



FOREVER CHEMICALS IN THE BODY UNDERSTANDING PFAS, THEIR SILENT INVASION, AND THE FIGHT FOR OUR HEALTH

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Abstract

From the abyssal depths of the ocean to the isolation of deserted islands, from delicate deserts to dense rainforests and even within the human body—there exists a pervasive class of synthetic compounds known as “forever chemicals,” scientifically termed PFAS.

Within the hushed sanctuaries of our homes, beneath the gleam of nonstick cookware, the water-repellent luster of our garments, and even within the refined formulations of everyday cosmetics, an insidious and unseen intruder has quietly embedded itself. Per- and polyfluoroalkyl substances—known by the deceptively innocuous acronym

PFAS—have acquired a far more foreboding epithet in modern discourse: “forever chemicals.” These meticulously engineered compounds, born of the formidable bond between carbon and fluorine atoms, embody a troubling paradox



of scientific ingenuity. Ubiquitous, unyielding, and alarmingly persistent, they have insinuated themselves into ecosystems, consumer products, and even the human body, emerging as one of the most pervasive and enduring environmental hazards of the contemporary era.

Since their accidental discovery in 1938, PFAS have infiltrated nearly every aspect of contemporary life. From the grease-resistant wrappers of fast-food burgers to the firefighting foams that protect airports and military installations, these chemicals have been prized for their remarkable resistance to heat, water, and oil. Yet this very durability, the property that made PFAS indispensable to industry, has become the source of a mounting public health crisis. They do not degrade in the environment. They do not break down in the human body. They persist, accumulating silently in our blood, our organs, and even the umbilical cords that connect mothers to their unborn children. The statistics are staggering. According to the Centers for Disease Control and Prevention, approximately 97% of Americans carry detectable levels of PFAS in their bloodstream. In Europe, researchers have identified over 17,000 contamination hotspots, with millions of citizens drinking water that exceeds recommended safety limits. The industrial use of PFAS has been so extensive, so thoroughly woven into the fabric of modern manufacturing, that these chemicals have been detected in the remote reaches of the Arctic, in the depths of the ocean, and in the rain that falls from our skies. This article embarks on a comprehensive exploration of forever chemicals and their relationship with the human body. We will examine the molecular architecture that grants PFAS their persistence, trace the pathways through which they enter our lives, and investigate the mounting scientific evidence linking these substances to a spectrum of adverse health outcomes. From the molecular mechanisms of bioaccumulation to the latest regulatory responses and emerging strategies for risk reduction, this narrative seeks to illuminate one of the most significant environmental health challenges of our time.

Keywords: PFAS (Per- and Polyfluoroalkyl Substances), Forever Chemicals, Bioaccumulation, Environmental Persistence, Endocrine Disruption, Maximum Contaminant Level (MCL).



Introduction

What Are Forever Chemicals?

The story of forever chemicals begins in a laboratory in New Jersey, where a chemist named Roy Plunkett was attempting to develop a new refrigerant. When he opened a pressurized canister of tetrafluoroethylene gas and found it had polymerized into a waxy solid, he had unwittingly created polytetrafluoroethylene, the compound that would become famous as Teflon. This serendipitous discovery launched an entire industry of fluorinated chemicals, each designed to repel water, resist stains, and withstand extreme temperatures.

PFAS constitute a vast family of man-made chemicals, numbering between 12,000 and 15,000 distinct compounds. They share a common structural feature: chains of carbon atoms bonded to fluorine atoms. This carbon-fluorine bond is one of the strongest in organic chemistry, requiring immense energy to break. It is this molecular fortitude that gives PFAS their remarkable properties and, paradoxically, their environmental menace. While most organic compounds degrade through natural processes, the carbon-fluorine backbone of PFAS remains intact for decades, centuries, or potentially millennia.

The Chemistry of Persistence

To comprehend the extraordinary persistence of PFAS, it is essential to examine the intrinsic nature of the carbon–fluorine bond. In organic chemistry, bond strength is quantified by bond dissociation energy—the energy required to cleave a chemical linkage. The carbon–fluorine bond exhibits an exceptionally high dissociation energy of approximately 485 kilojoules per mole, in contrast to roughly 350 kilojoules per mole for a carbon–hydrogen bond. Although this disparity may appear marginal at the molecular level, it translates into a substantial and clinically relevant difference in chemical stability. As a consequence, conventional environmental degradation pathways—including microbial metabolism, photolytic breakdown via solar radiation, and hydrolytic processes in aqueous systems—lack sufficient energetic capacity to effectively disrupt these highly stable bonds.

