contribute to prolonged wait times and disproportionate denials, exacerbating the suffering of veterans already battling service-related illnesses.

Department of Veterans Affairs Treatment Facility

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Recommendations and Call to Action

The evidence presented in this report underscores a critical and long-standing public health crisis affecting U.S. Navy Submariners. Decades of continuous exposure to a complex mixture of toxic chemicals, gases, and radiation within a sealed, oxygen-deficient environment, coupled with inadequate scientific study and policy recognition, have resulted in a profound human cost. To address this, the following recommendations and calls to action are imperative:

1. **Formal Recognition of Submariner Exposures:** The Secretary of War, the U.S. Congress, and the Department of Veterans Affairs must swiftly and aggressively acknowledge that Submariners, from 1947 to the present day, have faced chronic toxic and hazardous exposures in their unique operational environment. This acknowledgment should explicitly recognize that assurances of a non-hazardous atmosphere and low radiation exposure were inconsistent with the realities of continuous exposure to known contaminants, hazards, and radiation.
2. **Expanded PACT Act Eligibility:** Congress must amend the PACT Act to include all Submariners who served on submarines from 1947 through the present day as a presumptive exposure group. This will establish a clear service connection for a range of presumptive conditions, including but not limited to the respiratory, cardiovascular, cardiopulmonary, immune system, neurological, and carcinogenic health issues detailed in this report, thereby alleviating the unfair burden of proof on individual submarine veterans.
3. **Comprehensive Scientific and Medical Studies:** The Department of War and the VA must immediately commission and fund scientifically rigorous, independent studies on the submarine atmosphere. These studies must:
    - Conduct a full and accurate characterization of the submarine atmosphere, identifying all chemical, gaseous, and biological contaminants present across all operational classes and ages of nuclear submarines.
    - Investigate the effects of chemical mixtures, including potential antagonistic, additive, and synergistic interactions, rather than focusing solely on individual contaminants and gases.
    - Utilize advanced atmosphere monitoring and diagnostic equipment onboard deployed submarines to collect real-time, long-term data.
    - All collected historical atmospheric and radiation data must be made available, fully and unredacted, to the scientific and submarine veteran communities.
    - Focus on the effects of continuous, 24-hour-a-day exposure in a normobaric hypoxic environment, rather than relying on inappropriate industrial or hypobaric hypoxia models.
    - Evaluate the long-term health effects, including acute, subchronic, and chronic exposures, on all physiological systems.
    - Publish all research findings for independent peer review and public dissemination.

4. **Improved VA Claims Processing:** The Department of Veterans Affairs must undertake an immediate overhaul of its claims processing system for veterans who served on submarines. This includes:
    - Simplifying the application process and removing bureaucratic complexities that lead to disproportionate claim denials.
    - Updating Veteran Service Representative (VSR) training and resources to ensure they are fully informed about the unique toxic, hazardous, and radiation exposures of all Submariners and equipped with data-driven evidence to properly determine disability eligibility.
    - Fund a grant to create and publish a web-enabled reference guide on submarine terms, equipment, and chemicals for VSRs and Veteran Service Officers (VSOs).
    - Expediting the review of previously denied toxic-exposure-related and radiation-related disability claims from Submariners under the expanded PACT Act provisions (VA, 2024).
5. **Restoration of Redundancy in Oxygen Systems:** The Navy should re-evaluate the design of modern submarines that have reduced or eliminated redundant oxygen generation and storage systems. A return to Admiral Rickover's principles of robust redundancy for critical life support systems is essential to ensure crew safety and prevent prolonged hypoxic exposures during operational contingencies.

Conclusion

Submariner in Port Conducting Colors

The enduring legacy of the “Silent Service” is one of unmatched dedication, technical excellence, and sacrifice in defense of national security. Yet today, that legacy is overshadowed by the unaddressed health consequences of prolonged toxic, hazardous, and radiological exposures within the submarine environment. For decades, the unique challenges faced by Submariners have been minimized or ignored, resulting in systemic failures in scientific inquiry, policy oversight, and veteran care. The distinctive “submarine smell,” once dismissed as an inevitable characteristic of service beneath the sea, must now be recognized as a warning signal of chronic chemical saturation—one that demands rigorous, quantitative investigation.

The continued reliance on outdated and, at times, arbitrary exposure limits—set according to technological convenience rather than medical science—represents a foundational compromise of safety. These compromises have directly contributed to the health crisis now emerging among submarine veterans. Deficiencies in research design, the misapplication of exposure definitions, the disregard for complex chemical mixtures, and the failure to distinguish between normobaric and hypobaric hypoxia have left Submariners without the scientific validation necessary to substantiate their suffering. As a result, veterans have been systematically disadvantaged in their pursuit of recognition, treatment, and compensation, reflected in disproportionately high rates of claim denial.

This report is not merely an academic critique; it is an urgent demand for accountability. The United States government has an unambiguous moral obligation to honor the implicit contract made with its volunteer Submariners: that their health and well-being would never be sacrificed as the hidden cost of national defense. Fulfilling that obligation requires immediate action—expanding and clarifying eligibility under the PACT Act, commissioning independent and comprehensive research into submarine atmospheric contaminants and radiation exposure, and reforming VA claims processes to reflect the realities of submarine service.

Failure to act will perpetuate an avoidable injustice and erode trust between the nation and those who served it in the most demanding and unforgiving of environments. The time for silence has passed. The time for decisive action—in policy, science, and veteran care—is now.

References

Aksoy, M., & Erdem, S. (1978). Followup study on the mortality and the development of leukemia in 44 pancytopenic patients with chronic exposure to benzene. Blood, 52(2), 285–292. https://doi.org/10.1182/blood.V52.2.285.285

Aksoy, M., Dincol, K., Erdem, S., & Dincol, G. (1987). Hematotoxicity in 231 workers exposed to benzene-containing solvents and thinners in Turkey. European Journal of Hematology, 38(2), 161–165. https://doi.org/10.1111/j.1600-0609.1987.tb00001.x

Alexander, W.K., G.B. Briggs, K.R. Still, W.W. Jederberg, K. MacMahon, W.H. Baker, and C. Mackerer. 2001. Toxicity of 2,6-di-tert-butyl-4-nitrophenol (DBNP). Appl. Occup. Environ. Hyg. 16(4):487-495.

American Cancer Society. (2023, January 25). Benzene and cancer risk. https://www.cancer.org/cancer/risk-prevention/chemicals/benzene.html

Auletta, C.S. 1995. Acute, subchronic, and chronic toxicology. Pp. 69-127 in CRC Handbook of Toxicology, M.J. Derelanko and M.A. Hollinger, eds. Boca Raton: CRC Press.

