Invisible Enemies: The Pollutants Triggering Immune Dysfunction

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Today’s Presentation Part 1: Sources

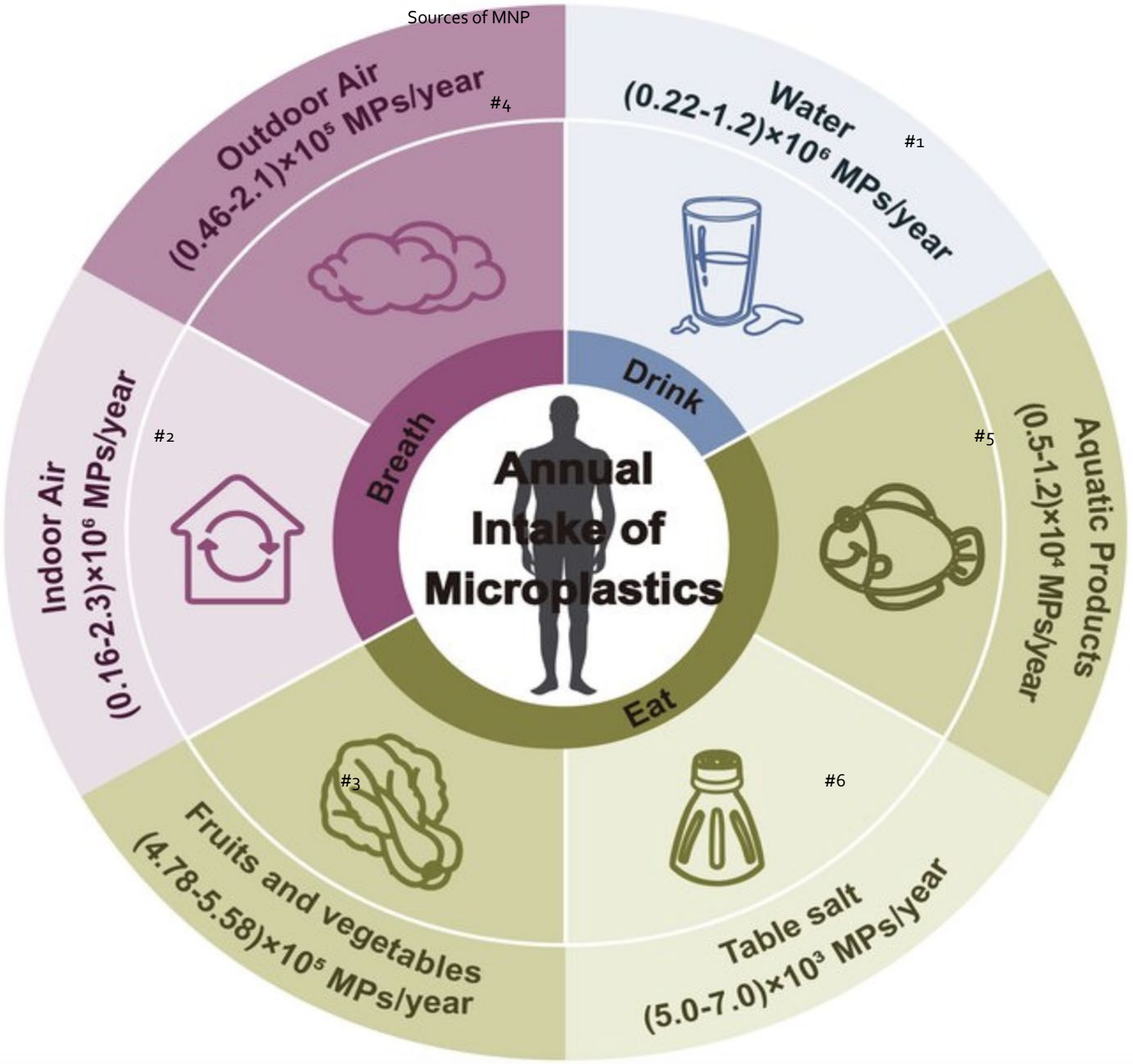
- Microplastics
- Dioxins
- Particulate Matter (PM)
- Formaldehyde
- Benzene

Microplastics

Indoor air has much more microplastics than outdoor air

Where They Come From

(notice they forgot to test coffee cups and lids)



DOI:10.1007/s40726-023-00273-8

Indoor air has more microplastics, texiles contribute.

Indoor Air=Dust

PET:Clear plastic bottles for water, soda and other drinks. Also textiles.

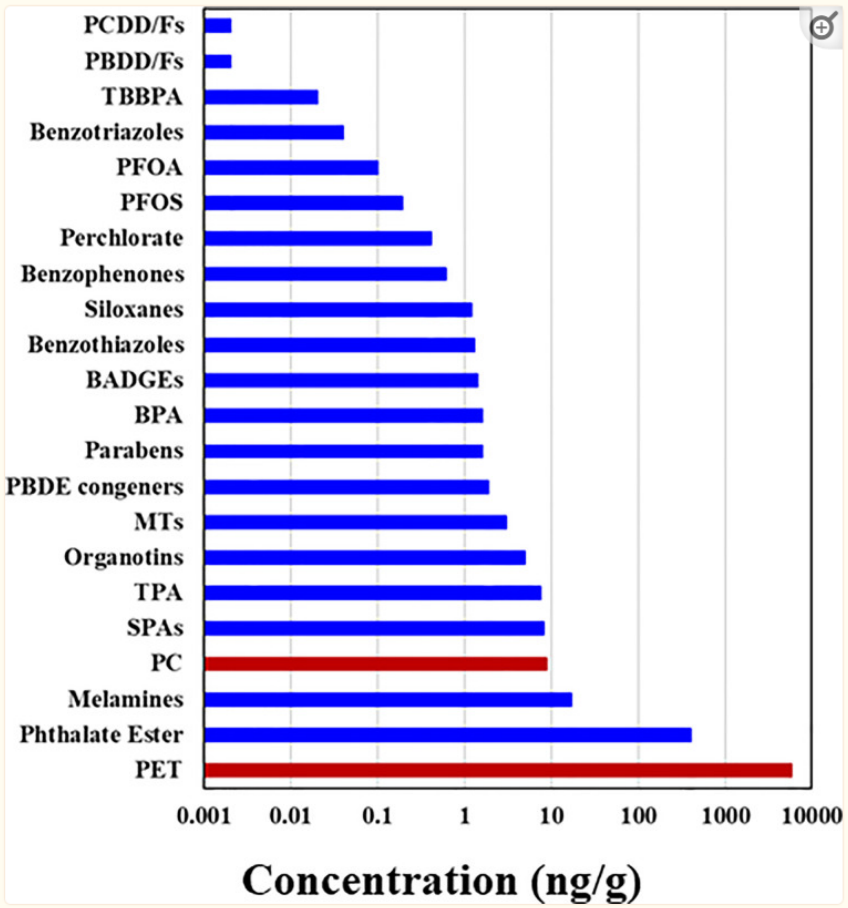


Figure 2

Median concentrations of polyethylene terephthalate (PET) and polycarbonate (PC)-based microplastics measured in indoor dust samples collected from Albany, New York, USA, compared with those of other chemicals [see Zhang

PMID: 34484127

Current Opinion in Environmental Science & Health
Volume 1, February 2018, Pages 1-5

Microplastics in air: Are we breathing it in?

Johnny Gasperi [1], Stephanie L. Wright [2], Rachid Dris [1], France Collard [1], Corinne Mandin [3], Mohamed Guerrouache [4], Valérie Langlois [4], Frank J. Kelly [2], Bruno Tassin [1]

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https://doi.org/10.1016/j.coesh.2017.10.002

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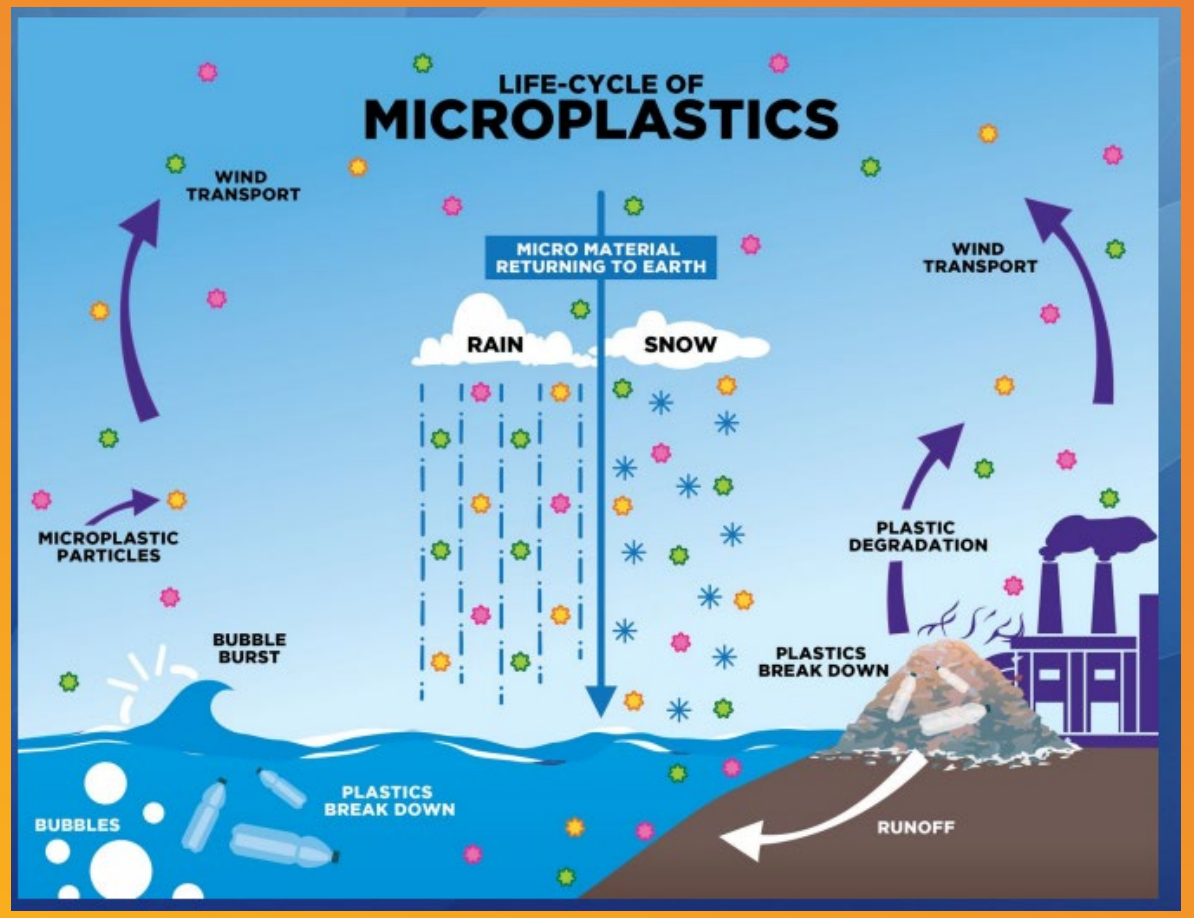
Highlights

- More than 60 million metric tons of plastic fibers were produced in 2016.
- Fragmented fibres – fibrous microplastics – are present in outdoor and indoor air.
- The inhalation of airborne fibrous microplastics is a question of size.
- Inhaled fibrous microplastics are likely to be biopersistent.
- Airborne fibrous microplastics may also carry pollutants.



Associated contaminants such as Polycyclic Aromatic Hydrocarbons (PAHs) could desorb and lead to genotoxicity while the plastic itself and its additives (dyes, plasticizers) could lead to health effects including reproductive toxicity, carcinogenicity and mutagenicity.

New Tech:
240,000 micro-nanoplastics in one plastic bottle of water

RESEARCH ARTICLE | CHEMISTRY |

Rapid single-particle chemical imaging of nanoplastics by SRS microscopy

Naixin Qian, Xin Gao, Xiaoqi Lang, +5, and Wei Min Authors Info & Affiliations

Edited by Eric O. Potma, University of California, Irvine, CA; received January 11, 2023; accepted October 24, 2023 by Editorial Board Memb Shaul Mukamel

January 8, 2024 | 121 (3) e2300582121 | https://doi.org/10.1073/pnas.2300582121

169,043 PDF/E

Significance

Micro-nano plastics originating from the prevalent usage of plastics have raised increasingly alarming concerns worldwide. However, there remains a fundamental knowledge gap in nanoplastics because of the lack of effective analytical techniques. This study developed a powerful optical imaging technique for rapid analysis of nanoplastics with unprecedented sensitivity and specificity. As a demonstration, micro-nano plastics ir bottled water are analyzed with multidimensional profiling of individual plastic particles. Quantification suggests more than 10^5 particles in each liter of bottled water, the majorit of which are nanoplastics. This study holds the promise to bridge the knowledge gap on plastic pollution at the nano level.

Microplastics in Paper Cups

- Polyethylene liner
- 25,000 microplastics released into hot beverage in 15 minutes
- Toxic heavy metals like Pb, Cr, and Cd were detected in the films which can be transferred into hot water
- The lid leaches BPA into the beverage as well

PMID: 33091697

MNP in the Human Brain- Feb 3rd, 2025

“nanoscale shard-like fragments” of polyethylene

increasing MNP concentrations (50%) over time in liver and brain (frontal cortex) samples from 2016-2024.

UNM Researchers Find Alarmingly High Levels of Microplastics in Human Brains – and Concentrations are Growing Over Time

By Michael Haederle | February 28, 2025

Caption

Nature Medicine 2025 PMID: 3990104

MNP in the Human Brain- Feb 3rd, 2025

Even greater accumulation of MNPs was observed in a cohort of decedent brains with documented dementia diagnosis, with notable deposition in cerebrovascular walls and microglia.

The technique detected and quantified 12 different polymers, the most common of which was **polyethylene**, which is widely used for **packaging and containers**, including **bottles and cups**.