Among the most extensively characterized PFAS are perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS). These perfluorinated eight-carbon chain compounds, commonly referred to as C8 substances, historically formed the backbone of the fluorochemical industry for several decades. PFOA functioned primarily as a processing aid in the industrial synthesis of polytetrafluoroethylene (PTFE), while PFOS constituted a key active component in 3M's Scotchgard textile and fabric protection systems. Although both compounds have been progressively phased out across the United States and numerous other jurisdictions, their replacement with shorter-chain PFAS—such as GenX compounds—has introduced new scientific and regulatory concerns. Emerging toxicological and environmental data suggest that, while these alternative compounds may exhibit reduced bioaccumulative potential on an individual basis, they nonetheless demonstrate environmental persistence and biological activity profiles that continue to warrant rigorous investigation and cautious appraisal.

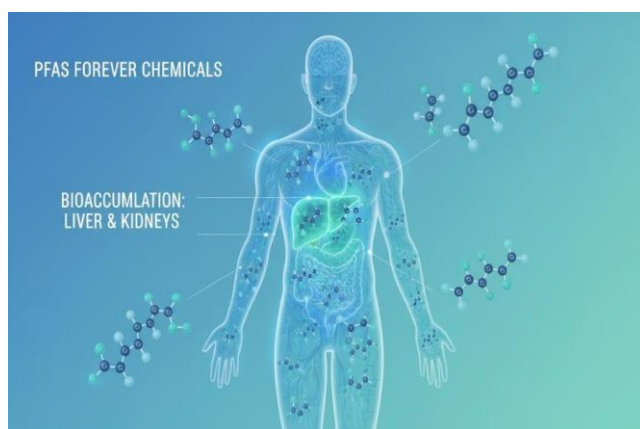


Figure 1: PFAS bioaccumulation in human organs, particularly the liver and kidneys

Routes of Human Exposure

The pathways through which PFAS enter the human body are as varied as the products that contain them. Understanding these routes of exposure is essential for both individual risk reduction and the development of effective public health interventions.

Research has identified drinking water, food consumption, inhalation, and dermal contact as the primary vectors of human exposure.



Drinking water represents the single most significant source of PFAS exposure for most populations. The chemicals enter water supplies through industrial discharge, leaching from landfills, runoff from firefighting training facilities, and the use of PFAScontaminated sewage sludge as agricultural fertilizer. Once in groundwater or surface water, PFAS travel with the flow, contaminating aquifers and reservoirs that serve as drinking water sources for millions of people. Studies have found that PFAS levels in drinking water can range from a few parts per trillion to concentrations exceeding regulatory limits by orders of magnitude in heavily contaminated areas.

Food represents another major exposure pathway. PFAS migrate into food from contaminated soil, water used in agriculture, food packaging materials, and processing equipment. Grease-resistant food wrappers, microwave popcorn bags, and compostable food containers frequently contain PFAS treatments that can leach into the food they contact. Fish and shellfish from contaminated waters can accumulate particularly high concentrations, as PFAS biomagnify through aquatic food webs.

Daily Life Sources

Transcending the domains of water and food, our everyday surroundings are replete with products imbued with PFAS. The sleek surfaces of nonstick cookware, when subjected to elevated temperatures, may liberate trace amounts of these compounds into both the air and the meals we prepare. Garments engineered for water resistance, along with stain-repellent upholstery and carpets, are often treated with PFAS-based finishes that enhance durability while quietly contributing to environmental persistence. Personal care items—ranging from select cosmetics to dental floss and shaving creams—may incorporate PFAS as emulsifying or film-forming agents, lending texture and longevity to their formulations. Even the seemingly innocuous dust that settles within our homes can serve as a reservoir for these substances, gradually accumulating as treated materials degrade and disperse microscopic particles over time.