Beard, R.R., and Noe, J.T. (1981). Aliphatic and alicyclic amines: Ethanolamine (2-Aminoethanol). Pp. 3168 in Patty's Industrial Hygiene and Toxicology, Vol. 2B. Toxicology, 3rd Rev. Ed., G.D. Clayton, and F.E. Clayton, eds. New York: John Wiley & Sons.

Board of Veterans' Appeals. (1991, September). *[Decision pertaining to Docket No. 91-13 740, BVA9421638]*. U.S. Department of Veterans Affairs. https://www.va.gov/vetapp94/files3/9421638.txt

Chen, L., & Wang, Y. (2023). Gender disparities in benzene metabolism: A pharmacokinetic analysis. *Toxicological Sciences, 189*(2), 45–60. https://doi.org/10.1093/toxsci/kfad012

Danzinger, P. (2025, June 13). *The Risk of Cancer from Being on a Submarine: Asbestos and Mesothelioma Explained*. Mesothelioma Lawyer Center. https://www.mesotheliomalawyercenter.org/blog/cancer-submarine-exposure/

DAV, MOAA, Jesinoski, B. A., & Kelly, B. T., USA (Ret). (2024). Ending the wait for Toxic-Exposed veterans. In *Ending the Wait for Toxic-Exposed Veterans*. https://www.dav.org/wp-content/uploads/EndingTheWait_Full-Report.pdf

Dean, J. B., & Stavitzski, N. M. (2022). The O2-sensitive brain stem, hyperoxic hyperventilation, and CNS oxygen toxicity. *Frontiers in Physiology, 13*. https://doi.org/10.3389/fphys.2022.921470

Debevec, T., & Millet, G. P. (2014). Discerning normobaric and hypobaric hypoxia: significance of exposure duration. *Journal of Applied Physiology, 116*(9), 1255. https://doi.org/10.1152/japplphysiol.00873.2013

Dryfoos, C. (2025, May 28). *Asbestos on submarines*. Mesothelioma Veterans Center. https://www.mesotheliomaveterans.org/veterans/navy/ships/submarines/

Ethanolamine (MEA). (2025). Toxic Free Foundation. Retrieved July 1, 2025, from https://thetoxicfreefoundation.com/database/ingredient/ethanolamine1

Fernández, A., López, R., & González, M. (2021). X-chromosome inactivation and benzene-related AML risk. Environmental Health Perspectives, 129(7), 077003.

GHO [Global Health Organization]. (2022). *Occupational exposure limits for chemical agents* (Report No. GHO-2022-441). https://www.who.int/publications

Gupta, S., Kumar, P., & Jain, N. (2021). Sex-specific CYP2E1 activity in benzene biotransformation. Archives of Toxicology, 95(8), 2789–2801.

IRIS Glossary. (2025, May 2). US EPA. https://www.epa.gov/iris/iris-glossary#s

Kassem, N. O., Kassem, N. O., Jackson, S. R., Liles, S., Daffa, R. M., Zarth, A. T., ... & Hovell, M. F. (2014). Hookah smoking: A growing threat to public health. *American Lung Association*.

Kimura, T. (2019). Hormonal modulation of metabolic enzymes. *Journal of Biochemical Toxicology, 33*(4), e22310.

Knight, R., MC,USN & NSMRL (Naval Submarine Medical Research Laboratory). (1986). *MEDICAL RESEARCH PROGRAMS, PAST AND FUTURE, FOR DESIGNING SUBMARINE ATMOSPHERES TO RETARD FIRES* (By C. A. HARVEY CAPT MC USN). https://apps.dtic.mil/sti/pdfs/ADA178354.pdf

Lee, H., Park, S., & Choi, J. (2021). Rodent models of benzene-induced hematotoxicity. *Toxicology Reports, 8*, 1534–1542.

Lee, S. (2025, June 14). *Understanding hematotoxicity in toxicology*. NumberAnalytics. Retrieved October 6, 2025, from https://www.numberanalytics.com/blog/ultimate-guide-hematotoxicity-toxicology

Martinez, R., García, M., & Díaz, F. (2020). Gender differences in benzene-related leukemia risk. *American Journal of Industrial Medicine, 63*(9), 789–797.

Millet, G., Faiss, R., & Pialoux, V. (2012). Point: Counterpoint: Hypobaric hypoxia induces/does not induce different responses from normobaric hypoxia. *Journal of Applied Physiology, 112*(10), 1783–1784. https://doi.org/10.1152/japplphysiol.00067.2012

National Library of Medicine. (1998). Benzene toxicity case studies. Agency for Toxic Substances and Disease Registry. https://www.ncbi.nlm.nih.gov/books/NBK591289/

Naval Submarine Medical Research Laboratory. (n.d.). *Diesel submarine atmosphere criteria and monitoring*.

NJ Dept of H&SS (New Jersey Department of Health and Senior Services). (2003). OZONE. In *New Jersey Department of Health and Senior Services*. Retrieved July 2, 2025, from https://nj.gov/health/eoh/rtkweb/documents/fs/1451.pdf

NRC (National Research Council). (1988). *Submarine Air quality: Monitoring the air in submarines: Health effects in divers of breathing submarine air under hyperbaric conditions*. Washington, DC: The National Academies Press. https://doi.org/10.17226/19146

NRC (National Research Council). 2004. Emergency and continuous exposure guidance levels for selected submarine contaminants. National Academies Press.

NRC (National Research Council). (2007). *Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 1*. Washington, DC: The National Academies Press. https://doi.org/10.17226/11170.

NRC (National Research Council). (2008). *Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 2*. Washington, DC: The National Academies Press. https://doi.org/10.17226/12032.

NRC (National Research Council). (2009). *Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 3*. Washington, DC: The National Academies Press. https://doi.org/10.17226/12741.

NSMRL (Naval Submarine Medical Research Laboratory). (1982, January 19). *The toxic effects of chronic exposures to low levels of carbon dioxide*. Defense Technical Information Center. Retrieved November 16, 2024, from https://apps.dtic.mil/sti/citations/ADA115053

Persson, O., Wadsö, L., Department of Heat and Power Engineering, Lund Institute of Technology, Lund, Sweden, Division of Building Materials, Lund Institute of Technology, Lund, Sweden, & Kockums AB, Malmö, Sweden. (2002). INDOOR AIR QUALITY IN SUBMARINES. In *Proceedings: Indoor Air 2002*. https://www.irbnet.de/daten/iconda/CIB7571.pdf

Rigsbee, J. T., USN. 1959. Man-made Air—The state of the art. *USNI Proceedings*, 85/1/671. https://www.usni.org/magazines/proceedings/1959/january/man-made-air-state-art#:~:text=The%20first%20carbon%20dioxide%20scrubbers%20installed%20in%20nuclear,were%20bulky%20and%20difficult%20to%20operate%20and%20maintain

Safety Data Sheet: Monoethanolamine [Online]; Carolina International Sales Co., Inc: Lavonia, GA, May 6, 2015. http://ciscochem.com/assets/monoethanolamine-sds.pdf (Accessed Nov. 7, 2024).