Microplastics Effect on Immune System

- Microplastics affect intracellular signaling pathways
- ROS generation leads to production of DAMPS (danger associated molecular patterns) and interfere with TLRs, cytokine responses in immune cells
- Influenced by the chemicals the plastics adsorb

View > Front Toxicol. 2022 Sep 27:4:956885. doi: 10.3389/ftox.2022.956885. eCollection 2022.

mpacts of microplastics on immunity

Wenjie Yang [1], Nahar Jannatun [1], Yanqiao Zeng [1], Tinghao Liu [1], Guofang Zhang [1], Chunying Chen [2 3], Yang Li [1]

Affiliations + expand

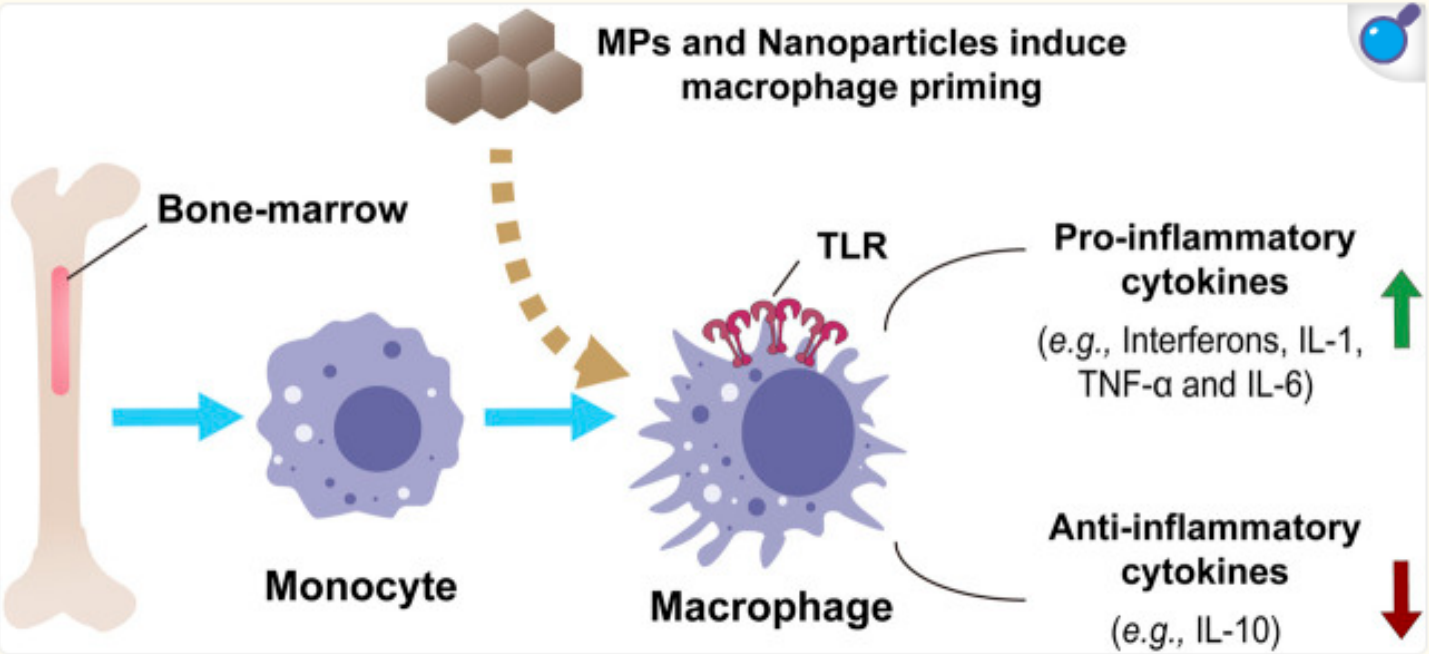
PMID: 36238600 PMCID: PMC9552327 DOI: 10.3389/ftox.2022.956885

Abstract

Most disposable plastic products are degraded slowly in the natural environment and continually turned to microplastics (MPs) and nanoplastics (NPs), posing additional environmental hazards. The toxicological assessment of MPs for marine organisms and mammals has been reported. Thus, there is an urgent need to be aware of the harm of MPs to the human immune system and more studies about immunological assessments. This review focuses on how MPs are produced and how they may interact with the environment and our body, particularly their immune responses and immunotoxicity. MPs can be taken up by cells, thus disrupting the intracellular signaling pathways, altering the immune homeostasis and finally causing damage to tissues and organs. The generation of reactive oxygen species is the mainly toxicological mechanisms after MP exposure, which may further induce the production of danger-associated molecular patterns (DAMPs) and associate with the processes of toll-like receptors (TLRs) disruption, cytokine production, and inflammatory responses in immune cells. MPs effectively interact with cell membranes or intracellular proteins to form a protein-corona, and combine with external pollutants, chemicals, and pathogens to induce greater toxicity and strong adverse effects. A comprehensive research on the immunotoxicity effects and mechanisms of MPs, including various chemical compositions, shapes, sizes, combined exposure and concentrations, is worth to be studied. Therefore, it is urgently needed to further elucidate the immunological hazards and risks of humans that exposed to MPs.

Keywords: combined exposure; immune response; immunotoxicity; microplastics; phagocytosis; protein-corona.

FIGURE 3.



Open in a new tab

In vitro model of macrophage priming and innate immune functions in human monocytes and monocyte-derived macrophages by nanoparticles or MPs. Bone-marrow-derived macrophages show an innate response in producing and regulating inflammatory and anti-inflammatory cytokines.

Microplastics and Immune Functions

PMID: 36238600

Microplastics and Cancer

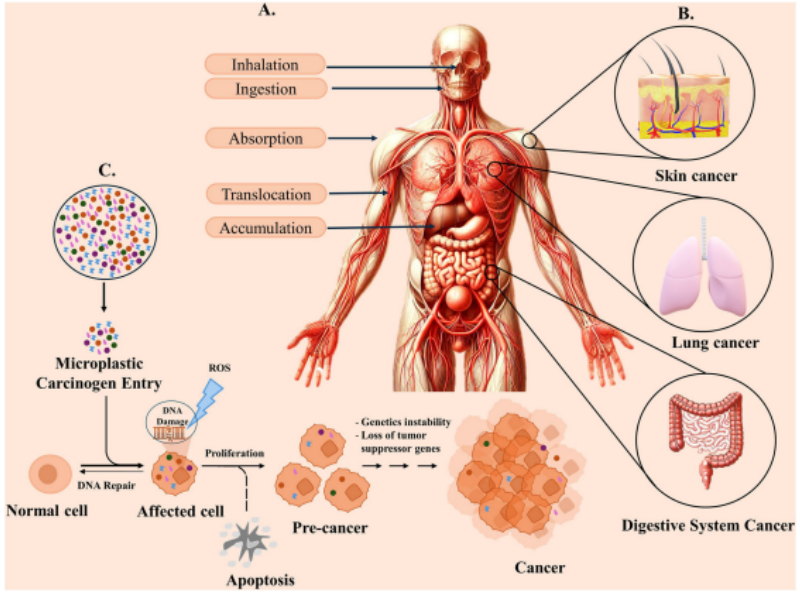


Fig. 1 **A** Microplastics enter the body through various routes including Inhalation, Ingestion, dermal absorption followed by translocation, and subsequent accumulation within tissues. **B** These microplastics can induce the development of various cancers such as Lung cancer, Skin cancer, and Digestive System Cancers. **C** Furthermore, the infiltration of microplastics into normal cells results in the accumulation of these particles within the cellular environment. This accumulation may instigate DNA damage within the cells via ROS or other mechanisms. Additionally, it may lead to the proliferation of affected cells, potentially transforming them into pre-cancerous cells. With the accumulation of genetic instability and the loss of tumor suppressor genes, these pre-cancerous cells can progress into full-blown cancer or undergo apoptosis

Cell Biochemistry and Biophysics (2024) 82:3109–3121
https://doi.org/10.1007/s12013-024-01436-0

REVIEW PAPER

Microplastics – A Growing Concern as Carcinogens in Cancer Etiology: Emphasis on Biochemical and Molecular Mechanisms

Naveen Kumar[1] · Mridul Lamba[1] · Ashok Kumar Pachar[2] · Sonal Yadav[1] · Arbind Acharya[3]

Accepted: 11 July 2024 / Published online: 20 July 2024

Abstract
In today's world, the widespread presence of microplastics is undeniable, with concentrations found in various environments, including up to 1000 particles per liter in seawater and up to 10 particles per cubic meter in the atmosphere. Originating from diverse sources, both intentional and unintentional, these minuscule fragments, measuring less than 5 mm, pose significant threats to environmental and human health. Recent research has uncovered a concerning link between microplastics and cancer, prompting urgent investigation. Studies demonstrate microplastics can infiltrate cells, disrupt biological processes, and potentially foster carcinogenic environments. From inducing DNA damage and oxidative stress to triggering inflammatory responses and dysregulating cellular pathways, microplastics exhibit a multifaceted capability in contributing to cancer development. Furthermore, microplastics act as carriers for a range of contaminants, compounding their impact on human health. Their accumulation within tissues and organs raises concerns for short and long-term health consequences, including chronic diseases, reproductive issues, and developmental abnormalities. This review explores the biochemical and molecular mechanisms underlying the interaction between microplastics and cellular systems, providing insights into routes of exposure and health effects, with a focus on lung, skin, and digestive system cancers. As we confront this pressing environmental and public health challenge, a deeper understanding of the microplastic-cancer relationship is crucial to safeguarding the well-being of present and future generations.

Keywords Plastics · Lung · Skin · Digestive system · Tumor

Introduction

Microplastics, identified as tiny plastic fragments smaller than 5 mm in length, have been found in diverse settings, including concentrations of up to 1000 particles per liter in seawater and up to 10 particles per cubic meter in the atmosphere [1–3]. These tiny fragments are broadly categorized into primary and secondary forms. Primary microplastics enter the environment at sizes less than 5.0 mm, originating from sources like microfibers from clothing, microbeads, plastic glitter, and plastic pellets. On the other hand, secondary microplastics emerge from the degradation of larger plastic items such as water and soda bottles, fishing nets, plastic bags, and tire wear. While microplastics stem from diverse sources like cosmetics, clothing, food packaging, and industrial activities, a notable proportion, particularly those present in oceanic environments, can be attributed to textiles and clothing, primarily due to the erosion of polyester. Recent studies have highlighted the significant contribution of textile fibers to marine microplastic pollution. For instance, a study by Oliveira et al. (2023) underscores the extensive release of microplastics from textiles into aquatic environments, emphasizing the urgency for mitigation strategies [1, 2, 4]. The primary

✉ Naveen Kumar
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Early Onset Colorectal Cancer and Microplastics?

Interactions between microbiome and intestinal wall- plastics damage both.

› Cancers (Basel). 2023 Jun 24;15(13):3323. doi: 10.3390/cancers15133323.

Could Microplastics Be a Driver for Early Onset Colorectal Cancer?

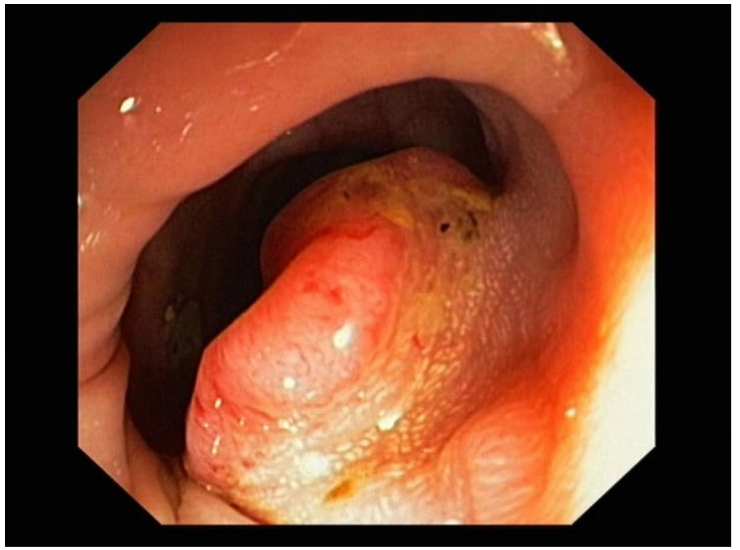
Shelley Li [1], Jacqueline I Keenan [1], Ian C Shaw [2], Frank A Frizelle [1]

Affiliations + expand

PMID: 37444433 PMCID: PMC10340669 DOI: 10.3390/cancers15133323

Abstract

Introduction: The incidence of colorectal cancer in those under 50 years of age (early onset colorectal cancer (EOCRC)) is increasing throughout the world. This has predominantly been an increase in distal colonic and rectal cancers, which are biologically similar to late onset colorectal cancer (LOCRC) but with higher rates of mucinous or signet ring histology, or poorly differentiated cancers. The epidemiology of this change suggests that it is a cohort effect since 1960, and is most likely driven by an environmental cause. We explore the possible role of microplastics as a driver for this change. **Review:** The development of sporadic colorectal cancer is likely facilitated by the interaction of gut bacteria and the intestinal wall. Normally, a complex layer of luminal mucus provides colonocytes with a level of protection from the effects of these bacteria and their toxins. Plastics were first developed in the early 1900s. After 1945 they became more widely used, with a resultant dramatic increase in plastic pollution and their breakdown to microplastics. Microplastics (MPs) are consumed by humans from an early age and in increasingly large quantities. As MPs pass through the gastrointestinal tract they interact with the normal physiological mechanism of the body, particularly in the colon and rectum, where they may interact with the protective colonic mucus layer. We describe several possible mechanisms of how microplastics may disrupt this mucus layer, thus reducing its protective effect and increasing the likelihood of colorectal cancer. **Conclusions:** The epidemiology of increase in EOCRC suggests an environmental driver. This increase in EOCRC matches the time sequence in which we could expect to see an effect of rapid increase of MPs in the environment and, as such, we have explored possible mechanisms for this effect. We suggest that it is



Colorectal Cancer (Colonoscopy)

This image shows adenocarcinoma of the colon.