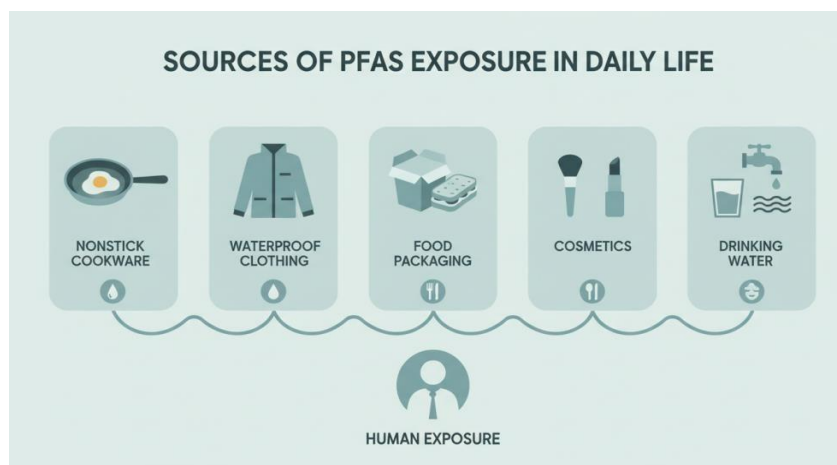


Figure 2: Common sources of PFAS exposure in daily life

PFAS Concentration Levels by Source

The following table presents typical PFAS concentration ranges across various environmental media, based on recent scientific literature and regulatory monitoring data:

Table 1: PFAS concentration levels across environmental sources

Environmental Source	Typical Range	Primary PFAS Detected
Drinking Water	0.1 - 100 ppt	PFOA, PFOS, GenX
Groundwater	1 - 1,000 ppt	PFOA, PFOS, PFHxS
Surface Water	0.1 - 50 ppt	PFOA, PFOS, PFNA
Fish Tissue	0.5 - 500 ng/g	PFOS, PFDA, PFUnA
Household Dust	10 - 1,000 ng/g	PFOA, PFOS, PFHxS
Human Blood Serum	1 - 50 ng/mL	PFOA, PFOS, PFHxS

Bioaccumulation in the Human Body

Once PFAS enter the human body, they embark on a complex journey through tissues and organs. Unlike many environmental contaminants that are metabolized and excreted within hours or days, PFAS resist the body's natural detoxification pathways.

The liver, which serves as the primary processing center for foreign chemicals, lacks the enzymatic machinery necessary to break the carbon-fluorine bond. As a result, PFAS circulate through the bloodstream for years, gradually accumulating in organs and tissues.

The half-life of PFAS in the human body, the time required for the concentration to decrease by half, ranges from approximately two to nine years depending on the specific compound. PFOA has a half-life of about 3.8 years in humans, while PFOS persists for roughly 5.4 years. This extended residence time means that even low-level, continuous exposure can lead to substantial body burdens over time. A person drinking water with PFAS at concentrations just above regulatory limits could accumulate concerning levels within years or decades.

Research has shown that PFAS exhibit a particular affinity for certain tissues. The liver and kidneys contain the highest concentrations, followed by blood serum, the thyroid gland, and testicular tissue. The placenta does not effectively block the transfer of PFAS from mother to fetus, meaning that prenatal exposure begins in the womb. Studies have detected PFAS in umbilical cord blood and breast milk, establishing that the first environment of the developing fetus is already contaminated with these persistent chemicals.

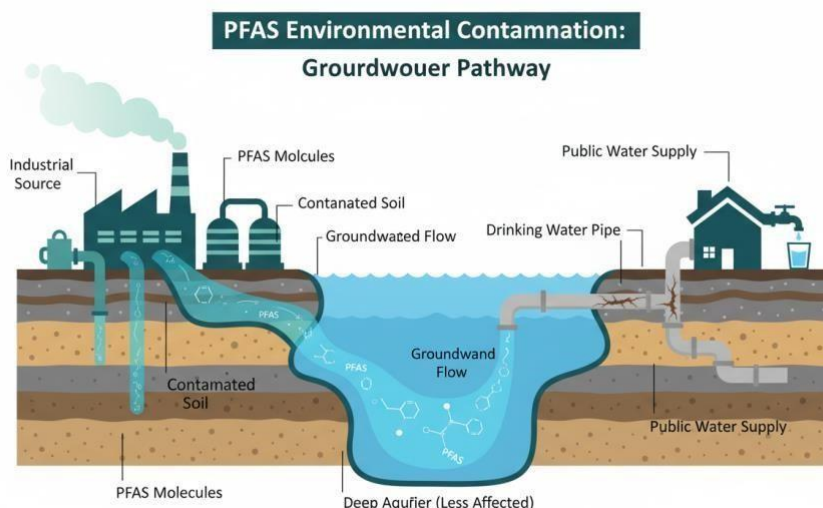


Figure 3: Environmental contamination pathways of PFAS into groundwater

Health Effects and Medical Evidence

The adverse health outcomes associated with PFAS exposure have been the focus of extensive and sustained scientific inquiry over the past two decades. Although