Sahu, R. P., Rajiv Gandhi Institute of Pharmacy, Faculty of Pharmaceutical Science & Technology, & AKS University Satna, MP-India. (2017). Principles of toxicology. *In Chapter-19* (pp. 172–175) [Book-chapter]. Retrieved September 8, 2025, from https://www.iipseries.org/assets/docupload/rsl20246CE780769D2B31C.pdf

Shea, M. L., Milroy, W. C., CAPT, MC, USN, Naval Submarine Medical Research Laboratory, Wands, R. C., Carhart, H., Wyatt, J., Williams, F., Einhorn, L., Nyers, R., Bondi, K., Lieberman, J., Eident, C., Goff, G., Gunter, D. O., Hienzsch, M., Kent, D. G., Knight, D. R., MC, USN, Lory, E., McQuire, E. K., CWO-4., . . . Wyatt, J. (1984). Proceedings of the Submarine Atmosphere Contaminant Workshop. In *Naval Submarine Medical Research Laboratory*. https://apps.dtic.mil/sti/pdfs/ADA145769.pdf

Sims, J. R., Tibbles, P. M., Jackman, R. P., Enzenauer, R. W., Watson, W. A., Jr, & Dinterman, R. E. (1999). *In Emergency and continuous exposure guidance levels for selected submarine contaminants* (NRC, 2004). National Academies Press.

(Singh & Patel, 2020)

Still, K. R., Jederberg, W. W., Briggs, G. B., Jung, A. E., Prues, S. L., Ritchie, G. D., Godfrey, R. J., Naval Health Research Center Detachment (Toxicology), GeoCenters, Inc., & ManTech Environmental Technology, Inc. (2002). *ABSORPTION, DISTRIBUTION, AND CLEARANCE OF 2,6-Di-tert-Butyl-4-nitrophenol (DBNP)*. Naval Health Research Center Detachment (Toxicology) - NHRC/TD. https://apps.dtic.mil/sti/tr/pdf/ADA411200.pdf

Sun, M., C. Sun, and Y. Yang. (1996). Effect of low-concentration CO2 on stereoacuity and energy expenditure. *Aviation, Space, and Environmental Medicine. 67*(1):34-39.

U.S. Department of the Navy. (2019, May 23). *Navy safety and occupational health manual* (OPNAVINST 5100.23H). https://cnreurafcent.cnic.navy.mil/Portals/78/NSA_Naples/Documents/Safety/Instructions/OPNAVINST%205100.23H%20Advance%20Change%201.pdf?ver=T9U3yXALZordfJqMTa-dIA%3D%3D

U.S. EPA [Environmental Protection Agency]. (2012). Chemical-specific reference values for benzene (CASRN 71-43-2). https://oaspub.epa.gov/eims/eimscomm.getfile?p_download_id=512648

U.S. EPA [Environmental Protection Agency]. (2016). *Benzene hazard summary* [PDF]. https://www.epa.gov/sites/default/files/2016-09/documents/benzene.pdf

U.S. EPA [Environmental Protection Agency]. (2024, May 28). *Volatile organic compound exemptions*. Retrieved April 13, 2025, from https://www.epa.gov/ground-level-ozone-pollution/volatile-organic-compound-exemptions

U.S. EPA [Environmental Protection Agency]. (2025, March 13).*Health effects of ozone pollution*. Retrieved July 2, 2025, from https://www.epa.gov/ground-level-ozone-pollution/health-effects-ozone-pollution

U.S. EPA [Environmental Protection Agency]. (2025b, March 27). *Health effects of ozone in the general population*. US EPA. https://www.epa.gov/ozone-pollution-and-your-patients-health/health-effects-ozone-general-population

U.S. EPA [Environmental Protection Agency]. (2025c, April 11). *Ozone Generators that are Sold as Air Cleaners*. US EPA. https://www.epa.gov/indoor-air-quality-iaq/ozone-generators-are-sold-air-cleaners

VA (US Department of Veterans Affairs). (2024, May 22). *New VA Health Eligibility Under the PACT Act*. Veterans Affairs. https://www.va.gov/clarksburg-health-care/programs/2024-pact-act-health-care-eligibility-what-changed/

VA (US Department of Veterans Affairs). (2025, April 21). *The PACT Act and your VA benefits*. Veterans Affairs. https://www.va.gov/resources/the-pact-act-and-your-va-benefits/

VA (US Department of Veterans Affairs), & Flynn, L. (2024, March 5). *All veterans exposed to* toxins *now eligible for VA health care* [Press-release]. VA Manchester Healthcare System. https://www.va.gov/files/2024-03/20240305%20Media%20Press%20Release_%20PACT%20Act%20Expansion%20Section%20103_PUBLISH.pdf#:~:text=At%20the%20direction%20of%20President%20Biden%2C%20VA%20is,earlier%20than%20called%20for%20by%20the%20PACT%20Act

VA (US Department of Veterans Affairs), & VHA (Veterans Health Administration). (n.d.). *Diseases Associated with Ionizing Radiation Exposure*. VA.gov | Veterans Affairs | Public Health. https://www.publichealth.va.gov/exposures/radiation/diseases.asp

Veterans Guide. (2025, March 24). *Asbestos on Submarines | Veterans Guide*. https://veteransguide.org/mesothelioma/navy/submarines/

Wright, L. (2025, March 12). *Asbestos on submarines*. MesotheliomaHope.com. https://www.mesotheliomahope.com/veterans/navy/ships/submarines/

Yang, Y., S. Changnian, and M. Sun. (1997). The effect of moderately increased CO_2 concentration on perception of coherent motion. *Aviation, Space, and Environmental Medicine. 68*(3):187-191.

