Cancer & Environment Network of Southwestern Pennsylvania

A Year in Review: Reflections on 2025 Peer-Reviewed Research on Cancer and Environment

Since 2021, the Cancer and Environment Network of Southwest Pennsylvania (CENSWPA) has produced an annual "Year in Review" highlighting peer-reviewed research on environmental exposures that may contribute to cancer risk. The purpose of this review is to synthesize emerging scientific findings and identify themes relevant to understanding potential cancer risks, as well as associated prevention needs and opportunities.

Based on peer-reviewed epidemiologic studies published in 2025 and identified through structured monthly PubMed searches, CENSWPA identified four notable topics:

1. Air Pollution and Cancer: A Pervasive Risk – Evidence increasingly links air pollution not only to lung cancer but to breast, bladder, and blood cancers, underscoring the systemic effects of inhaled contaminants and unequal patterns of exposure.
2. Endocrine-Disrupting Chemicals (EDCs): Multiple Mechanisms, Widespread Impact – Studies continue to demonstrate how hormone-disrupting chemicals—including bisphenols, phthalates, and other industrial or consumer product contaminants—may contribute to cancer risk through multiple mechanisms, including hormonal disruption, chronic inflammation, and interference with cellular signaling.
3. Microplastics: Tiny Particles, Growing Cancer Concerns – Research suggests that micro- and nanoplastics are now widely detected in human tissues, including tumors, and may promote cancer through mechanisms such as chronic inflammation, oxidative stress, and disruption of normal cellular signaling.
4. PFAS: Persistent Chemicals, Persistent Cancer Risks – These "forever chemicals" continue to demonstrate increased risk associated with multiple cancer types.

Air pollution, endocrine-disrupting chemicals, microplastics, and PFAS are pervasive in our environment. Exposures occur through the air we breathe, the water we drink, the food we eat, and the products we use. Because these exposures are widespread and most often involuntary, understanding their potential cancer implications is a public health priority. The following informational synthesis of selected peer-reviewed epidemiologic studies from the past year on the above four topics. is intended for informational purposes. The information should not be interpreted as individualized medical advice; as with most epidemiologic research, findings describe associations observed at the population level and should not be interpreted as establishing causation at the individual level.

CENSWPA is grounded in the public health principle that preventive action may be warranted when credible scientific evidence indicates potential harm—particularly when exposures are widespread—even as research continues to evolve. Continued study remains essential; however, so is the responsible application of current knowledge to reduce unnecessary and inequitable environmental cancer risks. Following each scientific synthesis, CENSWPA highlights prevention-oriented strategies and examples of current initiatives aimed at reducing environmental cancer risks in Southwestern Pennsylvania and beyond.

Air Pollution and Cancer: A Pervasive Risk

In 2025, CENSWPA reviewed more than 30 peer-reviewed studies examining the relationship between ambient air pollution and cancer. Together, this body of evidence suggests that long-term exposure to fine particulate matter ($PM_{2.5}$) and nitrogen dioxide (NO_2)—two common pollutants from traffic, industry, and fossil fuel combustion—is associated with increased cancer risk and poorer cancer outcomes across multiple organ systems.

Importantly, the literature shows that air pollution is not only linked to who gets cancer, but also how these pollutants contribute to more aggressive and deadly cancers, and how exposure impacts survival after diagnosis. These effects extend beyond the lung and include breast, digestive, prostate, pediatric, and central nervous system cancers. The burden is not evenly distributed: people living in lower-income communities, communities of color, and areas affected by industrial activity or wildfire smoke experience higher exposures and worse outcomes.

What is the research telling us?

Lung Cancer: Understanding Risk in Non-Smokers

The strongest and most mechanistically detailed evidence continues to center on lung cancer—particularly among people who have never smoked. A landmark genomic study analyzed lung tumors from never-smokers and found that exposure to high levels of air pollution leaves behind distinct "mutational fingerprints" in DNA (Díaz-Gayy, et al. 2025). These fingerprints resemble damage patterns previously associated with tobacco smoke, including mutations in the genes that suppress tumors and shortened telomeres (the latter of which is also associated with tumor suppression and cellular aging). Additional studies (Hutchings et al. 2025; Héluain et al. 2025) reinforced this finding and elevated how exposure to $PM_{2.5}$ triggers chronic inflammation in lung tissue and promote the growth of cells that already carry cancer-related mutations (such as changes in the EGFR gene –mutations in this gene cause lung cancers to grow and spread). Population-based studies report that long-term $PM_{2.5}$ exposure increases lung cancer incidence and mortality, particularly among individuals with higher inherited genetic risk (Zhu et al. 2025). Encouragingly, one study found that living in greener residential environments—often associated with lower air pollution—was linked to reduced lung cancer risk (Zhu et al. 2025).