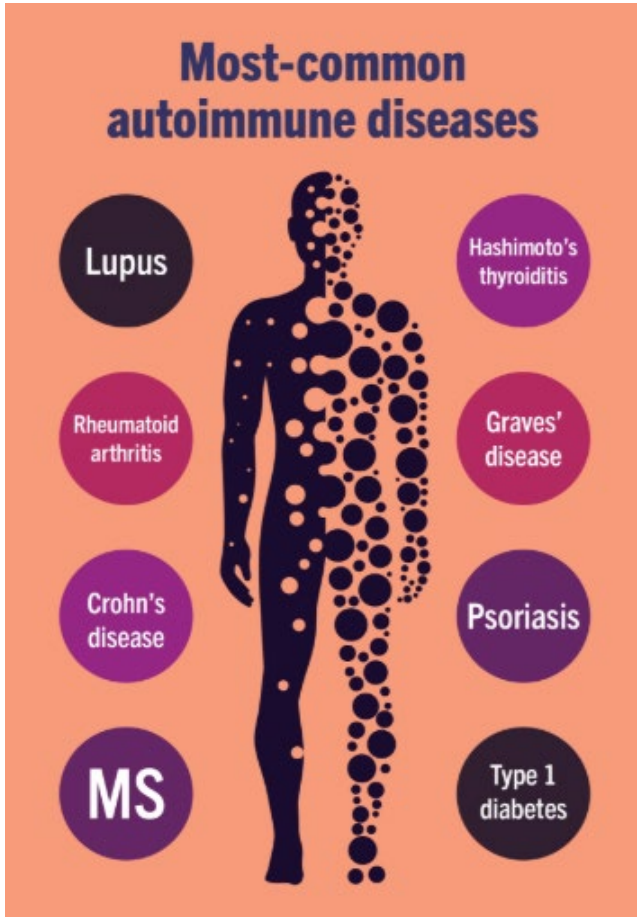
GASTROLAB/SCIENCE PHOTO LIBRARY

In these topics

Colorectal Cancer >

Colorectal Cancer Screening >

BPA and Autoimmunity



Review > Autoimmunity. 2018 Dec;51(8):370-377. doi: 10.1080/08916934.2018.1551374. Epub 2018 Dec 28.

Bisphenol A: A notorious player in the mosaic of autoimmunity

> Autoimmune Dis. 2014 Apr 7;2014:743616. doi: 10.1155/2014/743616

The Potential Roles of Bisphenol A (BPA) Pathogenesis in Autoimmunity

Datis Kharrazian [1,2,*]

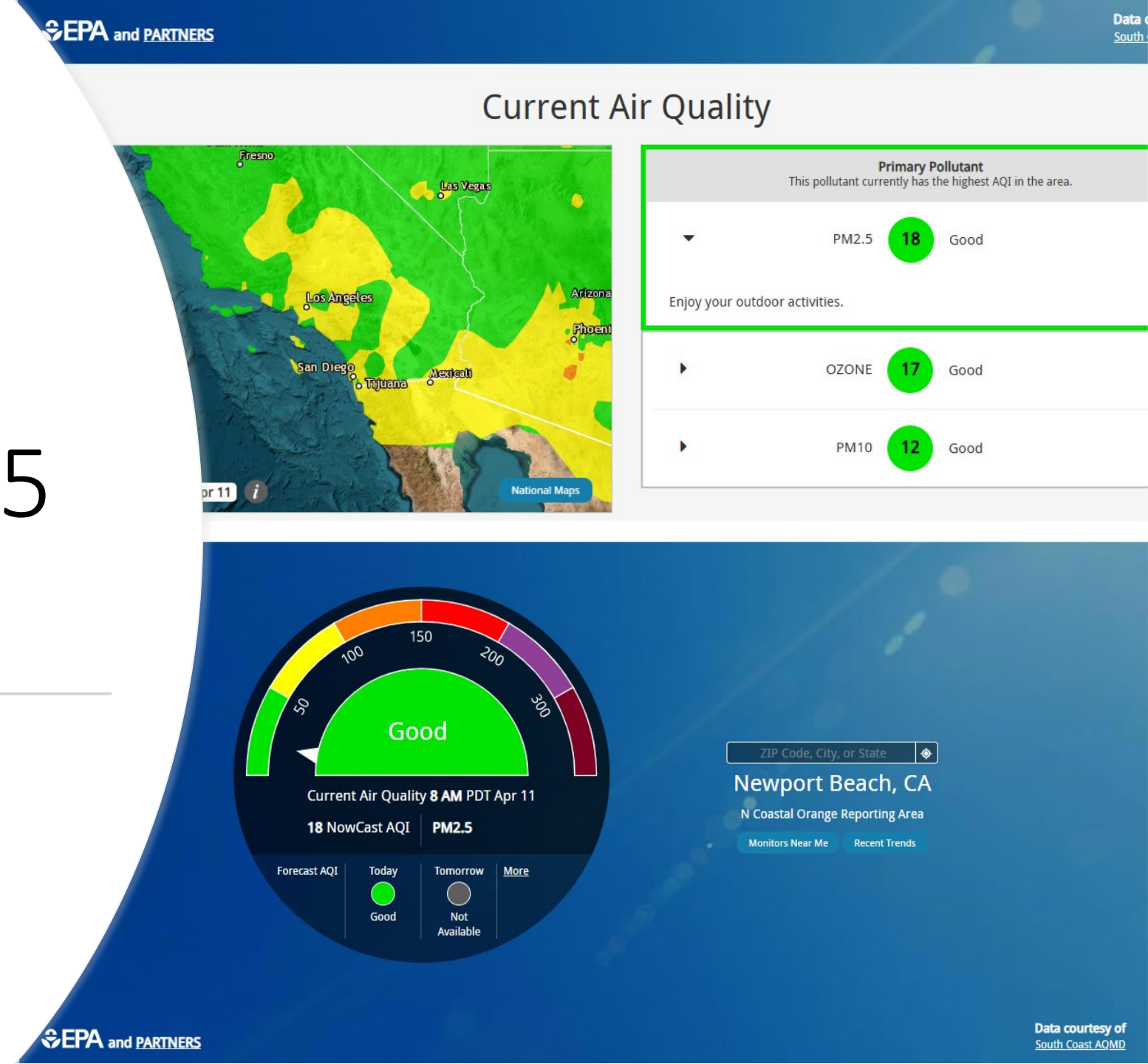
> Toxics. 2019 May 6;7(2):26. doi: 10.3390/toxics7020026.

The Associations between Immunological Reactivity to the Haptenation of Unconjugated Bisphenol A to Albumin and Protein Disulfide Isomerase with Alpha-Synuclein Antibodies

Datis Kharrazian [1 2 3], Martha Herbert [4 5], Aristo Vojdani [6 7]

Particulate Matter or PM2.5 and PM10

www.airnow.gov



Includes outdoor air that enters through open windows, doors

Indoor Air Sources of PM

- Combustion activities,
  - burning candles
  - use of fireplaces
  - use of unvented space heaters
  - kerosene heaters and
  - cooking
  - tobacco and other smoking products.

PM2.5 and Immune Dysfunction

- $PM_{2.5}$, which impairs the immune cells in response to microbial infections and potentially contributes to the development of severe diseases in the respiratory tract
- Several toxic components, including polycyclic aromatic hydrocarbons (PAHs), aliphatic/chlorinated hydrocarbons, nitro PAHs/ketones/quinones and cadmium, lead, zinc nickel, chromium, arsenic or copper, are usually in corporated in the $PM_{2.5}$
- PMID: 39231594

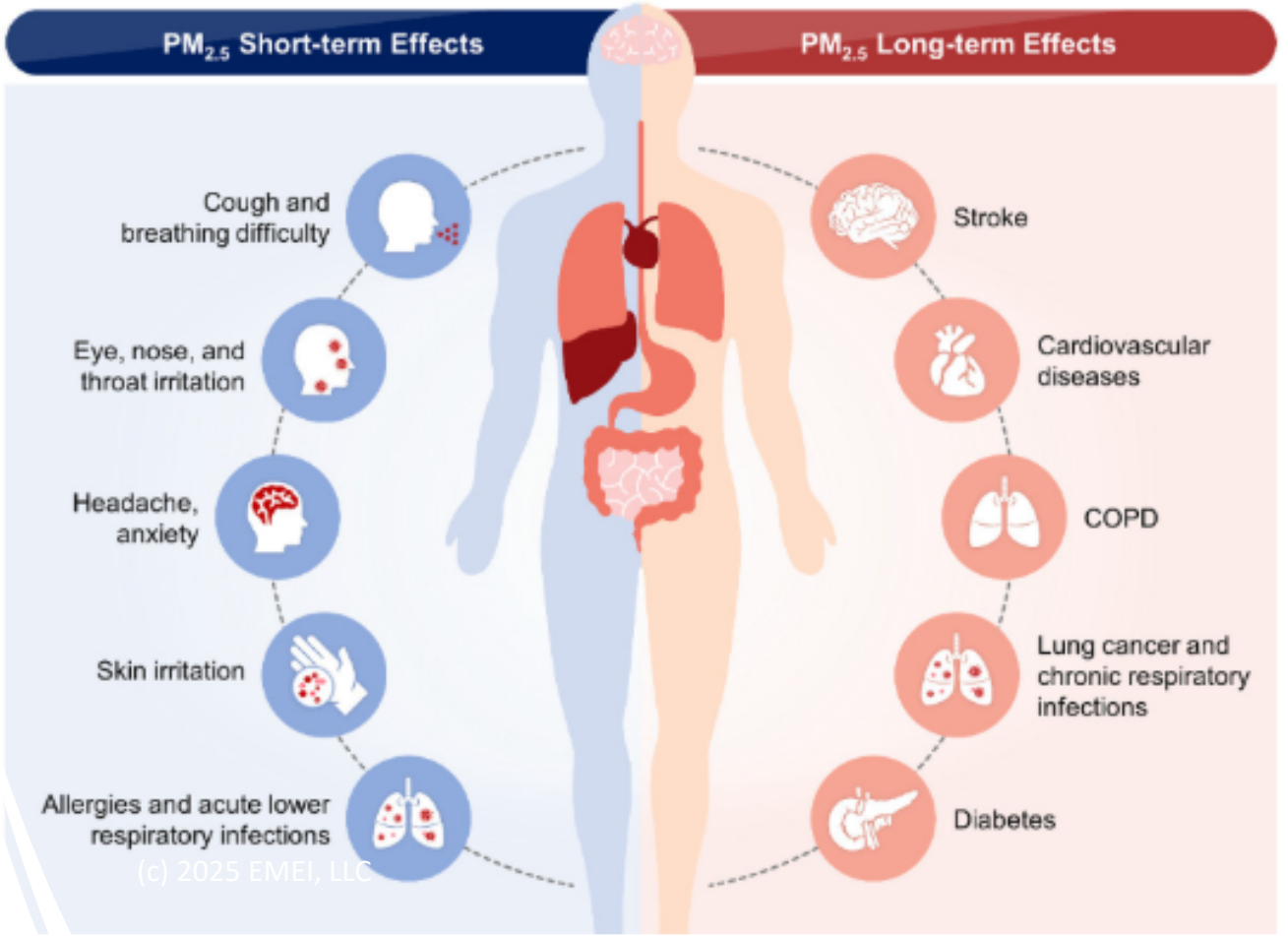
- $PM_{2.5}$ manipulates immune response and promotes disease development
- $PM_{2.5}$ disrupts the integrity of the epithelial barrier and the cytokine expression, the first line of defense against pathogens
- $PM_{2.5}$ alters the distribution of the alveolar macrophage population and its functions in the lung
- $PM_{2.5}$ stimulates inflammation cascades for recruiting immune cells to injured sites
- $PM_{2.5}$ exacerbates bacterial infectivity in the respiratory system
- $PM_{2.5}$ alters microbiota composition in the respiratory tract
- $PM_{2.5}$ impedes immune response against viral infections

Diseases Associated with PM

- Respiratory and Cardiovascular diseases
- IARC lists PM2.5 as a human carcinogen: breast, lung, digestive cancers
- Alzheimer's PMID: 35840480
- Autism PMID: 23404082
  PMID: 38365642

PMID: 37869561

2. Short- and long-term effects of $PM_{2.5}$ on human health. The inhalation of culate matter (PM) can irritate the lining of the nasal cavity, thereby inducing runny e and cough. Inhaled PM can also travel deep down the airways and enter the lungs, us triggering inflammation and causing shortness of breath, as well as worsening reexisting respiratory diseases such as asthma and chronic obstructive pulmonary disease (COPD). The inflammation can also spread to other parts of the body, leading to the risk of cardiovascular diseases. Lung cancer-related deaths are also related to the adverse effects of PM.



PM2.5 and Risk of Severe Covid- 19

- For every 1ug/m³ increase in PM2.5, 18% increase in risk for hospitalization for Covid-19
- For comparison, The peak reading in Los Angeles briefly topped 400 µg/m3 on January 8 in the LA Fires
- Average reading around 35
- Increased risk of hospitalization of 65 times

Respirology. 2021 Dec;26(12):1181-1187. doi: 10.1111/resp.14140. Epub 2021 Aug 30.

Air pollution and the pandemic: Long-term $PM_{2.5}$ exposure and disease severity in COVID-19 patients

Angelico Mendy [1], Xiao Wu [2], Jason L Keller [3], Cecily S Fassler [1], Senu Apewokin [4], Tesfaye B Mersha [5], Changchun Xie [6], Susan M Pinney [1]

Affiliations + expand

PMID: 34459069 PMCID: PMC8662216 DOI: 10.1111/resp.14140

Abstract

Background and objective: Ecological studies have suggested an association between exposure to particulate matter ≤2.5 µm ($PM_{2.5}$) and coronavirus disease 2019 (COVID-19) severity. However, these findings are yet to be validated in individual-level studies. We aimed to determine the association of long-term $PM_{2.5}$ exposure with hospitalization among individual patients infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

Methods: We estimated the 10-year (2009-2018) $PM_{2.5}$ exposure at the residential zip code of COVID-19 patients diagnosed at the University of Cincinnati healthcare system between 13 March 2020 and 30 September 2020. Logistic regression was used to determine the odds ratio (OR) and 95% CI for COVID-19 hospitalizations associated with $PM_{2.5}$, adjusting for socioeconomic characteristics and comorbidities.

Results: Among the 14,783 COVID-19 patients included in our study, 13.6% were hospitalized; the geometric mean (SD) $PM_{2.5}$ was 10.48 (1.12) µg/m^3. In adjusted analysis, 1 µg/m^3 increase in 10-year annual average $PM_{2.5}$ was associated with 18% higher hospitalization (OR: 1.18, 95% CI: 1.11-1.26). Likewise, 1 µg/m^3 increase in $PM_{2.5}$ estimated for the year 2018 was associated with 14% higher hospitalization (OR: 1.14, 95% CI: 1.08-1.21).