SUBMARINERS' ADVOCACY GROUP

Submariners Helping Submariners®

The Unseen Burden:

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

AMENDMENT 1 – JANUARY 2026

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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)

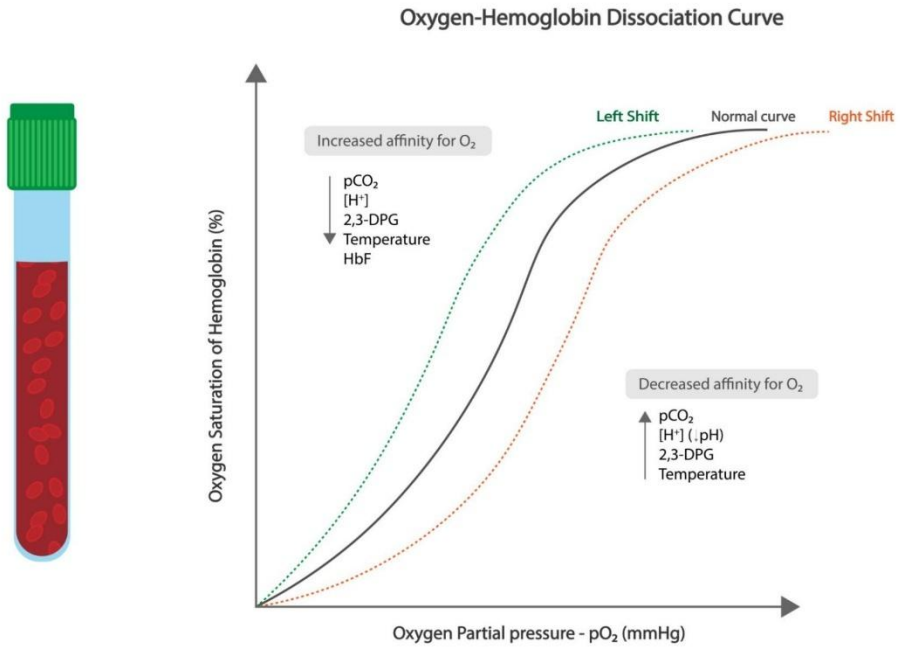
At standard pressure, nitrogen is physiologically inert. Its primary danger here is **displacement**. By occupying over 80% (Knight et al., 1990) of the available volume, it mechanically forces oxygen levels down. Unlike diving scenarios, there is no risk of nitrogen narcosis; the risk is purely the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



UKposters (n.d.)

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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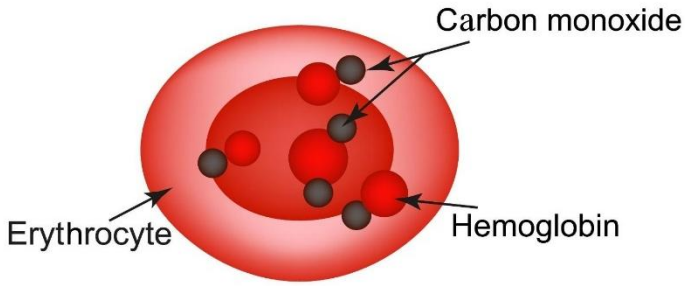
3. Analysis of Atmospheric Toxicants

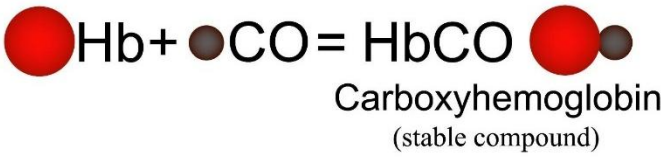
Carbon Monoxide (CO): The “Hypoxia Multiplier”

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).

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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References

Agency for Toxic Substances and Disease Registry. (2007). *Toxicological profile for benzene*. U.S. Department of Health and Human Services. https://www.ncbi.nlm.nih.gov/books/NBK591289/

Agency for Toxic Substances and Disease Registry. (2023). *Toxicological profile for benzene: Draft for public comment*. U.S. Department of Health and Human Services. https://www.epa.gov/sites/default/files/2014-03/documents/benzene_toxicological_profile_tp3_3v.pdf

Agency for Toxic Substances and Disease Registry. (2024). *Benzene: Toxicological profile* (Updated). U.S. Department of Health and Human Services. https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=40&tid=14

Alexander, W. J., McDougal, J. N., & Still, K. R. (2001). *Assessment of 2,6-di-tert-butyl-4-nitrophenol (DBNP) exposure in U.S. Navy submarines*. https://pubmed.ncbi.nlm.nih.gov/15910791/Cee, R. & Branch of Inorganic Methods Development. (1990). *CARBON DIOXIDE IN WORKPLACE ATMOSPHERES*. https://www.osha.gov/sites/default/files/methods/osha-id172.pdf

Chen, X., Zhang, Y., Li, J., & Wang, Q. (2025). Cardiac abnormalities induced by benzene exposure: Mechanisms and clinical implications. *Journal of Occupational and Environmental Cardiology, 12*(2), 101–115. https://pmc.ncbi.nlm.nih.gov/articles/PMC12273905/pdf/ijomeh-38-249.pdf

Dir. SSP (Director Strategic Systems Programs). (2013). *Implementation of confined space entry program for missile operations ashore and afloa*t (No. U031813011). Department of the Navy. Obtained via FOIA Request.

Guenette, J. A., & Koehle, M. S. (2012). Hypobaric vs. normobaric hypoxia: the lungs may be different. *Journal of Applied Physiology*, *112*(10), 1788–1794. https://doi.org/10.1152/japplphysiol.00356.2012

Hartley, W. R., & Smith, M. T. (2010). Advances in understanding benzene health effects and susceptibility. *Toxicology and Applied Pharmacology, 248*(1), 1–16. https://pmc.ncbi.nlm.nih.gov/articles/PMC4360999/

Hohenauer, E., Freitag, L., Costello, J. T., Williams, T. B., Küng, T., Taube, W., Herten, M., & Clijsen, R. (2022). The effects of normobaric and hypobaric hypoxia on cognitive performance and physiological responses: A crossover study. *PloS one*, *17*(11), e0277364. https://doi.org/10.1371/journal.pone.0277364

Knight, D. R., Tappan, D., V., Bowman, J. S., O'Neill, H. J., & Gordon, S. M. (1990). Submarine atmospheres. In Naval Submarine Medical Center, Groton, CT (United States). https://www.osti.gov/biblio/5196201

McMahon, K., & Launico, M. V. (2025, April 19). *Carbon monoxide toxicity*. StatPearls - NCBI Bookshelf. https://www.ncbi.nlm.nih.gov/books/NBK430740/

McHale, C. M., Zhang, L., & Smith, M. T. (2012). Current understanding of the mechanism of benzene-induced leukemia in humans: Implications for risk assessment. *Carcinogenesis, 33*(2), 240–252. https://pmc.ncbi.nlm.nih.gov/articles/PMC3271273/

Mikrostoker. (2016, January 25). Carboxyhemoglobin joining the hemoglobin carbon – Royalty-Free Vector | VectorStock. VectorStock. https://www.vectorstock.com/royalty-free-vector/carboxyhemoglobin-joining-the-hemoglobin-carbon-vector-7383118

Millet, G., Faiss, R., & Pialoux, V. (2012). Point: Counterpoint: Hypobaric hypoxia induces/does not induce different responses from normobaric hypoxia. *Journal of Applied Physiology, 112*(10), 1783–1784. https://doi.org/10.1152/japplphysiol.00067.2012

National Academies of Sciences, Engineering, and Medicine. (2008). *2,6-di-tert-butyl-4-nitrophenol*. In *Emergency and continuous exposure guidance levels for selected submarine contaminants* (Chapter 4). National Academies Press https://www.nationalacademies.org/read/12032/chapter/6

NRC (National Research Council). (2008). *Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 2*. Washington, DC: The National Academies Press. https://doi.org/10.17226/12032.