Breast Cancer: Incidence, Survival, and Industrial Emissions

Several large U.S. studies published in 2025 identify breast cancer as a key site of pollution-related risk. Long-term exposure to nitrogen dioxide (NO_2), a marker of traffic and industrial combustion, was consistently associated with higher breast cancer incidence (White et al. 2025; Ish et al. 2025). A meta-analysis of 22 studies found similar associations (Wang R et al. 2025). $PM_{2.5}$ was linked to estrogen receptor–negative tumors, a cancer subtype that is often more aggressive and harder to treat (White et al. 2025).

Beyond general pollution levels, researchers also examined specific industrial emissions. Exposure to pollutants such as nickel and trichloroethylene—both associated with manufacturing and industrial processes—was linked to increased breast cancer risk (Ish et al. 2025), highlighting the importance of local emission sources.

Air pollution also affects breast cancer outcomes after diagnosis. Older breast cancer patients exposed to higher levels of $PM_{2.5}$, NO_2, and ozone experienced higher mortality rates (Wei et al. 2025), reinforcing the idea that pollution can influence not only cancer development but also survivorship. Not all regions showed the same patterns; a Canadian study found no association between NO_2 exposure and breast cancer (Ahmadi et al. 2025). This difference may be due in part to lower pollution levels, underscoring how local exposure levels matter.

Digestive and Liver Cancers: Growing Evidence Beyond the Lung

Evidence documenting pollution-related risk to digestive organs continues to strengthen. Two large meta-analyses reported that long-term exposure to $PM_{2.5}$ and NO_2 increases both the incidence and mortality of liver cancer, particularly hepatocellular carcinoma (Odat et al. 2025; Wu et al. 2025). Studies of colorectal cancer patients found that higher exposure to particulate matter and sulfur dioxide was associated with worse survival, even among those diagnosed at earlier stages (Wang CW et al. 2025). Additional research linked air pollution to cancers of the upper digestive tract, including the esophagus (Tang et al. 2025).

Other Cancer Sites: Prostate, Brain, Ovarian, and Head and Neck

A growing number of studies extend the cancer–air pollution link to additional organ systems. For prostate cancer, exposure to multiple pollutants—including $PM_{2.5}$, nitrogen oxides, and benzene—was associated with increased risk (Chen Y et al. 2025). Mechanistic studies identified specific genes involved in inflammation and blood vessel function that may help explain how pollution promotes prostate tumor growth (Li et al. 2025).

Research also suggests that fine and ultrafine particles increase the risk of certain brain tumors, such as meningioma (Hvidtfeldt et al. 2025). For ovarian cancer, multiple studies investigated links with air pollution and survival, but findings were mixed. For example, Chirikova et al. (2025) found limited evidence for reduced survival associated with air pollution (broadly characterized) while Zhang YY et al. (2025) found that higher particulate exposure reduced survival, with physical activity partially buffering these effects. Using genetic approaches, researchers also provided evidence that nitrogen dioxide exposure may play a causal role in head and neck cancers (Huang et al. 2025).

Pediatric Cancer: Early-Life Vulnerability

Children represent a uniquely vulnerable population. Reviews published in 2025 emphasize that pollution burden is associated with poorer clinical outcomes among pediatric cancer patients (Metayer 2025). Other studies link prenatal exposure to air pollution with increased risk of childhood leukemia (Yen et al. 2025), reinforcing concerns that exposure during critical developmental windows may have lifelong consequences.

Global Burden and Unequal Impacts

Globally, researchers estimate that hundreds of thousands of cancer deaths each year are attributable to ambient air pollution (Zhou J et al. 2025; Feng et al 2025). While household air pollution has declined

in many regions, cancer burden related to outdoor $PM_{2.5}$ continues to rise (Deng et al. 2025). Across studies, the impacts of air pollution were consistently more severe in low-income communities and among racial and ethnic minority populations (Ji et al. 2025; VoPham et al. 2025). Wildfire smoke emerged as a growing concern, with evidence showing reduced survival among cancer patients exposed to repeated smoke events (VoPham et al. 2025).

What can we do to reduce cancer risks from air pollution in Southwestern Pennsylvania?

Reducing cancer risks associated with air pollution requires coordinated action at multiple levels—particularly in regions like Southwestern Pennsylvania, where legacy industrial activity, dense transportation corridors, and energy infrastructure contribute to ongoing and cumulative exposures.

Historically, fine particulate matter ($PM_{2.5}$) has been the primary focus of air pollution–related cancer risk in the region. However, recent scientific literature—especially studies published in the past year—has elevated the importance of other pollutants, including nitrogen dioxide (NO_2) and hazardous air pollutants such as benzene. These pollutants are associated with increased cancer incidence and reduced survival and are emitted in significant quantities by industrial sources common in Southwestern Pennsylvania.