Conclusion: Long-term $PM_{2.5}$ exposure is associated with increased hospitalization in COVID-19. Therefore, more stringent COVID-19 prevention measures may be needed in areas with higher $PM_{2.5}$ exposure to reduce the disease morbidity and healthcare burden.

Keywords: COVID-19 hospitalization; PM2.5 exposure; SARS-CoV-2; air pollution; coronavirus disease.

Dioxins


BREAKING NEWS
TOXIC CHEMICALS RELEASED FROM DERAILED TRAIN
DANGEROUS CHEMICAL RELEASED
DEPARTMENT OF TRANSPORTATION
UNITED STATES OF AMERICA
CBS NEWS
MSNBC
DEADLY DIOXINS IN
EAST PALESTINE, OHIO

9/11 Left a Toxic Legacy That Has Paid Out $15 Billion as of 12/31/24

9/11 World Trade Center Health Program

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Exhibition Home / Toxins and Health Impacts

Toxins and Health Impacts

The collapse of the Twin Towers created massive dust clouds that filled the air and left hundreds of highly populated city blocks covered with ash, debris, and harmful particles.

In Lower Manhattan, the plane crashes — which resulted in the collapse of the Twin Towers — created massive dust clouds that filled the air and left hundreds of highly populated city blocks covered with ash, debris, and harmful particles, including asbestos, silica, metals, concrete, and glass. Fires within the debris pile and the collapse of 7 WTC burned through the end of December 2001 with continued flare-ups in 2002, releasing carcinogenic combustion by-products. These contaminants remained in Lower Manhattan and parts of Brooklyn for an undetermined amount of time after 9/11. Responders, local workers, residents, students, and others had potential for acute exposures in the early days and continuing exposure from residual materials — indoors and outside — as well as exposure to toxic gases, smoke, vapors, and combustion

Pentagon After Attack September 11, 2001.
Photograph courtesy of the National Archive and Records Administration, DM-SD-02-03902.

9/11: Benzene, Dioxins, Asbestos, Heavy Metals, Cement, Plastics

The Impact of 9/11 on Cancer Rates

Sept. 3, 2024

The tragic events of September 11, 2001, not only reshaped the landscape of New York City but also had profound and lasting impacts on the health of those exposed to the aftermath. In the years since the attacks, first responders, rescue workers, and residents who encountered the toxic dust and debris at Ground Zero have faced increased risks of developing various cancers. The connection between 9/11 and the rise of these cancers underscores the need for continued support and care for those affected, as well as a recognition of the long-term health consequences borne by these individuals.

CA: A Cancer Journal for Clinicians
The flagship journal of the American Cancer Society

Review Article | Open Access

Cancer risk among World Trade Center rescue and recovery workers: A review

Paolo Boffetta MD, Charles B. Hall PhD, Andrew C. Todd PhD, David G. Goldfarb MPH, Maria J. Schymura PhD, Jiehui Li MBBS, MS, James E. Cone MD, MPH, Rachel Zeig-Owens DrPH, MPH

First published: 24 March 2022 | https://doi.org/10.3322/caac.21723 | Citations: 3

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We thank the following individuals, who were involved in the original studies included in this review: Amy R. Kahn, Baozhen Qiao, Dana Kristjansson, Robert M. Brackbill, Mark R. Farfel, Janette Yung, Erin Takemoto, Mayris P. Webber, Christopher R. Dasaro, Moshe Z. Shapiro, and David J. Prezant.

East Palestine was not the First or Last Chemical Disaster

Chemicals from East Palestine derailment spread to 16 US states, data shows

Rain and snow samples from Wisconsin to Maine and North Carolina after crash show highest pH levels over last decade

Debris from a Norfolk Southern freight train lies scattered and burning along the tracks in East Palestine, Ohio, on 4 February 2023. Photograph: Gene J Puskar/AP

One of the most potently immunosuppressive chemicals known:	thymic involution	decreased host resistance to pathogens and tumors	suppressed fetal lymphocyte development and maturation
suppressed adaptive immune responses- including antibody production	cytotoxic T lymphocyte (CTL) activity	delayed hypersensitivity responses.	PMID: 20146706

Dioxins and Cancer

Comparative Toxicogenomics Database

Home | Search | Analyze | Download | Commercial Users | Help

Dioxins

Basics | Gene Interactions | Genes | Diseases | Phenotypes | Comps | Pathways | GO | Exposure Studies | Exposure Details | References

These diseases are associated with *Dioxins* or its descendants. Each association is *curated* (M *marker/mechanism* and/or T *therapeutic*) and/or *inferred* (via a curated gene interaction).

Disease categories [Show chart]

Filter by | Disease category: Cancer | Association type: ALL | Filter

1–50 of 1,333 results.

First | Previous | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | Next | Last

	Chemical	Disease	Direct Evidence	Enrichment Analysis	Inference Network	Inference Score	References
1.	Tetrachlorodibenzodioxin	Breast Neoplasms	M		492 genes	185.20	376
2.	Tetrachlorodibenzodioxin	Carcinoma	M		163 genes	79.23	48
3.	Tetrachlorodibenzodioxin	Lung Neoplasms	M		255 genes	63.16	216
4.	Tetrachlorodibenzodioxin	Ovarian Neoplasms	M		125 genes	47.44	94
5.	Tetrachlorodibenzodioxin	Lymphatic Metastasis	M		29 genes	24.09	21
6.	Tetrachlorodibenzodioxin	Cholangiocarcinoma	M		28 genes	13.07	15
7.	Tetrachlorodibenzodioxin	Precancerous Conditions	M		96 genes	12.44	37
8.	Tetrachlorodibenzodioxin	Neoplasms	M		68 genes	10.62	73
9.	Tetrachlorodibenzodioxin	Liver Neoplasms	M		140 genes	8.55	79
10.	Tetrachlorodibenzodioxin	Neoplasms, Experimental	M		70 genes	6.77	65
11.	Tetrachlorodibenzodioxin	Adenoma, Liver Cell	M		10 genes	5.97	11
12.	Tetrachlorodibenzodioxin	Uterine Cervical Neoplasms	M		28 genes	5.68	20
13.	Dioxins	Neoplasms	M		27 genes	5.51	34
14.	Tetrachlorodibenzodioxin	Lymphoma, Non-Hodgkin	M		20 genes	5.00	17
15.	Dioxins	Precancerous Conditions	M		36 genes	4.64	23
16.	Tetrachlorodibenzodioxin	Multiple Myeloma	M		41 genes	3.69	35

Dioxins and the Liver

- Liver fibrosis
- Progression toward NAFLD, cirrhosis and cancer

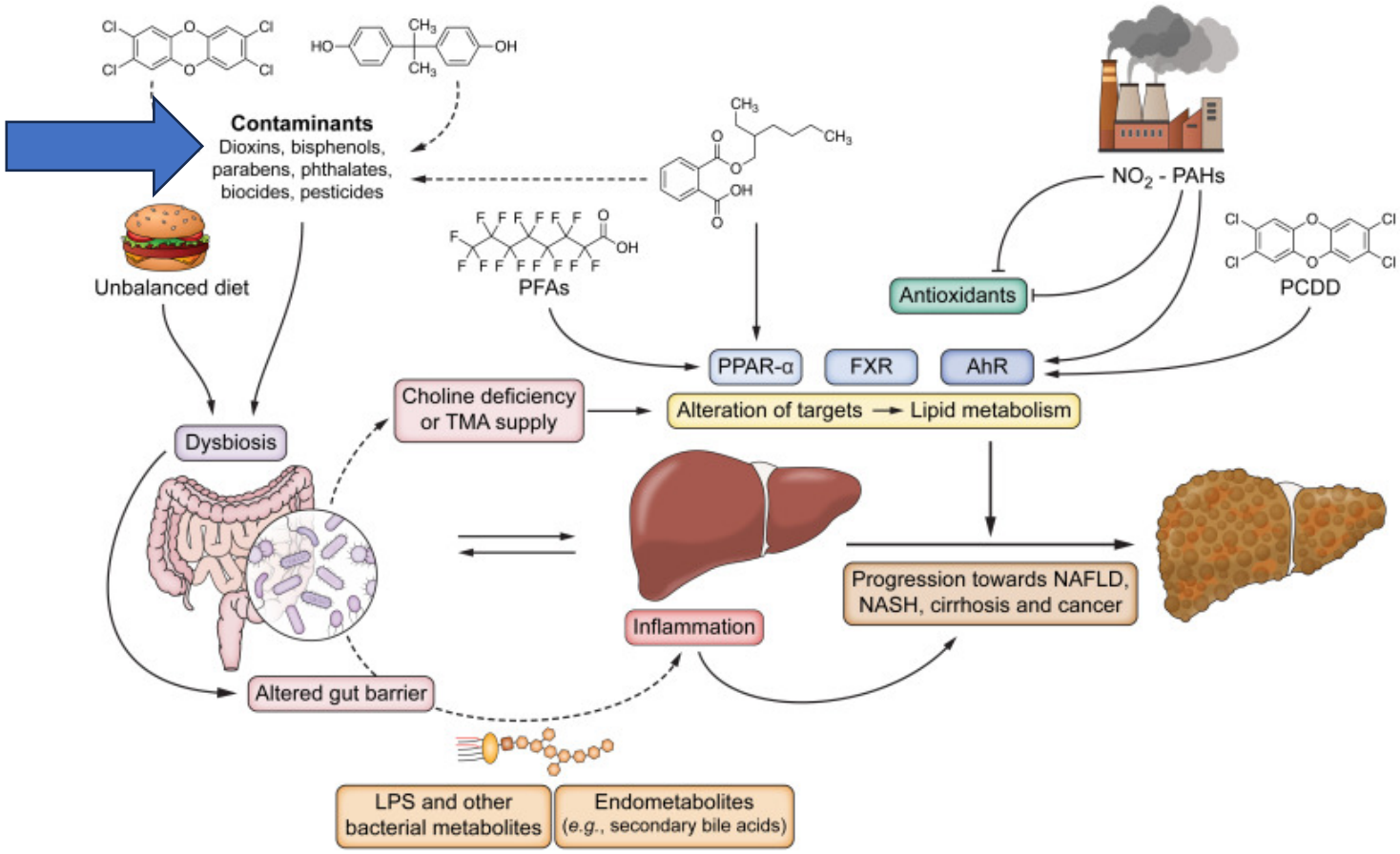

Fig. 1 Metabolic disruption and liver injury following exposure to contaminants and pollutants.

PMID: 36889360

Chemicals Linked to Reduced Vaccine Effectiveness

- PCBs
- Dioxins
- PFAS
- Lead, Arsenic
- Mercury Hair Hg concentrations exceeding 1.2 µg/g in older children were associated with 73.7 higher odds of becoming a non-responder against measles

vaccines MDPI

Vaccines (Basel). 2024 Nov 3;12(11):1252. doi: 10.3390/vaccines12111252

Exposure to Pollutants and Vaccines' Effectiveness: A Systematic Review

Carmela Protano [1], Federica Valeriani [2], Katia Vitale [1], Jole Del Prete [1], Fabrizio Liguori [3], Giorgio Liguori [4], Francesca Gallè [4,*]

Editor: Ralph A Tripp

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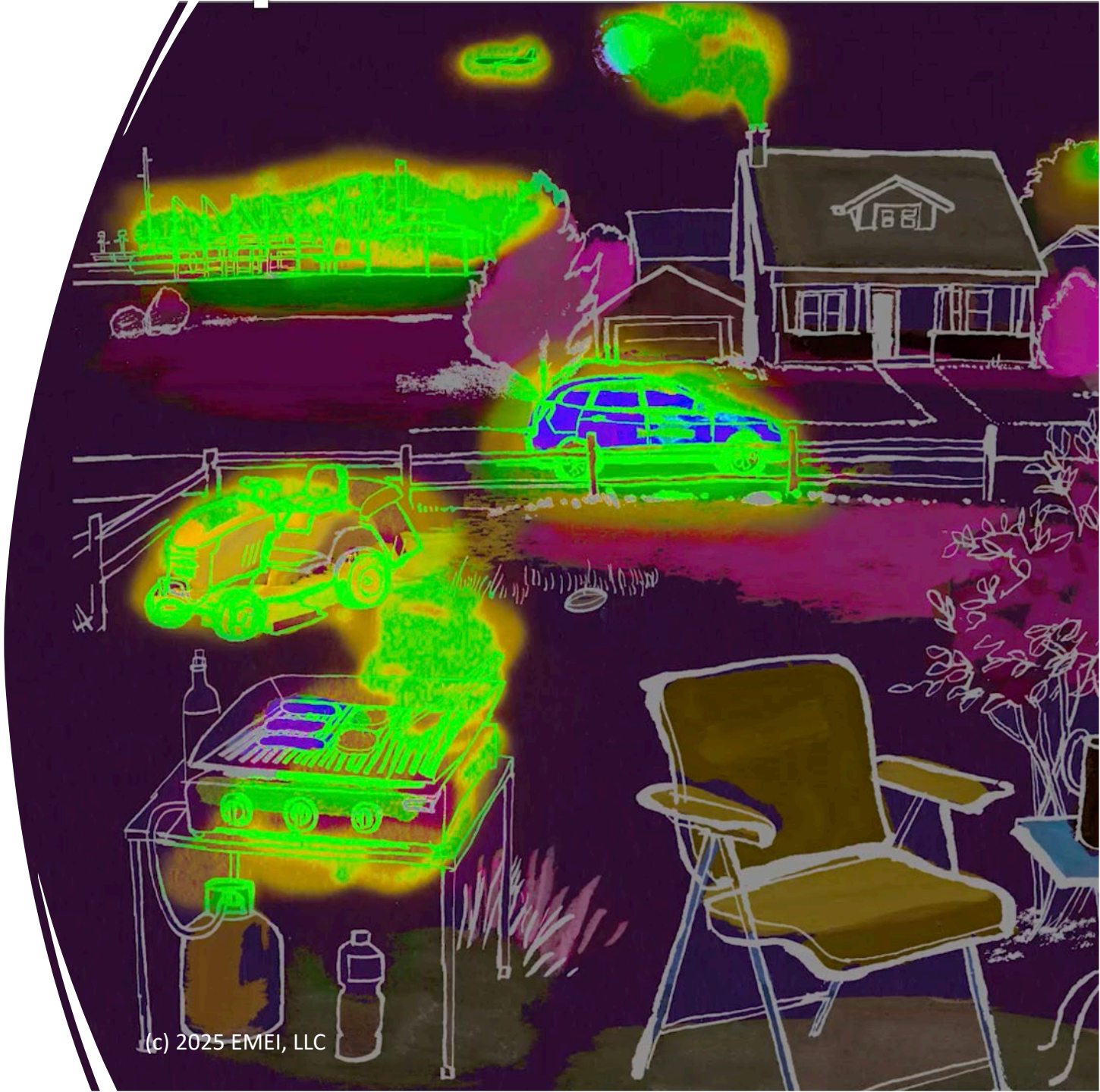
PMCID: PMC11599004 PMID: 39591155