Rosales, A.M., Shute, R.J., Hailes, W.S. *et al.* Independent effects of acute normobaric hypoxia and hypobaric hypoxia on human physiology. *Sci Rep* 12, 19570 (2022). https://doi.org/10.1038/s41598-022-23698-5

Ross, D. (1996). The mechanism of benzene-induced leukemia: A hypothesis and speculations on the causes of leukemia. *Environmental Health Perspectives, 104*(Suppl 6), 1219–1225. https://pmc.ncbi.nlm.nih.gov/articles/PMC1469721/

Sabik, L. M., Abbas, R. A., Ismail, M. M., & El-Refaei, S. (2009). Cardiotoxicity of Freon among refrigeration services workers: comparative cross-sectional study. Environmental Health, 8(1), 31. https://doi.org/10.1186/1476-069x-8-31

SAG (Submariners' Advocacy Group). (2025). The Unseen Burden: Toxic exposures and health impacts on U.S. navy submariners. In *Submariners' Advocacy Group*. https://submarinersadvocacygroup.org/assets/files/The_Unseen_Burden.pdf

Still, K. R., Jederberg, W. W., Briggs, G. B., Jung, A. E., Prues, S. L., Ritchie, G. D., Godfrey, R. J., Naval Health Research Center Detachment (Toxicology), GeoCenters, Inc., & ManTech Environmental Technology, Inc. (2002). *ABSORPTION, DISTRIBUTION, AND CLEARANCE OF 2,6-Di-tert-Butyl-4-nitrophenol (DBNP)*. https://apps.dtic.mil/sti/tr/pdf/ADA411200.pdf

Still, K. R., Styer, C. M., Alexander, W. J., & McDougal, J. N. (2005). Disposition of 2,6-di-tert-butyl-4-nitrophenol (DBNP), a submarine contaminant, in rats. *Environmental Research*, 98(3), 276–285. https://pubmed.ncbi.nlm.nih.gov/15910791/U.S. Environmental Protection Agency. (2008). *Interim acute exposure guideline levels (AEGLs) for benzene*. https://www.epa.gov/sites/default/files/2014-08/documents/benzene_interim_dec_2008_v1.pdf

UKposters. (n.d.). https://www.ukposters.co.uk/oxygen-hemoglobin-dissociative-curve-or-oxygen-dissociation-curve-odc-f479225634

Vesselinovitch, S. D., Mihailovich, N., & Rao, K. V. N. (1961). Mammalian toxicity and histopathologic effects of 2,6-di-tert-butyl-4-nitrophenol. *Toxicology and Applied Pharmacology*, 3(4), 381–394. https://doi.org/10.1080/10473220117206

SUBMARINERS' ADVOCACY GROUP

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The Unseen Burden:

Compounded Stressors and the Physiology of "Human Error"

AMENDMENT 2 – AUGUST 2026

The Unseen Burden:

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

Amendment 2:

Compounded Stressors and the Physiology of “Human Error”

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August 4, 2026

1. Executive Summary

When evaluating operational mishaps (such as collisions, groundings, or tactical errors) or chronic post-service health conditions, military investigations into human error historically evaluated human performance in isolation. They assumed a healthy individual operating under standard environmental conditions.

As established in ***The Unseen Burden*** and ***Amendment 1***, the compromised closed-loop atmosphere of a submarine presents a "Cascading Failure" scenario driven by normobaric hypoxia, hypercapnia, and a continuous "toxic cocktail" of possibly 130 to 200 known contaminants. Peer-reviewed medical, toxicological, and neurophysiological literature demonstrates that this chronic exposure toxic profile, when combined with circadian desynchrony, creates a compounded physiological burden.

In occupational and military medicine, the interaction of these compounding stressors is evaluated through a cumulative risk model that measures the total biological **allostatic load**—the physiological wear and tear resulting from repeated, chronic adaptation to multi-stressor environments. When this cumulative risk exceeds human biological thresholds, it results in what the Submariners' Advocacy Group (SAG) defines as a **Cascading Physiological Failure**.

2. Glossary of Terms

Allostatic Load: The cumulative physiological wear and tear on the body resulting from repeated, chronic biological adaptation to a multi-stressor environment (McEwen, 1998).

Cascading Physiological Failure: A condition defined by the Submariners' Advocacy Group (SAG) where the cumulative risk of compounding atmospheric, chemical, and biological stressors exceeds human biological thresholds. This triggers a multi-systemic breakdown—simultaneously attacking the respiratory, cardiovascular, central nervous, digestive, and immunological systems—while concurrently driving the predictable degradation of executive function (complex decision-making and risk assessment), working memory, and sensory processing. *(Note: As a SAG-defined conceptual model, this relies on the combined literature rather than a single external citation)*.

Cellular Asphyxia: A physiological state where cells are prevented from converting available oxygen into usable energy (ATP), such as through a mitochondrial blockade caused by specific aerosolized toxicants like DBNP (NRC, 2008). Unlike standard asphyxiation, this occurs at the metabolic level; the body may have adequate oxygen in the lungs and blood, but the cells remain bioenergetically starved (non-hypoxic asphyxia).

Circadian Desynchrony: A severe disruption of the human body's natural 24-hour internal clock, caused by schedules (such as the historic 18-hour submarine day)

that require sleep during biological “peak wakefulness” and work during biological “sleep troughs” (Goel et al., 2009).

Hypercapnia: A condition characterized by excessive carbon dioxide in the bloodstream, often resulting from a high-CO_2 ambient atmosphere (Patel et al., 2022).

Hyperpnea: Deep, rapid breathing forced by the central nervous system in response to elevated ambient carbon dioxide levels, which inadvertently acts to accelerate the inhalation of other atmospheric toxicants (Patel et al., 2022).

Multi-Stressor Environment: An occupational or operational setting where multiple biological, chemical, and physical hazards act simultaneously on the human body, producing a compounded or synergistic physiological burden (NIOSH, 2016).

Sleep Architecture: The natural, cyclical pattern of sleep stages—including deep Slow-Wave Sleep and REM sleep—required for central nervous system recovery and cognitive maintenance (Shattuck & Matsangas, 2015).