One example is the Shell Polymers Monaca Petrochemical Complex in Beaver County, which uses ethane, a byproduct of regional fracked gas production, to manufacture ethylene and polyethylene plastic pellets (commonly referred to as nurdles). Between January 2022 and November 2024, the facility released nearly 400 million pounds of air pollutants from malfunctions alone (Jones 2025). These releases included pollutants such as NO_2 and benzene—both linked in the literature above to breast and other cancers—as well as 1,3-butadiene and styrene, which are classified as known human carcinogens (Jones 2025; IARC 2026).

Emissions of this magnitude may contribute to cancer risks in surrounding communities. While most research focuses on outdoor air pollution, indoor air quality can also be severely affected, further increasing overall exposure and risk. In this context, local organizations play a critical role in translating scientific evidence into action. Members of CENSWPA support community education on air quality and health, advocate for stronger oversight and enforcement when industrial activities threaten public health, expand access to real-time air quality information in high-concern areas, and promote practical interventions to reduce indoor exposures. These efforts also include guidance on exposure reduction for residents, including individuals undergoing cancer treatment who may be especially vulnerable to air pollution.

At the municipal and county levels, stronger air monitoring and enforcement remain essential. Expanding neighborhood-scale monitoring near industrial facilities and major roadways can help identify pollution hotspots, inform targeted interventions, and increase accountability. Currently, several regional initiatives are enhancing transparency through real-time data collection and collaborative monitoring. For instance, the Beaver County Marcellus Awareness Community's (BCMAC) "Eyes on Air" platform provides rapid public disclosure of emissions from the Shell Polymers Monaca complex (BCMAC, 2026). Meanwhile, with U.S. EPA support, the Group Against Smog Pollution (GASP) in partnership numerous additional community-based and academic partners in the region are working with residents living near industrial facilities to collect data on air toxics and emissions while the FracTracker Alliance has

expanded its monitoring capacities related to emissions from petrochemical and unconventional gas industries (GASP 2026; FracTracker Alliance 2026). Carnegie Mellon University's CREATE Lab further bolsters these technical efforts as a key research partner in many of these monitoring initiatives, and through the creation of high-resolution tools like "Breathe Cam" to expose pollution events as they occur (BreatheCam, 2026). Additionally, the Environmental Health Project's "Compounds of Concern" tool connects specific industrial pollutants to documented health symptoms and impacts (Environmental Health Project, 2026). Together, these local organizational scientific networks provide the evidentiary framework necessary to strengthen public health protections and refine regulatory permitting processes.

Government agencies are crucial to provide programmatic direction and industry accountability in the protection of health and environment. Local governments can reduce long-term risk by integrating health considerations into land-use planning, zoning, and transportation decisions—particularly in communities that already experience disproportionate pollution burdens. State policy remains a powerful lever for change. Pennsylvania can draw on models from other states by strengthening emission limits, accelerating transitions away from high-polluting energy and manufacturing sources, and better incorporating cumulative impacts into permitting and enforcement decisions. Centering environmental justice—by prioritizing protections for low-income communities and communities of color that bear higher cumulative exposures—should be a core objective of these policies.

Finally, market-based and institutional actions can reinforce regulatory efforts. Health systems, universities, and major employers across Southwestern Pennsylvania can reduce their own emissions, invest in clean energy and transportation, and use their purchasing power to support cleaner industrial practices. Together, these strategies offer practical and complementary pathways to reduce air pollution exposure and improve cancer outcomes across the region.

Endocrine Disrupting Chemicals: Multiple Mechanisms, Multiple Cancers

Endocrine disrupting chemicals (EDCs) are substances that interfere with the body's hormone systems and are widely present in consumer products. By altering hormonal signaling that regulates cell growth, metabolism, and development, EDCs can increase vulnerability to cancer, particularly hormone-sensitive cancers such as breast, ovarian, and prostate cancer. Growing evidence links every day, low-level exposures to EDCs with multi mechanisms involved in cancer development and survivorship, including cancer initiation, progression and treatment resistance. In 2025, the literature CENSWPA reviewed explored a broad range of EDCs in relation to cancers including breast, ovarian and prostate.

What is the research telling us?