notable gaps in the literature persist—particularly with respect to the toxicological characterization of newer, short-chain PFAS variants—the cumulative body of evidence linking several well-established compounds to deleterious human health outcomes has become increasingly robust and methodologically sound. The most comprehensive epidemiological insights derive from the C8 Health Project, a largescale cohort study involving approximately 69,000 individuals in West Virginia and Ohio exposed to PFOA-contaminated drinking water, supplemented by decades of occupational health research among fluorochemical industry workers.

Population-based investigative studies have consistently delineated associations between PFAS exposure and a range of clinically significant physiological disturbances. Among the most consistently documented and biologically plausible findings is dyslipidemia, particularly elevations in serum cholesterol levels. Multiple peer-reviewed studies have demonstrated a positive correlation between circulating PFAS concentrations and increased total cholesterol as well as low-density lipoprotein (LDL) cholesterol, observed even at exposure levels commonly encountered in the general population. This lipid-altering effect raises important concerns regarding long-term atherogenic risk and the potential contribution of PFAS to cardiovascular morbidity at a population level.

The putative carcinogenic potential of PFAS has likewise prompted considerable regulatory and scientific scrutiny. In 2023, the International Agency for Research on Cancer (IARC) classified perfluorooctanoic acid (PFOA) as carcinogenic to humans, citing sufficient evidence for an association with renal cell carcinoma and limited evidence for testicular malignancies. Complementary analyses from the C8 Science Panel—constituted under a legal settlement framework—identified probable causal associations between PFOA exposure and multiple pathological conditions, including kidney and testicular cancers, ulcerative colitis, thyroid dysfunction, pregnancy-induced hypertensive disorders, and persistent hypercholesterolemia. Collectively, these findings underscore the evolving recognition of PFAS as a significant and multifaceted environmental health concern with substantial implications for preventive medicine and public health policy.



Immunological and developmental effects

One of the most concerning aspects of PFAS toxicity involves effects on the immune system. Studies have demonstrated that PFAS exposure is associated with reduced antibody responses to vaccines, potentially leaving individuals more vulnerable to infectious diseases. This immunosuppressive effect appears to be particularly pronounced in children, whose developing immune systems may be more susceptible to disruption. Research has also linked prenatal PFAS exposure to lower birth weights, though the clinical significance of these reductions remains a subject of ongoing investigation.

Endocrine disruption represents another major concern. PFAS have been shown to interfere with thyroid hormone function, which plays a critical role in metabolism, growth, and development. The structural similarity between certain PFAS and fatty acids may allow them to mimic or interfere with natural hormone signaling, potentially contributing to reproductive disorders, metabolic dysfunction, and developmental abnormalities.

Table 2: Summary of health effects associated with PFAS exposure

Health Effect	Evidence Strength	Key Findings
Kidney Cancer	Strong	IARC classified PFOA as carcinogenic; consistent evidence
Testicular Cancer	Moderate	Probable link established by C8 Science Panel
Thyroid Disease	Moderate	Associated with altered hormone levels
High Cholesterol	Strong	Consistent association across multiple studies
Immune Suppression	Moderate	Reduced vaccine antibody responses in children
Low Birth Weight	Moderate	Prenatal exposure linked to reduced fetal growth
Ulcerative Colitis	Moderate	Probable link in highly exposed populations

Regulatory Landscape

The regulatory response to PFAS contamination has evolved significantly over the past decade, though critics argue that action remains insufficient given the scale of the problem. In April 2024, the United States Environmental Protection



Agency took a landmark step by finalizing the first-ever national primary drinking water regulation for six PFAS compounds. This rule established legally enforceable Maximum Contaminant Levels of 4 parts per trillion for both PFOA and PFOS, with Maximum Contaminant Level Goals set at zero based on the latest science indicating that no level of exposure to these chemicals is without risk.