3. Background: The Biological and Psychological Baseline

While ***The Unseen Burden*** and ***Amendment 1*** detailed the chemical and atmospheric hazards onboard, this amendment examines how those continuous exposures compounded and magnified an additional physiological crisis: Circadian Desynchrony. To understand this synergistic failure, it is critical first to establish the crew’s compromised biological baseline.

For over 50 years (prior to the 2014 transition to a 24-hour standard), Submariners operated on a strict 18-hour watch cycle (Shattuck & Matsangas, 2015). Human biology is governed by a master internal clock located in the brain’s hypothalamus (the suprachiasmatic nucleus), which dictates 24-hour circadian rhythms (Goel et al., 2009).

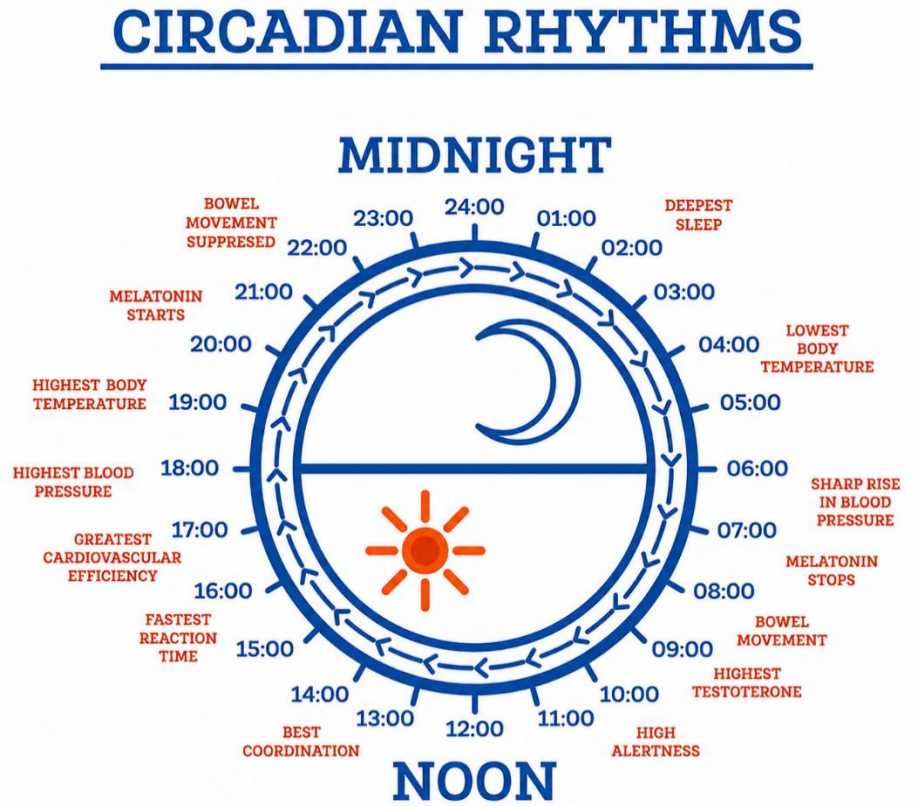

Figure 1: Circadian Rhythm. Scheme of Sleep Wake Cycle. Infographic Elements.

When Sailors were required to adopt an 18-hour day, they suffered from what sleep scientists call Circadian Desynchrony.

A. Biological Impacts

- **The 6-Hour Backward Shift:** Because the human body cannot natively adapt to an 18-hour day, sleeping 6 hours earlier every cycle meant Sailors were constantly trying to sleep during their biological “peak wakefulness” windows and were forced to stand watch during their biological “sleep troughs” (Shattuck & Matsangas, 2015).

- **Hormonal Chaos:** Melatonin (the sleep hormone) and cortisol (the stress/alertness hormone) may become completely desynchronized, resulting in metabolic disruptions, suppressed immune systems, and elevated cardiovascular stress (Goel et al., 2009; McEwen, 1998).

- **Sleep Architecture Failure:** During the historic 18-hour schedule, Sailors were required to sleep in rotating windows that defied natural circadian rhythms, resulting in severe sleep fragmentation and a documented failure to achieve restorative REM sleep (Goel et al., 2009; Shattuck & Matsangas, 2015).

- **Sustained Wakefulness:** Because their biological sleep was constantly interrupted, watchstanders frequently operated under conditions of chronic fatigue and sustained wakefulness. Foundational studies in occupational medicine demonstrate that this specific level of moderate sleep deprivation degrades cognitive and motor performance to an extent equivalent to a 0.05% Blood Alcohol Concentration (Dawson & Reid, 1997; Williamson & Feyer, 2000).

B. Psychological & Cognitive Impacts

- **Monotony and Micro-sleeps:** In the deep ocean, without daylight or dynamic stimuli, a sleep-deprived brain will experience involuntary "micro-sleeps"—complete lapses in cognitive responsiveness lasting 1 to 15 seconds (Poudel et al., 2014). During these brief periods, the brain's prefrontal and attention networks momentarily shut down (Goel et al., 2009). For a sonar tech, reactor operator, or helmsman, this poses a massive hidden operational risk.

- **Mood Decompensation:** Chronic circadian disruption is linked to decreased emotional resilience and compromised higher-order cognitive skills, such as executive function and decision-making—compounding the already heavy psychological toll of isolation and confinement (Krause et al., 2017).

4. The "Cascading Failure" Dynamics

The above baseline state of chronic exhaustion and hormonal chaos was not experienced in a vacuum. Submariners did not merely endure sleep deprivation in isolation; rather, they were subjected to a continuous, simultaneous exposure to both circadian disruption and the hazardous atmospheric conditions detailed in ***The Unseen Burden*** and ***Amendment 1***. Because this synergistic multi-stressor environment is continuous and chronic, the resulting physical and neurological depletion is not just additive—it may be exponentially magnified, driving multi-systemic degradation and profound allostatic overload.

The Mechanics of Allostatic Overload: In stress neurobiology, allostatic load represents the cumulative burden of continuous biological adaptation. On a submarine, the physiological systems governing sleep, oxygenation, metabolic pH, and cellular energy production are forced into perpetual overdrive.

- **Primary Allostatic Drivers:** Circadian desynchrony and continuous normobaric hypoxia.

- **Secondary Allostatic Multipliers:** Accelerated inhalation of atmospheric toxicants (Benzene, CO, DBNP, MEA) driven by hypercapnic hyperpnea.

- **The Cumulative Risk Outcome:** When these primary and secondary stressors act simultaneously, the crew's combined allostatic load reduces cognitive

reserve, potentially resulting in environmentally induced human errors, a predictable physiological outcome rather than a conscious failure of vigilance.