Breast Cancer

Phthalates are considered a ubiquitous endocrine disrupting chemical that is used as a softener in flexible plastics (PVC/vinyl), fragrances, and cosmetics, and are present in items like shower curtains, flooring, toys, food packaging, medical tubing, personal care products, perfumes, and even some medications. There are dozens of phthalates in this class, such as DEHP, DBP, BBP, and DINP[1]. Several studies published in 2025 both review and extend upon this body of evidence demonstrating impacts on breast cancer. Tiburcio et al. (2025) describe how research has demonstrated that the class of phthalates can act across multiple steps of the carcinogenic process. For example:

- Phthalates can stimulate processes in the body that encourage cells to grow and multiply, creating conditions that make it easier for cancer to start and progress (Chen FB et al. 2016; Chen et al. 2023).
- They have also been shown to help cancer cells avoid normal self-destruct signals and to increase the activity of genes that drive tumor growth (Chen FB et al. 2016; Chen FB et al. 2023). Because phthalates can mimic estrogen, they may interfere with normal hormone signaling in estrogen-sensitive breast cancers, further increasing tumor growth (Singh and Li, 2012; Ahern et al. 2019).
- In addition, phthalates appear to disrupt normal metabolism and promote cancer stem cells, which are linked to more aggressive disease (Yaghjyan et al. 2015; Karra et al. 2022; Cao et al. 2023).
- Perhaps most concerning, phthalates may help breast cancer spread to other parts of the body—the most dangerous stage of cancer—by increasing blood vessel growth around tumors and enabling cancer cells to invade distant organs (Buteau-Lozano et al. 2008; Hsieh et al. 2012; Tsai et al. 2014).
- Studies also suggest that phthalates may make some breast cancers harder to treat by reducing the effectiveness of chemotherapy drugs (Hsieh et al. 2022).

Through their review of both experimental and epidemiologic evidence, Tiburcio et al. (2025) raise concerns that long-term exposure to phthalates could contribute not only to breast cancer risk, but also to more aggressive and treatment-resistant disease.

[1]DEHP – Di(2-ethylhexyl) phthalate; DBP – Dibutyl phthalate BBP – Benzyl butyl phthalate; DINP – Diisononyl phthalate

Xiong and colleagues (2025) found compelling evidence linking triclosan with increased breast cancer risk. Despite being banned by the Federal Food and Drug Administration (FDA) in over the counter "anti-bacterial" hand/body soaps in 2017, triclosan – often used as an antimicrobial preservative – is still found in personal care products (toothpaste, cosmetics, deodorants) and in plastics, fabrics and toys. These researchers analyzed data from more than 4,400 participants in the National Health and Nutrition Examination Survey (NHANES) collected between 2005 and 2014 which measured levels of several EDCs in urine. Among the chemicals studied, triclosan stood out. Individuals with higher triclosan levels in their urine were significantly more likely to report having breast cancer compared to those with lower levels. The increased risk was observed at moderate and higher exposure levels, suggesting that even common exposures may be relevant. Risk rose with increasing exposure up to a point and then leveled off, indicating a complex dose–response pattern. The association was especially strong among people who were overweight, under the age of 60, and white, suggesting that certain groups may be more vulnerable to triclosan's effects.

Some metals and metalloids – called metalloestrogens – can mimic estrogen in the body and may contribute to breast cancer risk. Many of these metalloestrogens, such as arsenic, cadmium and nickel are known carcinogens associated with increased risk of several cancer types. Saint-Martin et al. (2025) used a long-term French cohort study that followed more than 66,000 women for over 18 years to examine whether dietary exposure to these metals was associated with estrogen receptor–positive breast cancer. Dietary exposure to metalloestrogens comes primarily from foods and beverages that contain trace metals or metalloids, either naturally or due to environmental contamination. One dietary exposure pattern—dominated by inorganic arsenic and vanadium—was linked to a higher risk of estrogen receptor–positive breast cancer. No significant associations were found when individual metals were analyzed separately, suggesting that combined, low-level exposures may be more important than single substances. Overall, the findings indicate that dietary mixtures of estrogen-active metals, even at relatively low levels, may increase breast cancer risk, highlighting the importance of addressing cumulative chemical exposures.

Koval et al. (2025) examined the association between chemicals found in house dust and breast cancer risk, specifically hormone-receptor-positive breast cancers. The study found increased risk of breast cancer associated with chemicals used frequently in personal care products. For example, risk was elevated in association with levels of:

- triethanolamine – a common ingredient in shampoos, lotions, creams, and cosmetics, used as a surfactant and pH adjuster;
- *nonylphenol ethoxylates* – a surfactant used in some personal care and cleaning products and known endocrine disruptors; and
- thiabendazole – a fungicide and preservative that can appear in treated products or household environments.

Ovarian Cancer

The ovaries are another hormonally sensitive organ and a focus of research regarding EDCs and ovarian cancer. Recent evidence suggests that bisphenols, including bisphenol A (BPA) and its substitute bisphenol S (BPS), may contribute to ovarian cancer development and aggressiveness. Common exposure sources included canned foods, plastics, thermal paper (often used for store purchase

receipts), and household dust, highlighting everyday pathways for BPA exposure. Alsaeed et al. (2025) conducted a case–control study finding that women with ovarian cancer had significantly higher urinary BPA levels than controls, along with increased oxidative stress and altered gene expression linked to tumor development. Mechanistic studies further show that low, environmentally relevant doses of BPA and BPS can promote ovarian cancer cell migration, invasion, and metastasis (Xu et al. 2025). These effects occur through activation of cancer-related signaling pathways and changes to the tumor microenvironment that support cancer growth. Notably, similar effects were observed for BPA and BPS, raising concerns about replacing BPA with closely related alternatives.