The EPA's regulatory action also addressed mixtures of PFAS through a Hazard Index approach, accounting for the combined health effects of co-occurring chemicals. For PFHxS, PFNA, and GenX chemicals, the agency set Maximum Contaminant Levels at 10 parts per trillion. Public water systems were given three years to complete initial monitoring and five years to implement treatment solutions if monitoring indicates exceedances. The agency estimates that this regulation will reduce PFAS exposure for approximately 100 million people, prevent thousands of deaths, and reduce tens of thousands of serious illnesses over time.

At the state level, numerous jurisdictions have enacted their own PFAS regulations, often setting more stringent standards than federal requirements. Michigan, New Jersey, and Vermont have established enforceable limits for multiple PFAS compounds in drinking water. Several states have banned PFAS in food packaging, firefighting foam, and consumer products. The European Union has proposed comprehensive restrictions on PFAS manufacturing and use, while individual member states have implemented their own monitoring and remediation programs.

Table 3: EPA Maximum Contaminant Levels for PFAS in drinking water (2024)

PFAS Compound	MCL (ppt)	Health Basis
PFOA	4.0	No safe level; carcinogenic to humans
PFOS	4.0	No safe level; carcinogenic to humans
PFHxS	10.0	Immunotoxicity and developmental effects
PFNA	10.0	Liver toxicity and developmental harm
GenX (HFPO-DA)	10.0	Liver and metabolic effects
PFAS Compound	MCL (ppt)	Health Basis
PFAS Mixtures	HI = 1.0	Cumulative risk assessment

Detoxification and Risk Reduction

Given the persistence of PFAS in the human body and the ubiquity of environmental contamination, individuals and communities are increasingly seeking strategies to reduce exposure and promote elimination of accumulated chemicals. While no medical treatment can completely remove PFAS from the body, research has identified several approaches that may accelerate excretion and reduce body burden.

Blood and plasma donation represents the most well-documented method for reducing PFAS levels. A landmark 52-week study conducted on Australian firefighters with occupational PFAS exposure found that regular blood or plasma donations significantly reduced serum concentrations of PFOS and PFOA. The mechanism appears straightforward: PFAS bind to proteins in blood, and removing blood effectively extracts these chemicals from circulation. As the body replaces donated blood, it draws PFAS from tissue reservoirs to maintain equilibrium, gradually depleting overall body stores.

Emerging research suggests that dietary interventions may also play a role. Recent pilot studies have shown promise for soluble fiber supplements, particularly psyllium and beta-glucan, in reducing PFAS levels. The mechanism involves the enterohepatic circulation of PFAS, these chemicals are excreted from the liver into bile, travel to the intestines, and are partially reabsorbed back into the bloodstream. Soluble fiber appears to trap PFAS in the gut, preventing reabsorption and promoting elimination through feces. One study documented an 8% decrease in PFOS and PFOA levels after just four weeks of fiber supplementation.

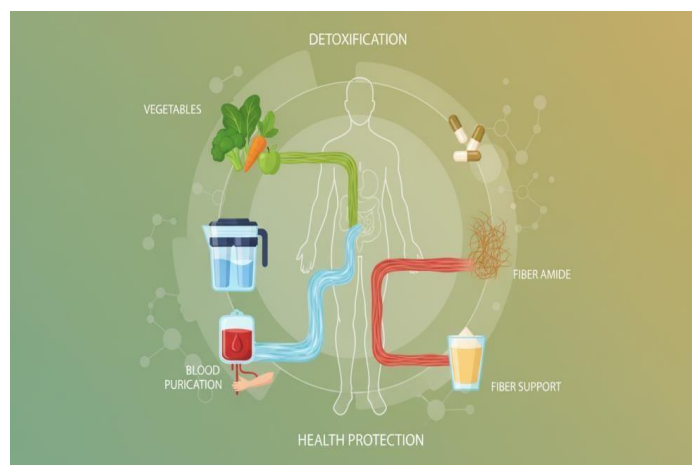


Figure 4: Strategies for PFAS detoxification and risk reduction



Practical Risk Reduction Measures

The health consequences associated with PFAS exposure, emerging over decades of rigorous scientific scrutiny, have been the focus of extensive investigation within the environmental and biomedical research community. Although important knowledge gaps persist—particularly concerning the toxicological profiles of emerging and shortchain PFAS variants—the accumulated evidence linking several well-characterized compounds to adverse human health outcomes has become increasingly compelling and methodologically robust. Among the most authoritative datasets are those derived from the C8 Health Project, a large-scale epidemiological investigation encompassing approximately 69,000 individuals in West Virginia and Ohio exposed to PFOA-contaminated drinking water, alongside decades of occupational cohort studies involving workers in the fluorochemical industry