Amendment 1 extensively details how the submarine's base atmosphere forces a harmful biological trap. Elevated Carbon Dioxide (CO_2) acts as a potent respiratory stimulant, forcing hypercapnic hyperpnea (deep, rapid breathing), which accelerates the absorption of systemic toxicants like Carbon Monoxide (CO) and Monoethanolamine (MEA). Simultaneously, 2,6-di-tert-butyl-4-nitrophenol (DBNP) uncouples mitochondrial oxidative phosphorylation to cause cellular asphyxia. ***Amendment 2*** builds upon this foundation to demonstrate the "Synergistic Trap": how these atmospheric conditions interact with and magnify the impairment of a sleep-deprived brain.

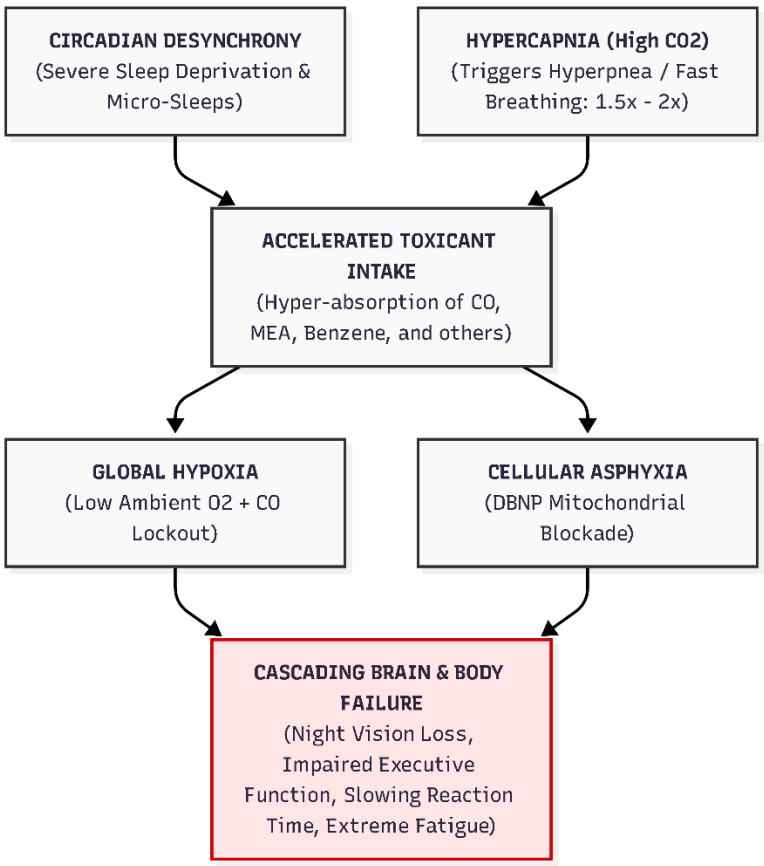

Figure 2: Synergistic Trap

A. The Cognitive Multiplier of Hypercapnia and Hypoxia

- **The Foundation:** As detailed in ***Amendment 1***, CO_2 maintained at 0.8% to 2.0% acts as a breathing accelerator (hyperpnea), while ambient oxygen (O_2) is intentionally maintained at 19% or lower (normobaric hypoxia).
- **The Compounded Reality:** From a cognitive standpoint, meta-analyses and systematic reviews confirm that acute and subchronic exposure to these

hypoxic conditions degrades reaction time, response accuracy, and short-term working memory (Ramírez-delaCruz et al., 2024). Hypoxia specifically attacks higher-order cognitive domains, accelerating impairment as oxygen saturation declines (Post et al., 2023). When forced onto an exhausted, micro-sleeping watchstander, this hypoxia may eliminate whatever cognitive reserves remain.

- **The Chronic Escalation:** It is critical to distinguish between the temporary fatigue of civilian shift-workers and the occupational reality of the submarine force. As established in ***The Unseen Burden*** (Auletta, 2002), toxicological exposures are defined by duration: acute (short-term), subchronic (repeated exposures up to 90 days), and chronic (continuous or repeated exposures lasting 90 days or more). While most civilian studies observe acute or subchronic fatigue, Submariners are routinely subjected to chronic exposure. Furthermore, because these continuous multi-stressor exposures are repeated over multiple patrols and across a career, the effects become cumulative. The biological damage compounds because the body is never given sufficient recovery time in a clean, oxygen-rich, and naturally lit environment to clear the biological and toxicological burden before the next exposure cycle begins.

B. Cellular Asphyxia Meets Neurological Exhaustion

- **The Foundation:** ***Amendment 1*** proves that aerosolized DBNP acts as a potent uncoupler, preventing brain and muscle cells from converting available oxygen into usable energy (ATP). Benzene exposure induces hematologic collapse, thus further reducing the blood's oxygen-carrying capacity. *(Note: Please refer to **The Unseen Burden** and **Amendment 1** for comprehensive toxicological data regarding DBNP, MEA, and Benzene)*.

- **The Compounded Reality:** Circadian desynchrony (mandated 18-hour watch loops) prevents access to restorative Slow-Wave and REM sleep, severely degrading the prefrontal cortex (PFC)—the region governing complex decision-making and risk assessment (Goel et al., 2009). Furthermore, the brain is simultaneously subjected to the physiological impacts of **chronic exposure to a normobaric, hypoxic atmosphere** (continuous ambient $O_2 \leq 19\%$), which independently reduces higher-order cognitive functions, impairs judgment, and slows reaction times. When DBNP-induced cellular starvation is combined with this chronic hypoxic baseline and profound PFC exhaustion, the brain simply lacks the bioenergetic capacity, the oxygen saturation, and the structural rest required to maintain vigilance.

- **Compromised Cellular Resilience:** Circadian desynchrony inherently disrupts cellular repair cycles and metabolic function. When this pre-existing biological exhaustion is combined with the atmospheric oxygen deprivation of normobaric hypoxia and the chemical interference of toxicant exposure, the

body's cells are stripped of their ability to either defend against or recover from the resulting asphyxiation.

C. Delayed Neurophysiological Threat Processing

- Recent neuroimaging and electrophysiological studies reveal that under hypoxic conditions, brain wave markers associated with pre-attentive sensory processing and novelty detection are significantly suppressed (Borden et al., 2024).
- Watchstanders experiencing this combined environment may suffer a measurable time lag between the onset of an environmental threat (e.g., depth change or sonar target contact) and conscious cognitive processing (Borden et al., 2024).
- **Synergistic Cognitive Degradation:** The neurological slowing caused by atmospheric and chemical hazards does not occur in a well-rested brain. It strikes a central nervous system already degraded by chronic circadian disruption. When the delayed threat processing of the atmospheric environment merges with the degraded executive function and working memory of the 18-hour watch cycle, the watchstander experiences a clinically significant and operationally critical decline in operational reaction time and complex decision-making.