Prostate Cancer

Studies in 2025 expand on the body of knowledge that EDCs may be responsible for promoting cancer progression. BPA exposure was shown to reprogram mitochondrial energy metabolism in cancer cells (meaning that BPA is changing how cells "make fuel," helping them grow faster and survive longer than they normally would), activating key enzymes and estrogen receptor signaling that support tumor growth (Zhou X et al. 2025). Analysis of human prostate tissue and urine confirms that BPA and phthalates accumulate in the prostate, providing a direct link between environmental exposure and cancer progression (Zhang T et al. 2025).

What can we do to reduce potential cancer risks associated with endocrine disrupting chemicals?

Reducing potential cancer risks from endocrine-disrupting chemicals (EDCs) requires coordinated action across policy, advocacy, education, and market pathways.

Local community-based organizations play a critical role in advancing these efforts. For example, in Southwestern Pennsylvania, CENSWPA member Women for a Healthy Environment (WHE) engages in community education and advocacy that directly intersects with EDC exposure reduction. WHE's programs—such as Healthy Homes, Healthy Schools, and Healthy Early Learning Centers—provide residents with information on environmental risks in everyday products, including potential sources of endocrine disrupting chemicals. Consumer education—through awareness campaigns and informed purchasing—creates pressure for companies to adopt substitutes to hormone-disrupting chemicals.

At the state level, Pennsylvania policymakers have the authority to strengthen protections by drawing on instructive models from other states. States, including California, Minnesota, and Washington, have already restricted bisphenols, phthalates, and flame retardants in a range of consumer products. Importantly, California and Washington State have mandated the use of alternatives assessment to avoid replacing one toxic chemical for another in industrial processes containing these toxic chemicals. Market-based strategies further complement these policy efforts. Corporate reformulation, supply chain engagement, and integration of chemical safety into environmental, social, and governance (ESG) criteria encourage the adoption of safer chemistries.

Together, these actions—anchored in local NGO/consumer-oriented initiatives, guided by state policy models, and reinforced through market strategies—offer multiple pathways to reduce EDC exposures in Southwestern Pennsylvania.

Microplastics: Tiny Particles, Growing Cancer Concerns

In CENSWPA's 2024 Year in Review, we first highlighted emerging literature linking microplastics with cancer. Microplastics—plastic particles typically defined as less than 5 millimeters in size—are now recognized as ubiquitous environmental contaminants, detected in air, water, food, and human tissues. As evidence of widespread human exposure has grown, so too has concern about their potential role in adverse health outcomes, including cancer. Collectively, this body of literature positions exposure to microplastics as a credible yet incompletely characterized environmental cancer risk factor.

What is the research telling us?

Although this remains a relatively new area of research, several review articles published in 2025 synthesized a rapidly expanding body of evidence on microplastics and cancer. Across studies, microplastics have been detected within a wide range of tumor tissues and, in some cases, at higher concentrations than in adjacent noncancerous tissue (Mishra et al., 2025; Bogani et al., 2025). The review studies are largely focused on the biological mechanisms by which microplastics can contribute to the development of cancer. Experimental and patient-derived evidence suggests that micro- and nanoplastics may contribute to carcinogenesis through converging mechanisms, including oxidative stress, chronic inflammation, DNA damage, metabolic disruption, and interference with processes that regulate cell growth, cell death, and immune response (Bogani et al., 2025; Gałęziewska et al., 2025; Mishra et al., 2025). Several reviews also emphasize that microplastics can act as carriers for other hazardous substances—such as endocrine-disrupting chemicals, heavy metals, and persistent organic pollutants—potentially amplifying their toxic effects (Cirillo et al., 2025; Bogani et al., 2025).

Colon and Other Gastric Cancers

With rising rates of colorectal cancer, particularly among younger adults, there is growing interest in the potential role of additional risk factors not previously considered, including microplastics. Among the studies reviewed by CENSWPA, microplastics exposure as a potential risk factor for colon and other gastric cancers is especially prominent. Microplastics have been detected directly in human colorectal and stomach tissues, often at higher levels than in nearby healthy tissue, indicating that these particles can accumulate in the digestive tract over time (Pan et al., 2025; Chen L et al., 2025).