A growing body of epidemiological literature has persistently delineated significant associations between PFAS exposure and a broad constellation of metabolic, endocrine, and immunological perturbations. One of the most reproducible and well-substantiated correlations is with dyslipidemia, particularly elevated serum cholesterol levels. Multiple peer-reviewed studies have reported positive associations between circulating PFAS concentrations and increased total cholesterol as well as low-density lipoprotein (LDL) cholesterol, observed even at exposure levels prevalent in the general population. This dysmetabolic profile raises clinically significant concerns regarding long-term cardiovascular morbidity, especially in the context of widespread environmental exposure.

The potential carcinogenicity of PFAS has also attracted substantial regulatory and scientific attention. In 2023, the International Agency for Research on Cancer (IARC) classified PFOA as carcinogenic to humans, citing sufficient evidence for kidney cancer and limited evidence for testicular cancer. Complementary findings from the C8 Science Panel—established under a legal settlement framework—identified probable causal associations between PFOA exposure and a range of pathologies, including renal and testicular malignancies, ulcerative colitis, thyroid dysfunction, pregnancy-induced hypertensive disorders, and persistent hypercholesterolemia. Collectively, these findings underscore the growing recognition of PFAS as a significant and multifaceted environmental health hazard.



Conclusion

The story of forever chemicals in the body is a story of unintended consequences, of scientific ingenuity that outpaced our understanding of long-term impacts. The same molecular properties that made PFAS remarkably useful, their resistance to heat, water, and degradation, have transformed them into a persistent threat to human health and environmental integrity. We find ourselves in a situation where these manufactured chemicals have become ubiquitous contaminants, detectable in the blood of nearly every person on Earth.

The evidence linking PFAS exposure to adverse health effects has reached a point where inaction is no longer scientifically defensible. The associations with kidney cancer, testicular cancer, thyroid disease, immune suppression, and developmental harm are supported by extensive peer-reviewed research. The EPA's decision to set Maximum Contaminant Level Goals at zero for PFOA and PFOS reflects a growing consensus that there is no safe level of exposure to these particular compounds.

Yet there are reasons for cautious optimism. Regulatory frameworks are evolving, with the EPA's national drinking water standards representing a watershed moment in environmental policy. Technologies for removing PFAS from water are proven and effective, if costly. Scientific research into detoxification strategies, from blood donation to fiber supplementation, is yielding actionable insights. The phased replacement of long-chain PFAS with shorter-chain alternatives, while not without its own concerns, demonstrates that industrial adaptation is possible.

The challenge ahead is immense. Cleaning up decades of environmental contamination will require sustained investment, technological innovation, and political will. The approximately 17,000 contamination hotspots identified in Europe represent only the known contamination; countless other sites likely remain undetected. The thousands of PFAS compounds in current use, many with limited toxicity data, present a continuing source of exposure even as legacy chemicals are phased out.

For individuals, awareness is the first line of defense. Understanding the sources of PFAS exposure enables informed choices about water filtration, food consumption, and product selection. For communities, advocating for comprehensive water testing, supporting regulatory enforcement, and demanding



corporate accountability can drive systemic change. For society as a whole, the forever chemicals crisis serves as a sobering reminder that the materials we create, no matter how useful in the moment, can impose lasting burdens on human health and the natural world.

The carbon–fluorine bond, meticulously constructed in laboratories decades ago, has revealed a stability and tenacity far surpassing initial scientific projections. Liberating ourselves from its entrenched legacy will necessitate an equal convergence of scientific ingenuity, regulatory fortitude, and collective, sustained action. Although these substances may be “forever” in their environmental persistence, our exposure to them remains neither inevitable nor immutable.

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