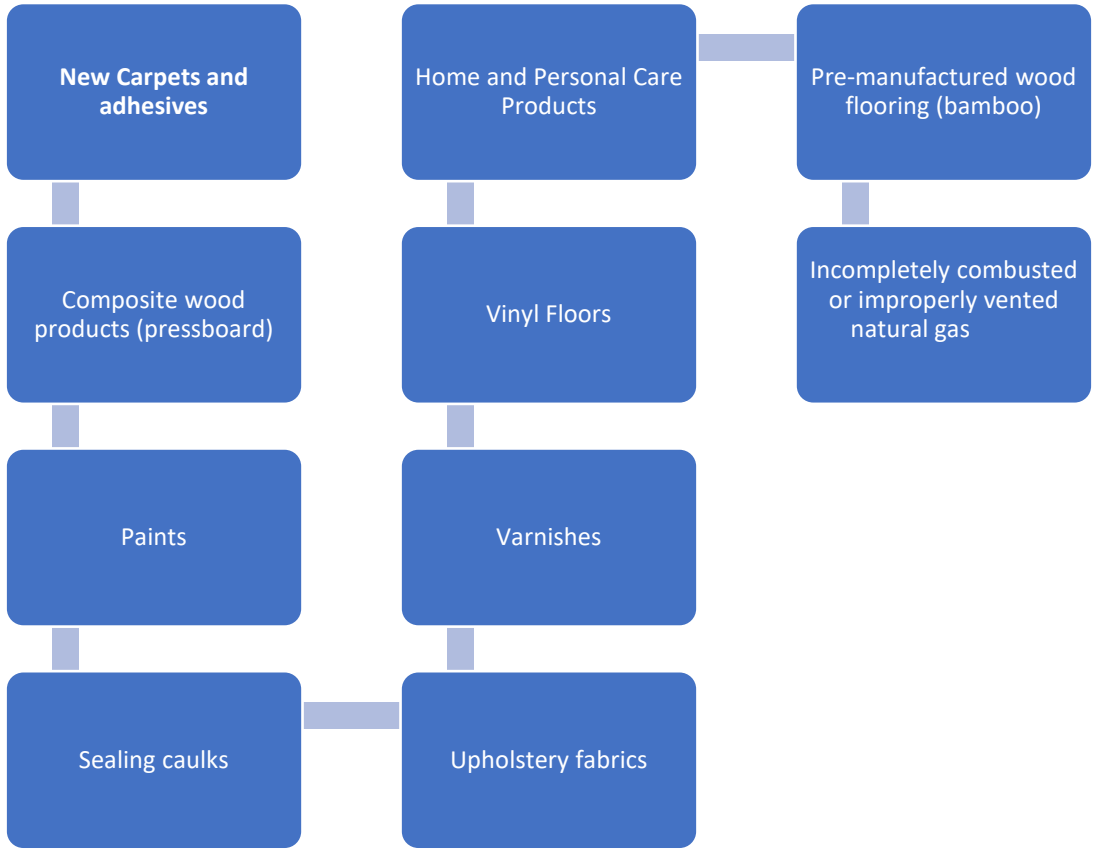
Abstract

Background: Many human activities release harmful substances, contaminating the air, water, and soil. Since exposure to environmental pollutants is currently unavoidable, it is

Formaldehyde and Benzene



Solvents: Exposure in the Home



- Air fresheners (plug-in: formaldehyde)
- Air cleaners that produce ozone
- Cleaning and disinfecting chemicals
- Cosmetics
- Fuel oil, gasoline propane
- Moth balls
- Vehicle exhaust running a car in an attached garage

Centers for Disease Control and Prevention and U.S. Department of Housing and Urban Development. Healthy housing reference manual. Atlanta: US Department of Health and Human Services; 2006.

Formaldehyde

Formaldehyde- a simple molecule made from oxygen, carbon, hydrogen

Considered the most carcinogenic air pollutant in indoor and outdoor air

Formaldehyde is a colorless gas at room temperature with a pungent smell.

Synonyms include: methanal, formalin, paraform, methylene glycol, methyl aldehyde, formol, formalin, formic aldehyde, paraform, formol, formalin (methanol-free), formalith, methylene oxide, tetraoxymethalene, oxomethane, morbicid acid and oxymethylene.

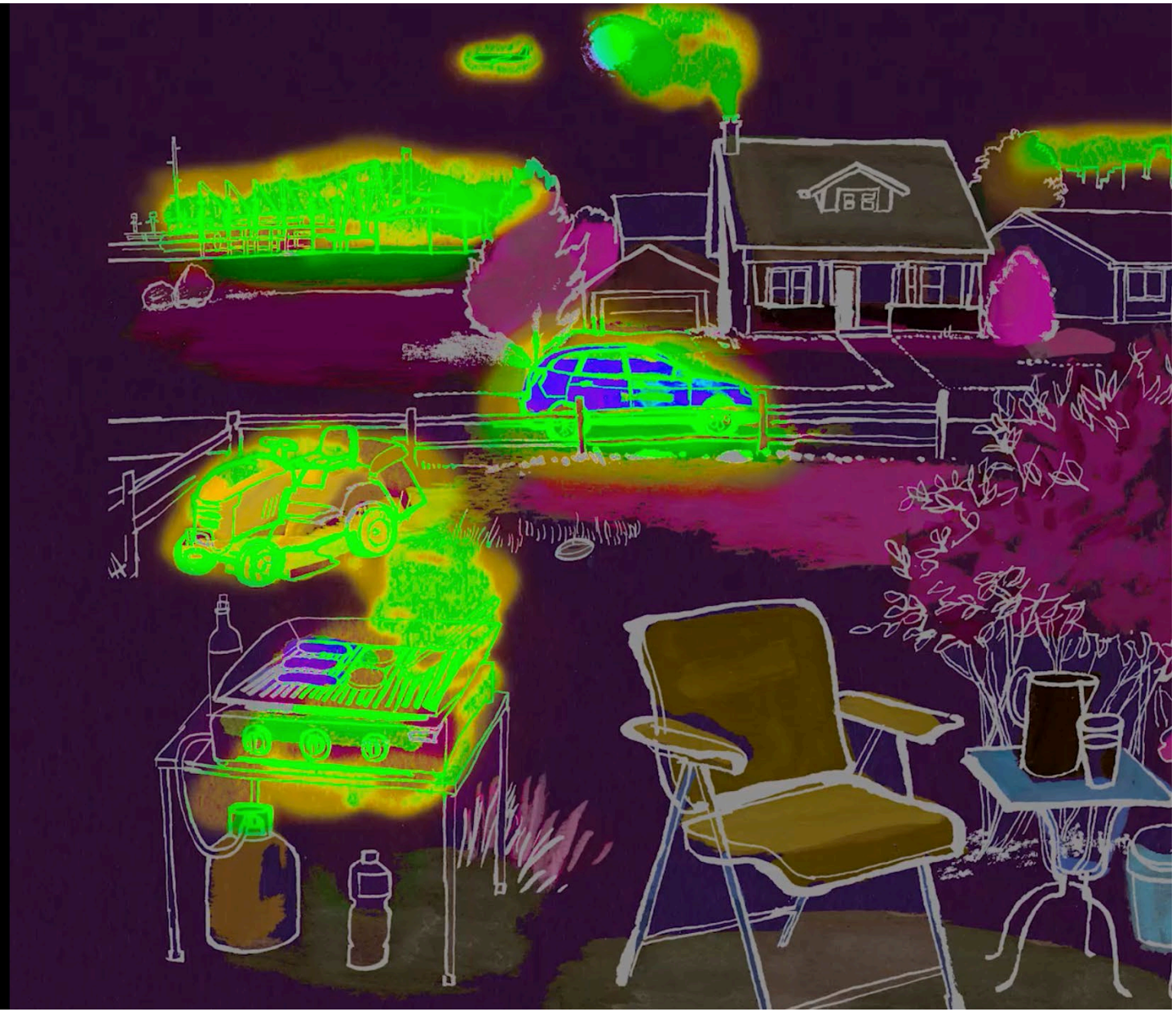
PROPUBLICA

Environment

Formaldehyde Causes More Cancer Than Any Other Toxic Air Pollutant. Little Is Being Done to Curb the Risk.

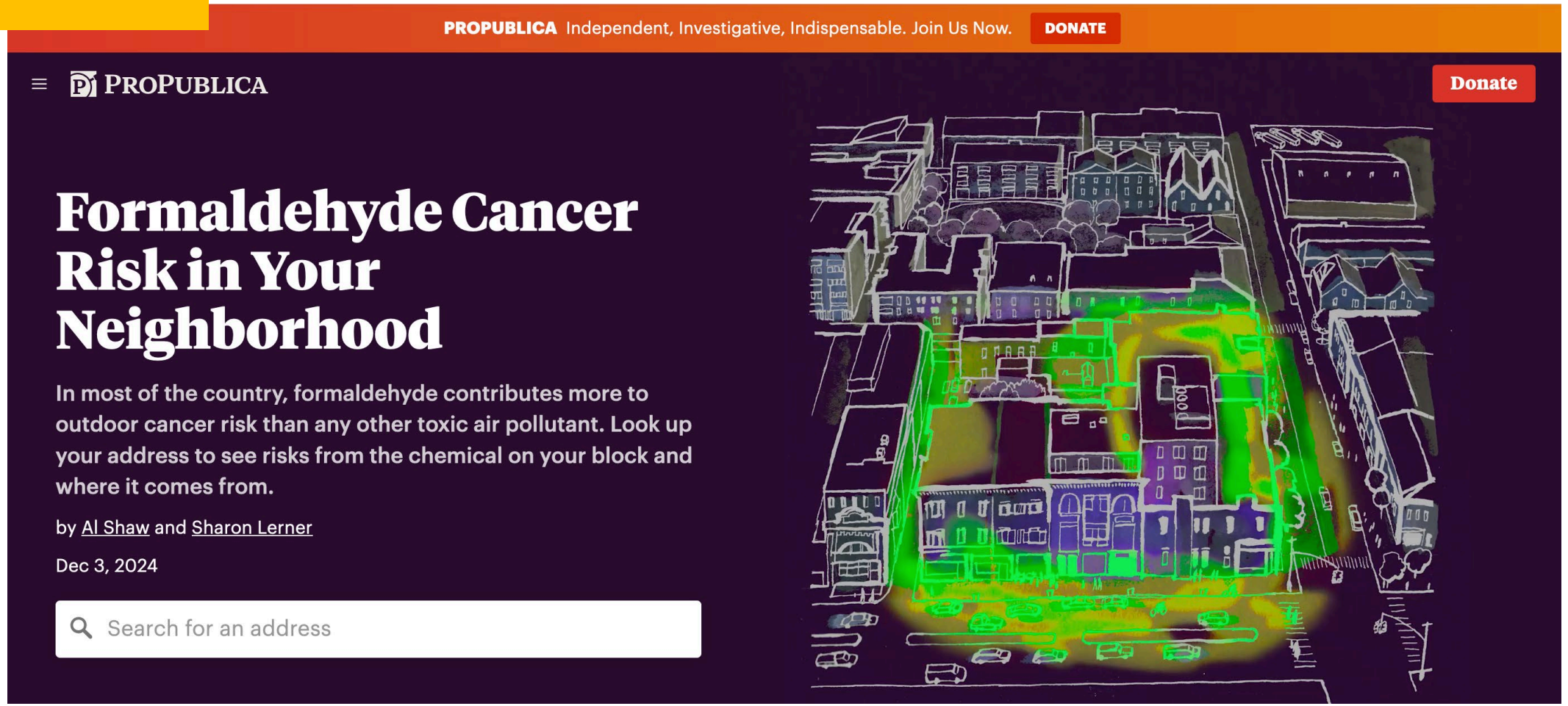
by Sharon Lerner and Al Shaw

Dec. 3, 5 a.m. EST



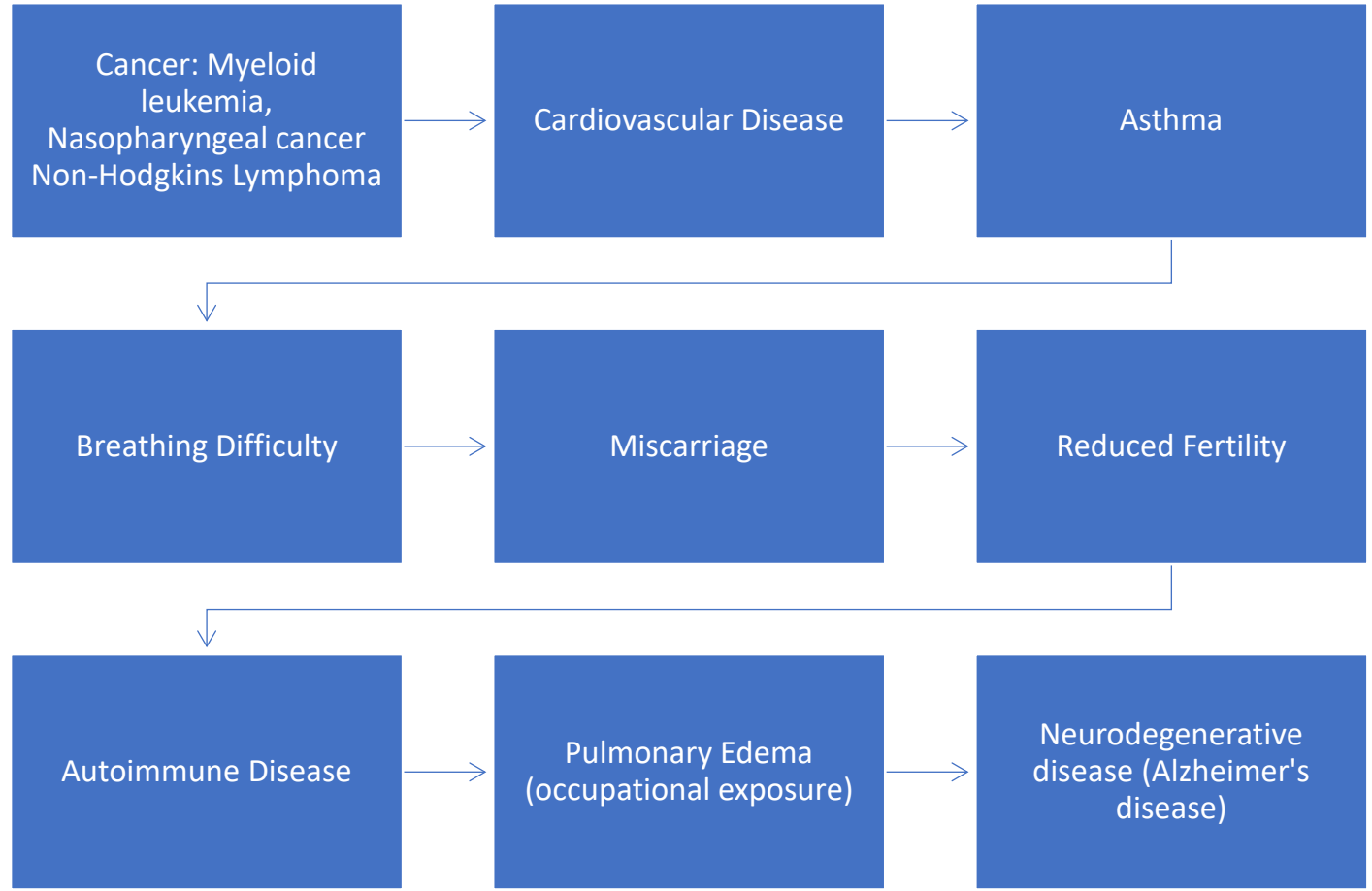
https://www.propublica.org/article/formaldehyde-epa-trump-public-health-danger