Laboratory and animal studies suggest that once present in the gut, microplastics can irritate tissues, disrupt the balance of healthy gut bacteria, and promote chronic inflammation and oxidative stress—conditions known to support cancer development (Wen & Lin, 2025). In colorectal cancer specifically, exposure to microplastics has been shown to promote tumor growth and reduce the effectiveness of chemotherapy, potentially by enabling cancer cells to activate protective survival mechanisms (Pan et al., 2025). Even long-term exposure to low doses of very small plastic particles (nanoplastics) has been associated with changes that increase cancer cell movement and spread, a key feature of metastasis (Kim et al., 2026). In stomach cancer, higher microplastic burdens have been linked to more aggressive disease, including lymph node metastasis and gene expression patterns associated with poorer survival (Chen L et al., 2025). While these findings do not yet establish causation in humans, together they raise concern that ongoing exposure to microplastics may worsen cancer risk and outcomes in the gastrointestinal tract.

Lung Cancer

The literature reviewed by CENSWPA also points to potential links between microplastics and lung cancer development, progression, and treatment response. Studies indicate that inhaled plastic particles can persist in lung tissue, where they may irritate cells, increase oxidative stress, and damage DNA—biological changes associated with cancer initiation and tumor growth (Sychowski et al., 2025). Microplastics may further intensify harm by carrying heavy metals and other contaminants into lung tissue (Sychowski et al., 2025).

Experimental studies suggest that microplastic exposure can promote lung cancer cell growth and reduce the effectiveness of radiation therapy by helping cancer cells survive treatment-related stress (Zhou H et al., 2025). Evidence from human lung tumor samples further indicates that higher levels of microplastics—particularly those bound to metals—are associated with more aggressive disease and poorer survival outcomes (Liu R et al., 2025). Although research is still emerging and definitive cause-and-effect relationships have not been established, these findings suggest that microplastics may represent an underrecognized factor influencing lung cancer progression and treatment outcomes.

What can we do to reduce potential cancer risks associated with microplastic pollution?

Evidence from human tissues, laboratory studies, and environmental monitoring increasingly suggests that microplastics may contribute to cancer. In Southwestern Pennsylvania, facilities such as the Shell Polymers Monaca Petrochemical Complex in Beaver County produce plastic pellets, or “nurdles,” which are a significant source of microplastic pollution. These pellets can escape into the environment during production, transport, or handling, eventually breaking down into micro- and nanoplastics. Once incorporated into consumer products, they may also contribute to human exposure through food, air, and water. Combined with toxic chemical emissions from fracking operations and associated industrial activity, these releases exacerbate regional environmental contamination and associated cancer risks. Reducing these risks requires coordinated action across multiple levels, even in the context of limited federal oversight and ongoing deregulatory pressures that allow industries to pollute despite clear impacts on public health and the environment.

At the state level, Pennsylvania policymakers already have authority under laws such as the Clean Streams Law and the Solid Waste Management Act—demonstrated in enforcement actions such as the 2025 Styropek settlement—to hold polluters accountable for releases of plastic pellets and other contaminants. Other states are taking additional steps: California is considering listing microplastics as priority chemicals for regulation in consumer products and implementing a statewide microplastics strategy to reduce releases at their source, while Rhode Island is exploring bans on intentionally added microplastics. Pennsylvania could look to these examples to develop policies prioritizing source reduction, monitoring, and industrial accountability. Industries can complement these efforts by adopting best practices for containment of nurdles and other plastics, investing in emissions-reducing technologies, and publicly reporting environmental performance.

Communities and individuals also play a critical role. Other cities and towns in Southwest Pennsylvania can follow Pittsburgh’s lead and issue ordinances banning the use of single use plastic bags (City of Pittsburgh). Local not-for-profit organizations are also leading the way. For example, PennEnvironment and Three Rivers Waterkeeper conduct monthly “nurdle patrols” of waterways and have pursued citizen

enforcement actions under the Clean Water Act to halt illegal discharges. PennFuture combines legal advocacy and grassroots campaigns to challenge excessive industrial emissions and push for stronger oversight of petrochemical facilities. The Plastics Collaborative under Humane Action Pennsylvania is reducing single-use plastics and educating communities about actions that limit microplastic pollution. Building on these efforts through enhanced community science, expanded reporting, and sustained engagement with policymakers can increase accountability and support policies that protect public health. Individuals can further reduce exposure by choosing microbead-free personal care products, synthetic-fiber alternatives (e.g., choose cotton fabrics over polyester or nylon), and minimally packaged foods, and by participating in local cleanup initiatives.

Emerging global efforts, such as negotiations on a United Nations Plastics Treaty, reinforce these strategies by emphasizing source reduction, stricter controls on microplastic emissions, and safer product design to limit both environmental contamination and toxic exposures. Aligning local and regional action with these international priorities provides a clear path forward: by reducing microplastic and chemical releases now, communities in Southwestern Pennsylvania can protect public health and the environment while research continues to clarify the full scope of risks.