5. Matrix of Multi-Stressor Interactions

This matrix illustrates how the biological stressors identified in ***Amendment 2*** interact with the atmospheric toxicants established in ***Amendment 1*** to produce predictable operational failures.

Primary Stressor (Biological)	**Secondary Co-Factor (Atmospheric)**	**Combined Physiological Outcome**	**Operational Impact on Watch**
Circadian Shift (18-Hr Loop) (Goel et al., 2009)	Normobaric Hypoxia (≤ 19% O_2) (Ramírez-delaCruz et al., 2024)	Compounded prefrontal cortex exhaustion; severe reduction in working memory span (Goel et al., 2009; Post et al., 2023).	Delayed target recognition, misinterpretation of bathymetric/tactical charts.

Primary Stressor (Biological)	Secondary Co-Factor (Atmospheric)	Combined Physiological Outcome	Operational Impact on Watch
Hypercapnia (High CO_2) (Patel et al., 2022)	Inhaled Vapors (Benzene / MEA) (SAG, 2026, Amendment 1)	Accelerated inhaled dose rate via hyperpnea; chemical erosion of pulmonary membranes (Patel et al., 2022).	Rapid onset of throbbing vascular headaches, pulmonary irritation, and systemic fatigue.
Cardiovascular Hyperarousal (Mansukhani et al., 2019)	Cellular Uncoupling (DBNP) + CO (SAG, 2026, Amendment 1)	Delivery blockade combined with cellular utilization blockade ("Cellular Asphyxia") acting on a highly stressed cardiovascular system.	Loss of fine motor control, rapid physical exhaustion during critical casualties.

Table A2-1: Multi-Stressor Matrix.

6. Conclusion

The medical and toxicological evidence demonstrates that Submariners do not operate in a vacuum of simple fatigue; they are subjected to a profound, inescapable biological trap. When the severe neurological degradation of circadian desynchrony collides with a chronic, normobaric hypoxic atmosphere and systemic toxicant exposure, a cascading physiological failure is the medically predictable result.

Furthermore, the National Research Council has historically pointed out the need to account for the synergistic interactions of atmospheric contaminants. The compounded effects of this hazardous atmospheric baseline, toxic mixture of multiple chemicals, weapons radiation, and severe circadian desynchrony have never been scientifically evaluated as a unified whole. Until formal, multi-stressor research is commissioned to definitively quantify this combined allostatic load, medical reviewers, VA adjudicators, and military investigators must operate on the probability that these synergistic environmental conditions could induce the possibility of operational failure and long-term physiological harm.

Operational mishaps, tactical errors, and chronic post-service health conditions can no longer be reflexively dismissed as isolated incidents of human error, individual watchstander negligence, the natural aging process, presumed family history, or unrelated medical anomalies. As established in Amendment 1, injuries from continuous toxic and atmospheric exposures often carry long latency periods. When this underlying toxicological damage interacts with the profound allostatic overload and cellular degradation driven by chronic circadian desynchrony, the resulting multi-systemic conditions may take years or decades to manifest fully. Consequently, adjudicators and medical reviewers must evaluate these late-onset conditions not as common civilian ailments, but as the delayed culmination of a multi-stressor environment, compounded by a complex occupational exposure profile.

References

Auletta, C. S. (2002). *Acute, subchronic, and chronic toxicology*. In M. J. Derelanko & M. A. Hollinger (Eds.), CRC Handbook of Toxicology (2nd ed., pp. 69–127). CRC Press. (Note: Your text cited 2002, while the bibliography listed 1995; 2002 is the standard edition for this specific data).

Borden, C. K., McHail, D. G., & Blacker, K. J. (2024). The time course of hypoxia effects using an aviation survival trainer. *Frontiers in Cognition*, 3, 1375919. https://doi.org/10.3389/fcogn.2024.1375919

Dawson, D., & Reid, K. (1997). Fatigue, alcohol and performance impairment. *Nature*, 388(6639), 235. https://doi.org/10.1038/40775

Goel, N., Rao, R., Durmer, J. S., & Dinges, D. F. (2009). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology*, 29(4), 320–339. https://doi.org/10.1055/s-0029-1237117

Krause, A. J., Simon, E. B., Mander, B. A., Greer, S. M., Saletin, J. M., & Walker, M. P. (2017). The sleep-deprived human brain. *Nature Reviews Neuroscience*, 18(7), 404–418. https://doi.org/10.1038/nrn.2017.55

Mansukhani, M. P., Covassin, N., & Somers, V. K. (2019). Apneic sleep, insufficient sleep, and hypertension. *Hypertension*, 73(4), 744–756. https://doi.org/10.1161/hypertensionaha.118.11780

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, 338(3), 171–179. https://doi.org/10.1056/NEJM199801153380307

NIOSH (National Institute for Occupational Safety and Health). (2016). *Mixed Exposures Research Agenda: A Report by the NORA Mixed Exposures Team*. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

NRC (National Research Council). (2008). *Emergency and continuous exposure guidance levels for selected submarine contaminants: Volume 2*. The National Academies Press. https://doi.org/10.17226/12032

Patel, S., Miao, J. H., Yetiskul, E., Anokhin, A., & Majmundar, S. H. (2022). *Physiology, Carbon dioxide retention*. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK482456/

Poudel, G. R., Innes, C. R., Bones, P. J., Watts, R., & Jones, R. D. (2014). Losing the struggle to stay awake: Divergent thalamic and cortical activity during microsleeps. *Human Brain Mapping*, 35(1), 257–269. https://doi.org/10.1002/hbm.22178

Ramírez-delaCruz, M., Bravo-Sánchez, A., Sánchez-Infante, J., Abián, P., & Abián-Vicén, J. (2024). Effects of acute hypoxic exposure in simulated altitude in healthy adults on cognitive performance: A systematic review and meta-analysis. *Biology*, 13(10), 835. https://doi.org/10.3390/biology13100835

Shattuck, N. L., & Matsangas, P. (2015). Psychomotor vigilance performance predicted by Epworth Sleepiness Scale scores in an operational setting with the United States Navy. *Journal of Sleep Research*, 24(2), 174–180. https://doi.org/10.1111/jsr.12243

Submariners' Advocacy Group. (2026). *The unseen burden: Toxic exposures and health impacts on U.S. Navy submariners* (Amendment 1).

Williamson, A. M., & Feyer, A. M. (2000). Moderate sleep deprivation produces impairments in cognitive and motor performance equivalent to legally prescribed levels of alcohol intoxication. *Occupational and Environmental Medicine*, 57(10), 649–655. https://doi.org/10.1136/oem.57.10.649