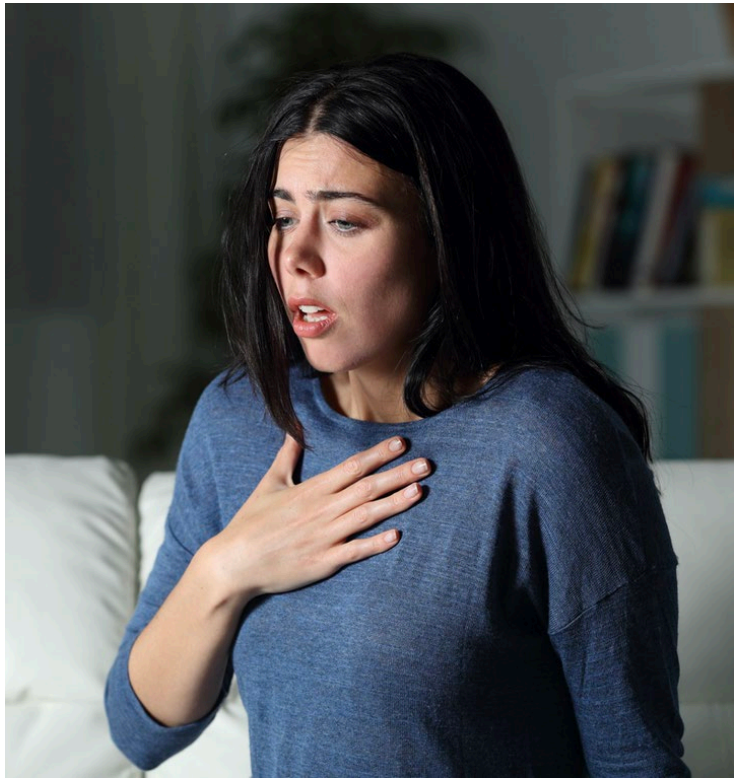
Formaldehyde in Outdoor Air



https://projects.propublica.org/formaldehyde-cancer-risk-map/

Formaldehyde Exposure-Related Conditions





PMID: 37236589/PMID: 19436030/PMID: 27802225

Formaldehyde is strongly linked to autoimmune disease

Open Journal of Rheumatology and Autoimmune Diseases, 2013, 3, 1-6
http://dx.doi.org/10.4236/ojra.2013.31001 Published Online February 2013 (http://www.scirp.org/journal/ojra)

Scientific Research

Case Report: Autoimmune Disease Triggered by Exposure to Hair Straightening Treatment Containing Formaldehyde

James Dahlgren[1], Rhett Roback[1], Maria Dominguez[1], Vera Byers[2], David Silver[3], Edward Faeder[4]

[1]James Dahlgren Medical, Santa Monica, USA; [2]Immunology Inc., Incline Village, USA; [3]UCLA David Geffen School of Medicine, Los Angeles, USA; [4]Srf Environmental & Health Management Inc., Diamond Bar, USA.
Email: jamesgdahlgren@yahoo.com

Formaldehyde is linked to autoimmune disease

"Brazilian hair treatments, containing high levels of formaldehyde (up to 11%), have become regularly used and have the potential to expose clients to toxic levels in excess of current regulatory standards. The product's label indicated that it was "Formaldehyde-Free" .We report on a patient who underwent a **single hair treatment** and subsequently developed a life-threatening autoimmune disease that necessitated plasmapheresis" which is an invasive procedure that removes plasma from the body in order to remove autoantibodies and the foreign material that may have initiated the autoimmune condition.

Open Journal of Rheumatology and Autoimmune Diseases, 2013, 3, 1-6
http://dx.doi.org/10.4236/ojra.2013.31001 Published Online February 2013 (http://www.scirp.org/journal/ojra)

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[1]James Dahlgren Medical, Santa Monica, USA; [2]Immunology Inc., Incline Village, USA; [3]UCLA David Geffen School of Medicine,

Formaldehyde is also directly toxic to the immune system

Formaldehyde suppresses the immune system and directly affects:

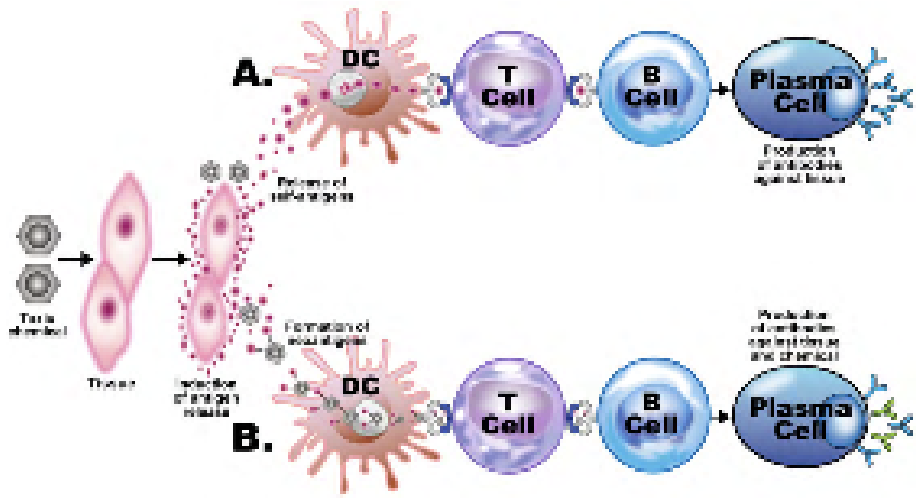
Natural Killer cells- which fight cancer and infections

Regulatory T cells- involved in preventing autoimmunity

CD8+ effector memory T cells- remember and respond to previously seen bacteria, viruses (like Covid), and other pathogens

PMID: 22767408

Solvents and Autoimmunity



pathophysiology

MDPI

Review

The Role of Exposomes in the Pathophysiology of Autoimmune Diseases I: Toxic Chemicals and Food

Aristo Vojdani [1,2,*] and Elroy Vojdani [3]

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2 Cyrex Laboratories, LLC, Phoenix, AZ 85034, USA
3 Regional Medical, 11620 Wilshire Blvd., Ste. 420, Los Angeles, CA 90025, USA; evojdani@gmail.com
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Abstract: Autoimmune diseases affect 5–8% of the world's population. It is now known that genetics play a relatively small part in the pathophysiology of autoimmune disorders in general, and that environmental factors have a greater role. In this review, we examine the role of the exposome, an individual's lifetime exposure to external and internal factors, in the pathophysiology of autoimmune diseases. The most common of these environmental factors are toxic chemicals, food/diet, and infections. Toxic chemicals are in our food, drink, common products, the air, and even the land we walk on. Toxic chemicals can directly damage self-tissue and cause the release of autoantigens, or can bind to human tissue antigens and form neoantigens, which can provoke autoimmune response leading to autoimmunity. Other types of autoimmune responses can also be induced by toxic chemicals through various effects at the cellular and biochemical levels. The food we eat every day commonly has colorants, preservatives, or packaging-related chemical contamination. The food itself may be antigenic for susceptible individuals. The most common mechanism for food-related autoimmunity is molecular mimicry, in which the food's molecular structure bears a similarity with the structure of one or more self tissues. The solution is to detect the trigger, remove it from the environment or diet, then repair the damage to the individual's body and health.

Keywords: exposome; autoimmune disease; environmental factors; toxic chemicals; food; molecular mimicry

check for updates

Citation: Vojdani, A.; Vojdani, E. The Role of Exposomes in the Pathophysiology of Autoimmune Diseases I: Toxic Chemicals and Food [illegible]. 2021, 28, 512–540. https://doi.org/10.3390/[illegible]

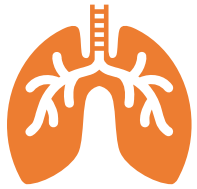
EPA Airtoxscreen Data- Formaldehyde

- According to ProPublica's analysis of the EPA's 2020 AirToxScreen data, some **320 million people** live in areas of the U.S. where the lifetime cancer risk from **outdoor exposure to formaldehyde is 10 times higher than the agency's goal for population cancer risk.**

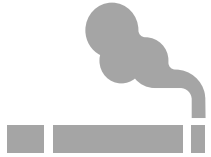
- In the Los Angeles/San Bernardino, California, area alone, some **7.2 million people** are exposed to formaldehyde at a **cancer risk level more than 20 times** higher than the EPA's goal.

- https://www.epa.gov/AirToxScreen/2020-airtoxscreen

EPA Airtoxscreen Report- Formaldehyde in the Average Home



The typical home has a formaldehyde level more than **3 times higher** than the EPA reference level for respiratory symptoms.

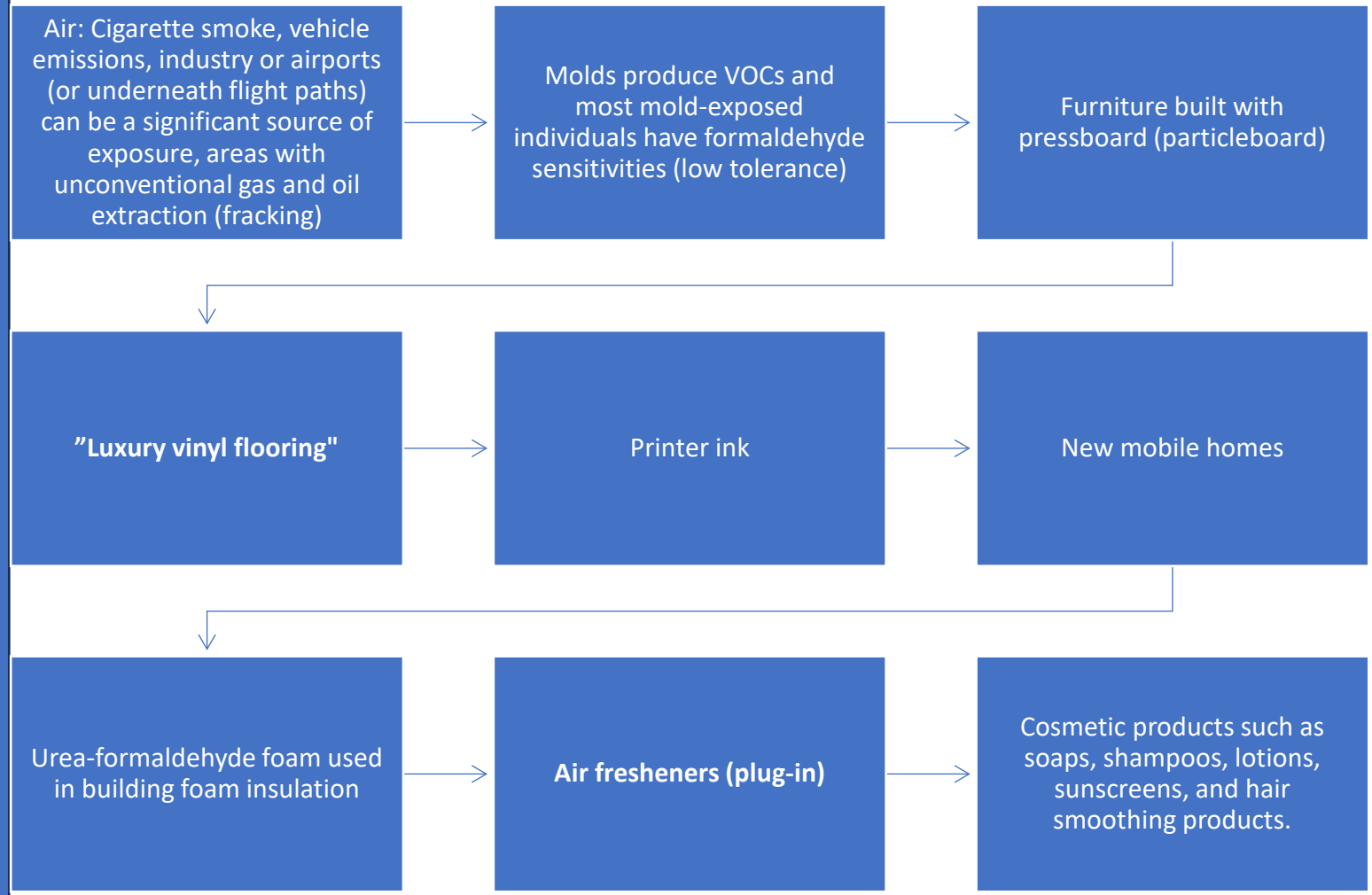


Over a lifetime of exposure to the formaldehyde in an average home, a person's risk of developing cancer is **more than 250 times the risk level that the Clean Air Act sets as a goal** (and this estimate didn't even include myeloid leukemia).