PFAS: Persistent Chemicals, Persistent Cancer Risks

This is the third year that CENSWPA has covered the topic of PFAS in its Year in Review briefs. PFAS (per- and polyfluoroalkyl substances) are a large class (i.e., over 9,000 substances) of chemicals—often called "forever chemicals" because of their extreme persistence in the environment and the human body. PFAS have been widely used for decades due to their water-, stain-, and grease-resistant properties, appearing in products ranging from nonstick cookware, food packaging, and stain-resistant carpets to textiles, upholstered furniture, firefighting foams, and certain personal care products. They are also found in drinking water, soil, and indoor dust near industrial sites, airports, and military training facilities. Because PFAS are highly stable, they accumulate over time, creating long-term exposure risks for people and wildlife alike.

What is the research telling us?

In 2025, the scientific literature on per- and polyfluoroalkyl substances (PFAS) grew substantially, expanding our understanding of how these long-lasting chemicals shape cancer risk and outcomes across multiple organ systems. While earlier research focused heavily on kidney and testicular cancers, the newest studies broaden that scope to include thyroid, ovarian, and gastrointestinal cancers. These findings also raise critical new concerns about how PFAS may influence cancer progression, treatment response, and long-term survivorship. A central message emerging from this body of work is that PFAS are not only linked to who develops certain cancers, but may also shape how cancers behave, including whether they respond to therapy, often hindering the effectiveness of life-saving treatments.

Chemotherapy Resistance: An Emerging Threat

One of the most striking themes in 2025 is the evidence that PFAS exposures may actively undermine cancer treatments. Two independent studies document how PFAS can promote resistance to chemotherapy—an effect with serious implications for patients living in contaminated communities.

Among patients with colorectal cancer, exposure to PFOA and PFOS induced resistance to commonly used chemotherapy agents, making tumors harder to treat in individuals with higher environmental burdens (Liu J et al. 2025). In addition, for patients with ovarian cancer, chronic PFAS exposure was shown to disrupt mitochondrial function—the energy-producing machinery of cells—ultimately shielding tumor cells from the intended effects of chemotherapy (Rickard et al. 2025).

Thyroid Cancer and Hormonal Disruption

Thyroid cancer remains a major concern associated with PFAS exposure as a known effect of these substances is alterations to the thyroid gland. However, the 2025 picture is proving to be more complex. Multiple studies confirmed that PFAS exposures disrupt circulating thyroid hormone levels (Jung et al. 2025; Wang R et al. 2025a). However, this hormonal disruption did not consistently translate into higher thyroid cancer incidence. In fact, some PFAS compounds showed inverse associations with thyroid cancer in specific study populations. Systematic reviews further reported no clear positive relationship between PFAS and thyroid cancer risk (Sassano et al. 2025). These mixed findings highlight the importance of better understanding individual PFAS compounds, exposure timing,

Gynecologic and Reproductive Cancers

New studies in 2025 provide robust evidence linking PFAS to cancers of the reproductive system. High serum PFAS concentrations were associated with increased risk of ovarian cancer (Jones RR et al. 2025; Yang et al. 2025). Additional mechanistic research revealed how PFOA may interfere with the body's immune response in ways that allow ovarian cancer to grow or behave more aggressively, thus worsening patient outcomes (Li J et al. 2025). Emerging evidence also connects PFAS to endometrial cancer: pre-diagnostic serum levels were associated with elevated risk (Madrigal et al. 2025). Findings for breast cancer were more nuanced. The Cancer Prevention Study II cohort – a very large ongoing research study run by the American Cancer Society that has enrolled over 1.2 million adults across the U.S. and Puerto Rico – reported no overall association between PFAS exposure and breast cancer but detected important differences by tumor subtype. These findings suggest that PFAS may influence cancer development differently depending on underlying biology (Shahi et al. 2025).

PFAS also continues to raise concern for male reproductive cancers. A study examining early-life exposures found that PFAS exposure during fetal development increased the risk of testicular germ cell cancer later in adulthood, reinforcing the long latency nature of PFAS-associated risks (Uldbjerg et al. 2025).

Gastrointestinal Cancers

Systematic reviews published in 2025 reported mixed or null associations between PFAS and the incidence of gastrointestinal cancers, including colorectal and gastric cancers (Paudel et al. 2025; Zhang S et al. 2025). However, when considered alongside new evidence described above showing that PFAS can impair chemotherapy effectiveness in colorectal cancer (Liu J et al. 2025), the emerging concern may be less about PFAS as a risk factor for disease onset of GI cancers and rather more about exposure risks associated with disease progression and altering treatment outcomes.

Developmental Origins of Adult and Childhood Cancer

A consistent theme across the 2025 literature is the heightened vulnerability associated with early-life exposure. Several studies continue to strengthen the "window of susceptibility" framework, showing that prenatal PFAS exposure—measured through maternal serum levels or residential dust—was associated with increased risk of childhood acute lymphoblastic leukemia (Morimoto et al. 2025; Metayer et al. 2025). These findings reinforce growing concerns that PFAS exposures during fetal and infant development may set the stage for cancer later in life.

Short-Chain PFAS

As long-chain PFAS compounds (the most common being PFOA[2] and PFOS[2]) were phased out, industries increasingly turned to short-chain PFAS replacements such as PFBA[2] and PFBS[2]. Multiple

[2] PFOA - Perfluorooctanoic acid; PFOS – Perfluorbutanesulfonic acid; PFBA – Perfluorooctanesulfonic acid; PFBS – Perfluorooctanesulfonic acid

studies over the years have documented these as regrettable substitutes, as the replacements induce similar health concerns. Although cancer outcomes remain under-studied, a notable computational toxicology study in 2025 revealed that these alternatives interact with key cancer-related proteins (such as HDAC3 and PPARD) suggesting that short-chain PFAS likely to pose similar reproductive and carcinogenic risks (Jain et al. 2025). This growing body of evidence challenges the assumption that replacement PFAS compounds are inherently safer and underscores the need for precautionary approaches as the chemical landscape evolves.

What can we do to reduce potential cancer risks associated with PFAS?

While the evidence linking these “forever chemicals” to cancer continues to mount, progress on prevention remains slow. The prevention strategies CENSWPA identified in previous years remains relevant, but the urgency has never been higher.

To effectively reduce cancer risks, we must move beyond playing "whack-a-mole" with individual chemicals. We need strategies that address PFAS as a class. Decades of research show that these substances are persistent, bioaccumulative, and toxic. Historically, restricting a single compound has only led to replacing one harmful chemical with a similar, less-studied cousin. Real-world exposures are not experienced in isolation; exposures occur as complex mixtures in our water, soil, air, and everyday products such as textiles and food packaging. Only by restricting the entire class can we achieve meaningful risk reduction.

The European Union is moving toward a universal PFAS restriction. Because global companies often manufacture products to meet the most stringent standards, this will eventually benefit U.S. consumers. While federal regulatory pathways remain stalled, the landscape for PFAS prevention at the state-level has shifted from "emerging" to "aggressive" actions. A coalition of proactive states is leading the charge by banning PFAS as a class rather than chemical-by-chemical. For example, Minnesota began phased prohibitions that will culminate in a total ban on intentionally added PFAS by 2032. Maine and New Mexico have taken similar steps to ban PFAS by 2032. Other states are actively pursuing new prohibitions or reporting requirements to reduce PFAS use in every-day products.

While many states are already implementing class-based bans and disclosure requirements, Pennsylvania’s continued strategy of isolated, product-specific restrictions is a growing liability. For example, a bipartisan bill is moving through the PA House to ban PFAS in cosmetics, dental floss, and menstrual products. Additionally, while the state recently passed a ban on PFAS-containing firefighting foam (Class B), these actions remain narrow in scope. Unlike the "leader" states that have banned PFAS as an entire class, Pennsylvania still lacks comprehensive, class-based protections on general consumer textiles, furniture, and industrial "upstream" production. Without a transition to class-based policy, the Commonwealth risks becoming a "downstream market" for toxic products that are restricted elsewhere.

Because PFAS are extremely persistent, legacy contamination in our environment will pose risks for decades, even if production stops today. We must pair upstream prevention with active mitigation. We need transparent, public access to data regarding drinking water, private wells, and hotspots near industrial sites and landfills. Remediation of contaminated soil and improved wastewater treatment are essential to prevent secondary exposures. On an individual level, certified point-of-use filtration (like

activated carbon or reverse osmosis) can substantially reduce PFAS in drinking water while we wait for long-term infrastructure upgrades.

Reducing PFAS exposures in daily life is possible with individual and institutional actions. Consumer products act as ongoing reservoirs for PFAS. Cookware, carpets, and upholstery can release these chemicals into indoor air and dust over time. Schools and healthcare facilities can use their purchasing power to demand PFAS-free furnishings and food service ware. Being mindful of older "stain-resistant" or "water-repellent" products and avoiding new PFAS-containing materials can limit chronic exposure. For those undergoing cancer treatment or with heightened vulnerability, targeted guidance on water filtration and indoor air quality is a critical, immediate intervention.

Conclusion

The 2025 scientific literature continues to strengthen the evidence that environmental exposures—including PFAS, air pollution, microplastics, and endocrine-disrupting chemicals contribute to cancer risk, progression, and survivorship. Across these research areas, common themes emerge: exposures are widespread, effects are cumulative, and risks are unevenly distributed, with greater burdens falling on vulnerable communities. While important knowledge gaps remain, the science points toward the need for prevention-oriented action that reduces exposures before disease occurs. Pairing this growing body of evidence with practical strategies—such as improved monitoring, safer product choices, institutional purchasing policies, and state-level policy leadership—offers critical pathways to protect public health while the science documenting harms continues to evolve.

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