Among the products that can emit high levels of formaldehyde in these scenarios, according to the report, are automotive-care products like car waxes, along with crafting supplies, ink and toner, photographic supplies and fabrics, building materials, textiles and leather goods.

"there may not be a feasible way currently to reduce the average indoor level of formaldehyde to a point where there is no or almost no potential risk."

Where Does Formaldehyde Come From?



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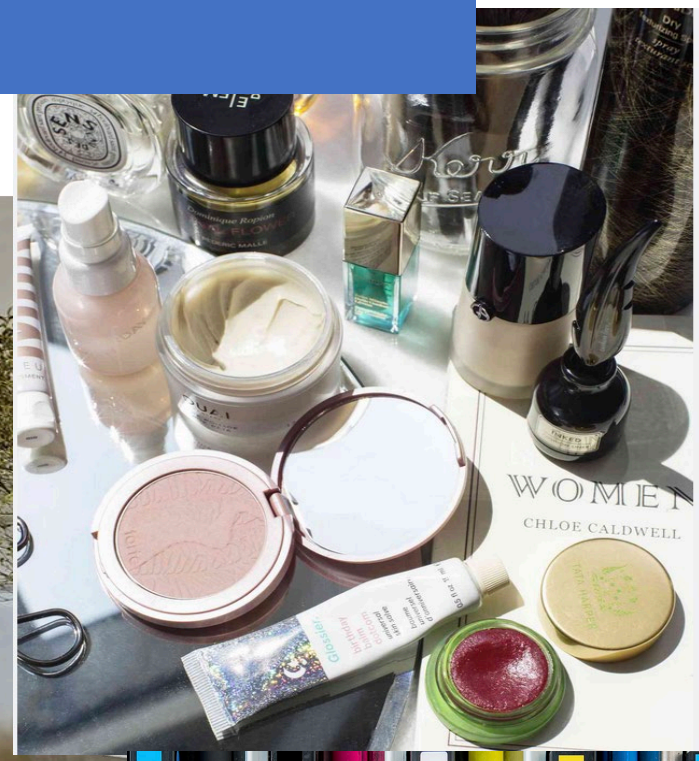
Sources of Formaldehyde in the Home

Mobile Home Tour



All *EHP* content is accessible to individuals with disabilities.
A fully accessible (Section 508–compliant) HTML version of this article is available at http://dx.doi.org/10.1289/ehp.122-A27.

Cooking Up Indoor Air Pollution

Emissions from Natural Gas Stoves

Natural gas cooking appliances, which are used by a third of U.S. households, can contribute to poor indoor air quality, especially when used without an exhaust hood.[1] Gas stoves emit nitrogen dioxide (NO_2), carbon monoxide (CO), and formaldehyde (HCHO), each of which can exacerbate various respiratory and other health ailments.[2,3,4] In a study reported in this issue of *EHP*, researchers from Lawrence Berkeley National Laboratory and Stanford University developed a simulation model to estimate gas stoves emissions and the exposures experienced by different household members.[5]

The model used a sample cohort representing Southern California households, of which more than half use natural gas to cook. The investigators obtained data on the homes and the occupants, including how often they cooked breakfast, lunch, and dinner. The team estimated air exchange rate (i.e., the rate at which outdoor air replaces indoor air), the amount of time people spent at home, and outdoor profiles for NO_2 and CO (indoor concentrations of these two pollutants are heavily influenced by outdoor levels, whereas HCHO concentrations typically depend on a variety of sources). They assumed one adult cooked in each home and that any children aged 0–5 years would be in close proximity to the adult while he or she was cooking.

Emissions from gas stove burners can reach potentially harmful levels if the cook does not use a venting hood.

© Food Photography by Eising/Corbis

Measuring Formaldehyde Exposure

EPA/CDC/NIH/OSHA/NIOSH/ATSDR consensus: no clinical lab measurements of formaldehyde or metabolites reflect recent exposure.

This is because the metabolic breakdown products of formaldehyde are eliminated quickly through urine and exhaled breath.

What's left? Home air evaluation!

Air Detection Monitors

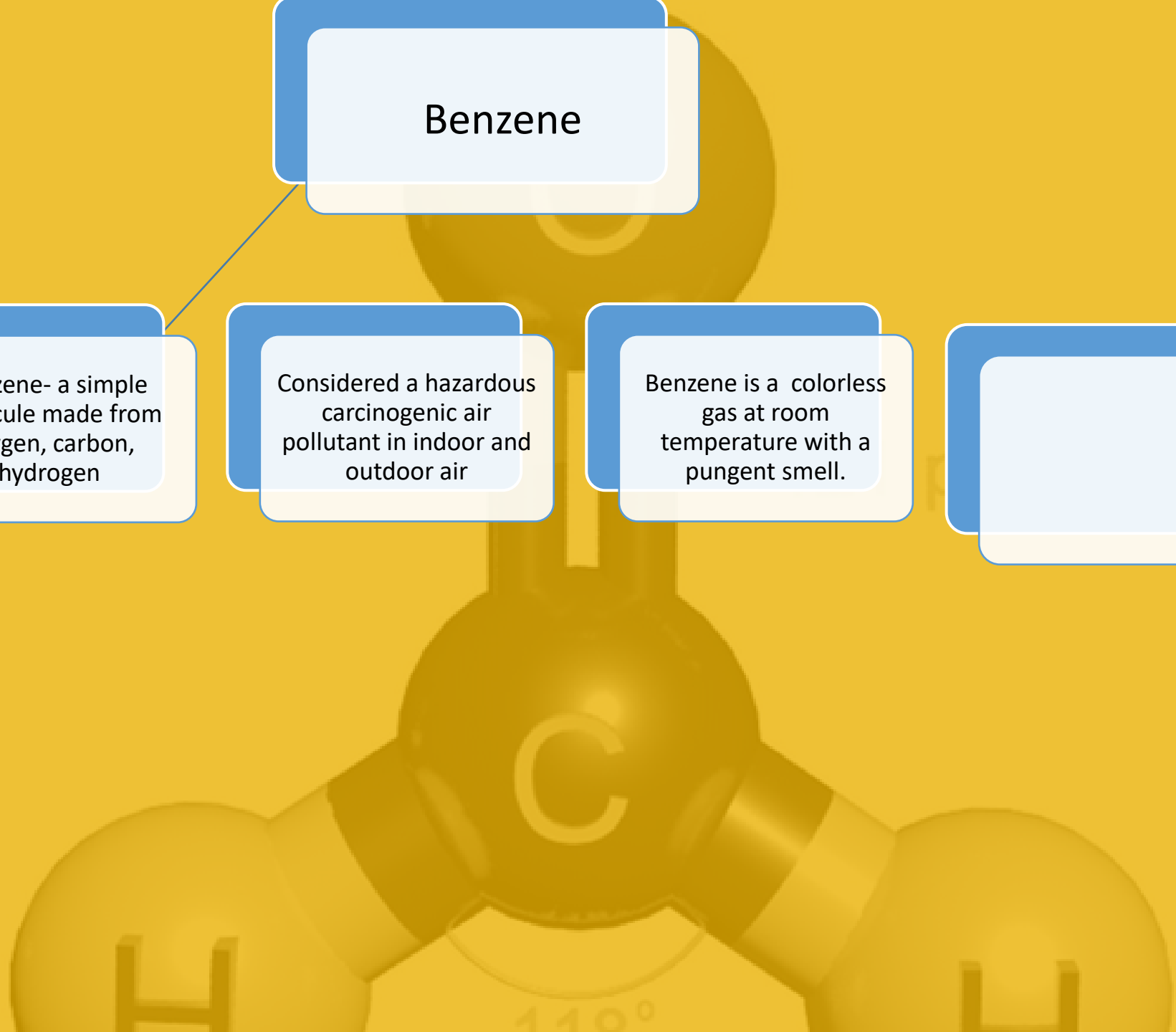
- Economical Model: AirKnight 9in1 Indoor Air Quality Monitor: $134.00
- Sensitivity down to < .1 ppm IMPORTANT! Sensitivity can be seen at .3 ppm or below.

Benzene

Benzene- a simple molecule made from oxygen, carbon, hydrogen

Considered a hazardous carcinogenic air pollutant in indoor and outdoor air

Benzene is a colorless gas at room temperature with a pungent smell.



Benzene- Hazardous Air Pollutant

- Benzene is in the top 20 for toxic chemicals in the United States, based on the amount in the environment.
- Hazardous air pollutants (HAPs), also known as toxic air pollutants are known or suspected to cause cancer or other serious health impacts.
- Exposures: urban air pollution, smoking, proximity to unconventional oil and gas wells (fracking), automobile exhaust

Benzene- 33% of cancer risk from gas and oil

According to the NEI, over 28,000 tons of benzene was emitted by oil and gas sources, accounting for 29% of the elevated cancer risk from oil and gas based on 2017 reported emissions.

Benzene is a constituent of raw natural gas, so leaks and deliberate releases of gas (venting) are the primary source of benzene pollution from the oil and gas industry.

The Air Toxics Screening Assessment (AirToxScreen)

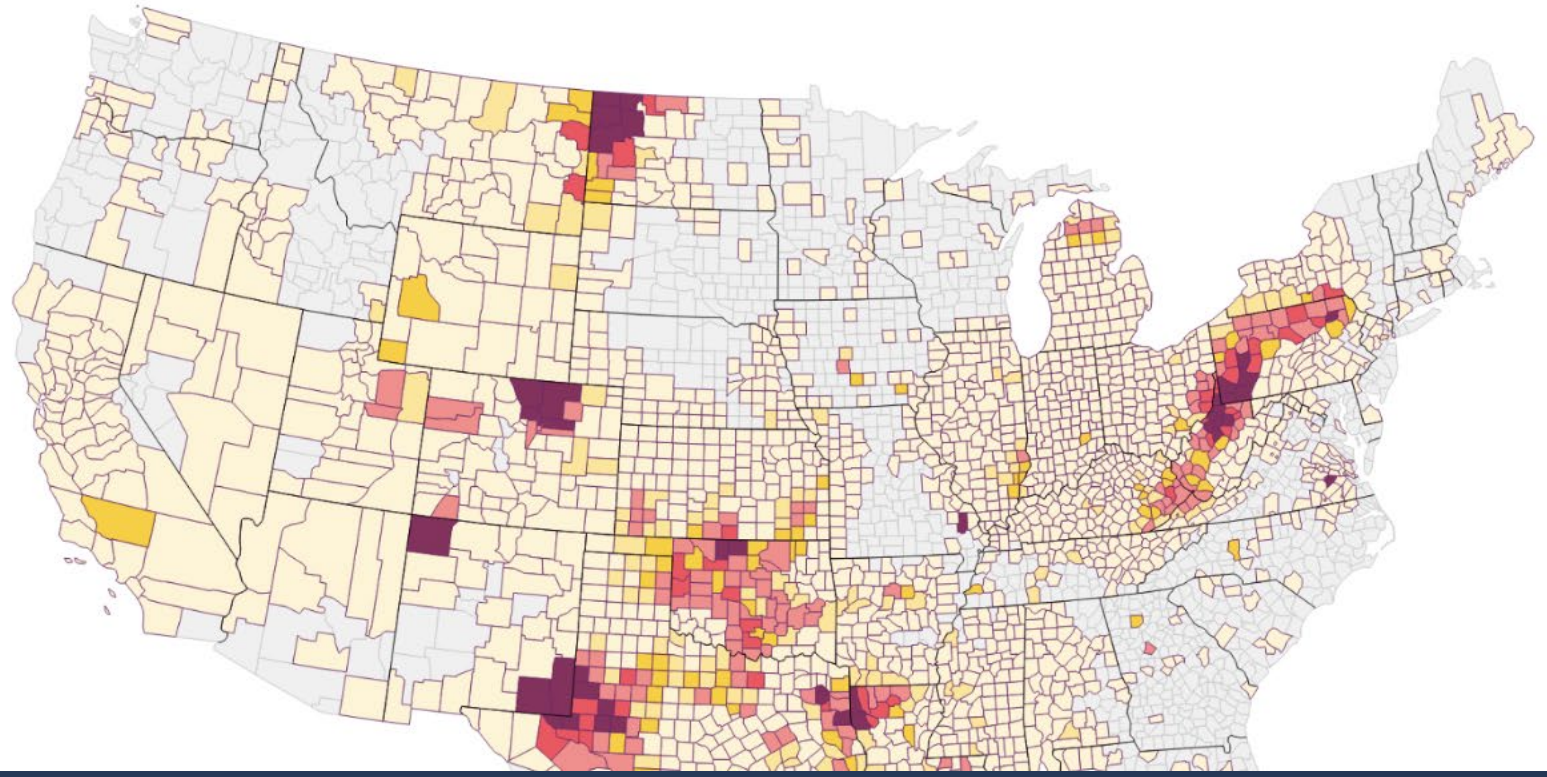
In March 2022, the U.S. Environmental Protection Agency (EPA) released the results of its Air Toxics Screening Assessment (AirToxScreen)—based on air pollution estimates collected through the National Emissions Inventory (NEI).

The purpose of AirToxScreen is to identify and prioritize air toxics, emission source types, and locations that are of greatest potential concern when looking at overall health risk in populations.

AirToxScreen estimates cancer risk that can result from toxic air emissions.

Map ES-1: National Map of Risk Level by County

The 236 counties that face cancer risk above EPA's 1-in-a-million level of concern are pink, red, or dark red.



In the last 8 years the total number of people living in counties with elevated cancer risk increased significantly: from 9 million to 14 million.

Greatest health risk: New Mexico, Texas, Colorado, Pennsylvania, Oklahoma, Louisiana, West Virginia, and North Dakota.

[Fossil Fumes 2022 Update Report A Public Health Analysis of Toxic Air Pollution from the Oil and Gas Industry Clean Air Task Force Sept 2022]

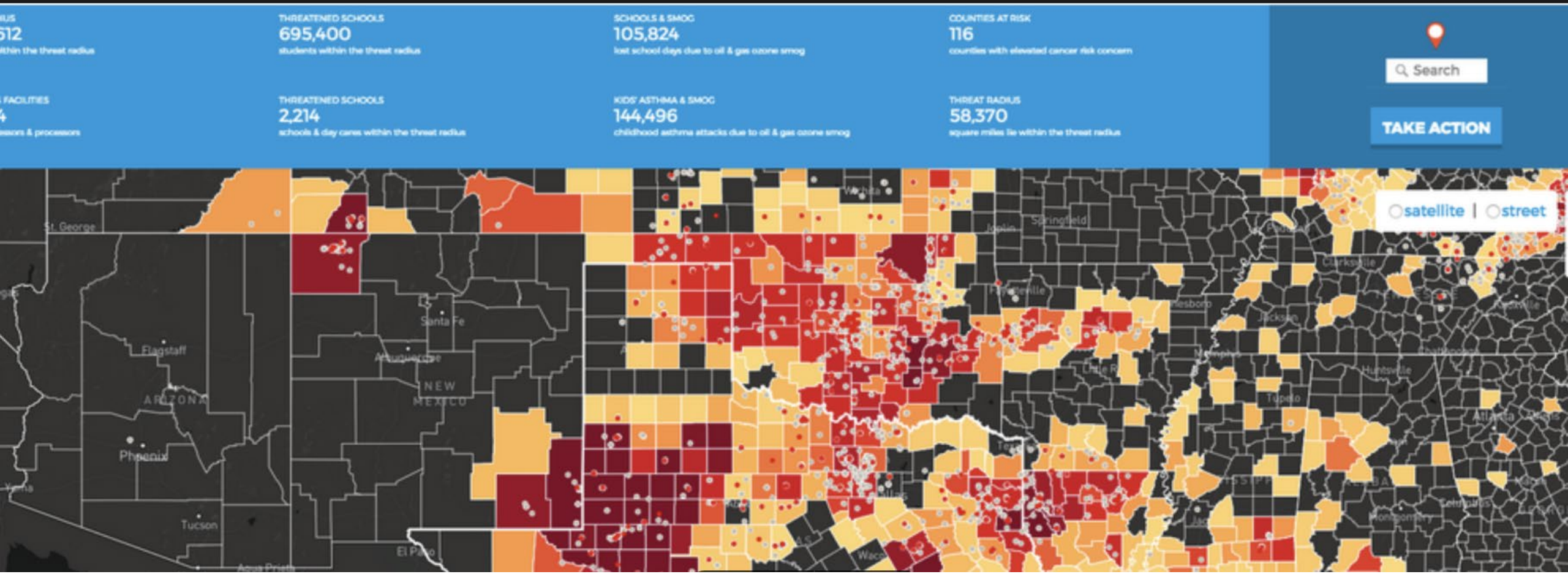


Table ES-1: List of Top 5 Oil and Gas Hazardous and Carcinogenic Air Pollutants in 2017 National Emissions Inventory

Note: These 5 pollutants account for 99% of the cancer risk from oil and gas facilities.

Hazardous Air Pollutant	Tons Emitted Per Year from Oil & Gas Industry	Health Impacts
Formaldehyde	37,826	Cancer and respiratory symptoms
Benzene	28,021	Cancer, anemia, brain damage and birth defects, and respiratory tract irritation
Acetaldehyde	5,491	Cancer and respiratory irritant
Ethylbenzene	2,200	Respiratory irritant, and increased risk of blood and neurological disorders
1-3 Butadiene	650	Increases risk of cancer and cardiovascular diseases

Table ES-2: List of High-Risk Counties

Counties with Cancer Risk Above 1 in 100,000					
CO, Weld*	NM, Eddy*	WV, Doddridge			
Counties with Cancer Risk Above 1 in 250,000					
CO, Adams*	LA, De Soto	NM, Lea*	PA, Greene	TX, Karnes	TX, Yoakum
CO, Boulder*	LA, Lafourche	NM, San Juan*	PA, Washington	TX, Loving	VA, Prince George
CO, Broomfield*	ND, McKenzie	OK, Alfalfa	PA, Westmoreland	TX, Martin	WV, Ritchie
CO, Larimer*	ND, Mountrail	OK, Grant	PA, Wyoming	TX, Panola	WV, Tyler
LA, Assumption	ND, Williams	PA, Fayette	TX, DeWitt	TX, Upshur	WV, Wetzel

Memory Foam: mattresses/pillows/topper

"Exposure levels estimated for sleeping child/infant indicate that the bed can be a significant contributor to VOC exposure, yielding concerning exposure levels for a few compounds."

61 Petroleum-based compounds. Almost all are Solvents/VOCs

Benzenes (VOCs)	Hexanes	Aldehydes & alcohols	Ethers & esters	Polycyclic Aromatic Hydrocarbons PAHs	Chlorinated organic hydrocarbons

Environ. Sci. Technol. 2019, 53, 15, 9171-9180
https://www.ewg.org/healthyhomeguide/mattresses/

Carpet = hexane + styrene + benzene + toluene

- Synthetic carpets are made from nylon fibers with a polypropylene backing.
- Styrene and 4-phenylcyclohexane (4-PC), both of which come from the latex backing are used on 95 percent of carpets.
- The “new carpet” aroma is the odor of 4-PC off-gassing, which is an eye– and respiratory-tract irritant that may also affect the central nervous system.
- **The adhesive used to affix the carpet to the floor typically contains benzene and toluene, some of the most harmful VOCs**

Solvents: BTEX (benzene, toluene, ethylbenzene, xylene)

Exposures: urban air pollution, smoking, proximity to unconventional oil and gas wells (fracking), automobile exhaust

Conditions: asthma, upper respiratory problems, infertility (women), increased risk of spontaneous abortion, altered menstrual cycles and altered levels of FSH, LH and testosterone in men. BTEX contributes ground level ozone which predisposes adults and children to COPD. Prolonged daily or nightly exposure related to headaches.

WHEN THE WIND BLOWS

TRACKING TOXIC CHEMICALS IN GAS FIELDS AND IMPACTED COMMUNITIES

https://comingcleaninc.org/assets/media/documents/When%20the%20Wind%20Blows.pdf

TABLE 4

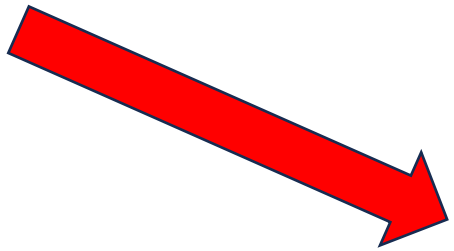
Levels of Chemicals in Urine Samples of Pavillion Residents, Wyoming (August 2014), µg/g Creatinine, Selected Findings **n=11 individuals from Pavillion community** 27 urine samples and 27 blood samples

Chemical Metabolite	Parent Chemical	Median Level	Maximum Level
Hippuric acid	Toluene, cinnamaldehyde	322,959	1,197,549
Mandelic acid	Ethylbenzene, styrene	215	2466
4-Methylhippuric acid	Xylene	92	1395
2-Methylhippuric acid	Xylene	81	631
3-Methylhippuric acid	Xylene	99	643
Phenylglyoxylic acid	Ethylbenzene, styrene	53	411
trans, transmuconic acid	Benzene	369	2046
PMA (N-Acetyl-S-(phenyl)-L-cysteine)	Benzene	BDL	1.0
MHBMA (N-Acetyl-S-(2-hydroxy-3-butenyl)-l-cysteine)	1,3-butadiane	BDL	0.8
HEMA (N-Acetyl-S-(2-hydroxyethyl)-l-cysteine)	Acrylonitrile, vinyl chloride	1.3	3.6
CNEMA/CYMA (N-Acetyl-S-(2-cyanoethyl)-L-cysteine)	Acrylonitrile	1.3	31.9
3-HPMA (N-Acetyl-S-(3-hydroxypropyl)-l-cysteine)	Acrolein	388	7058
2-HPMA (N-Acetyl-S-(2-hydroxypropyl)-l-cysteine)	Propylene oxide	35	162
AAMA (N-Acetyl-S-(2-carbamoylethyl)-l-cysteine)	Acrylamide	99	199
HPMMA (N-Acetyl-S-(3-hydroxypropyl-1-methyl)-lcysteine)	Crotonaldehyde	235	630

YELLOW = Level that exceeds median in urine of general population

ORANGE = Level that exceeds 95th percentile in urine of general population

µg/g = micrograms/gram



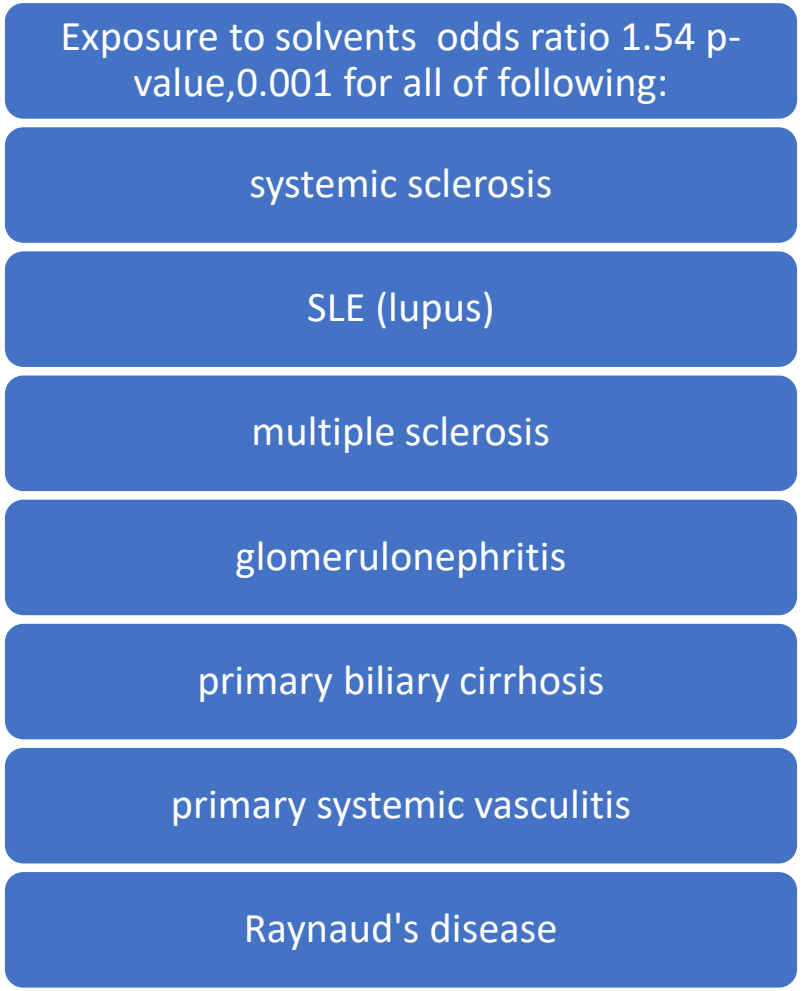
Benzene-" There is probably no safe level of exposure to benzene"

Hematotoxic: decrease in T cells, B cells, low platelets, WBC, pancytopenia and ↓ hemoglobin	Leukemias (highest for myeloid and monocytic leukemias)	Hypothalamic- pituitary-adrenocortical activity stimulated, cortisone level↑	Altered thyroid labs: lowered T3 hormone, elevated T4
In Texas, census tracts with the highest benzene levels had elevated rates of all leukemias	Dysmenorrhea, Menstrual disorders	Behavioral, motor and cognitive changes	Sperm motility, DNA damage

Annu. Rev. Public Health 2010. 31:133–48./ *Toxicol Appl Pharmacol*. 2014;276(2):83-94.

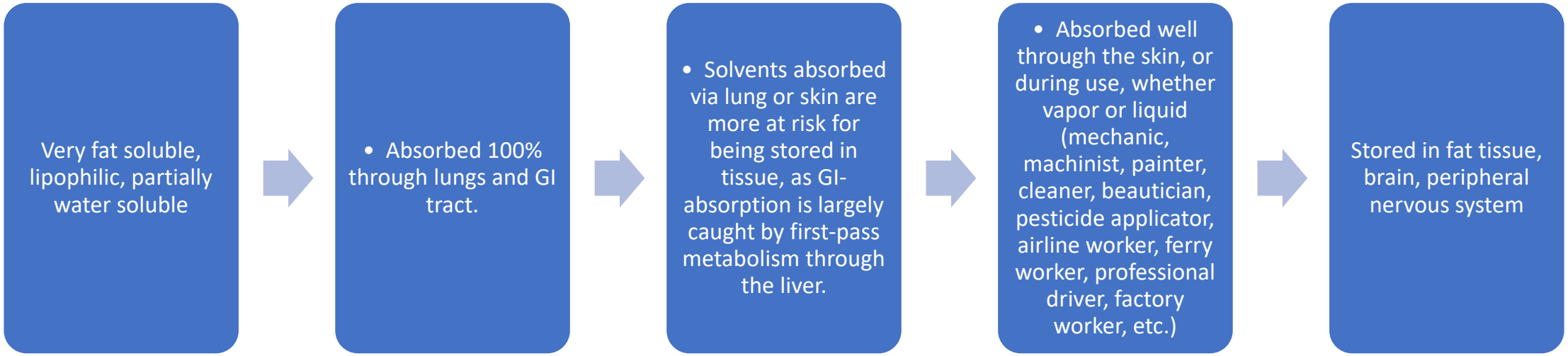
-Martyn T. Smith Superfund Research Program, Division of Environmental Health Sciences, School of Public Health, University of California, Berkeley, California

Autoimmune disease: TCE, benzene, toluene



PLoS ONE 7(12): e51506. doi:10.1371/journal.pone.0051506

Solvent Metabolism



Solvents end up in our fat tissue

Since 1976, the Environmental Protection agency (EPA) has been engaged in the *National Human Adipose Tissue Survey* (NHATS)

In this study, adipose samples are taken from cadavers and elective surgeries from all regions of the country and the levels of toxins are measured

Five of the chemicals--*OCDD* (a dioxin), *styrene, 1,4-dichlorobenzene, xylene*, and *ethylphenol*--were found in 100% of all samples.

Another nine chemicals--***benzene, toluene, chlorobenzene, ethylbenzene, DDE***, **three *dioxins* and one *furan*--were found in 91-98% of all samples.**

http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55204#Download

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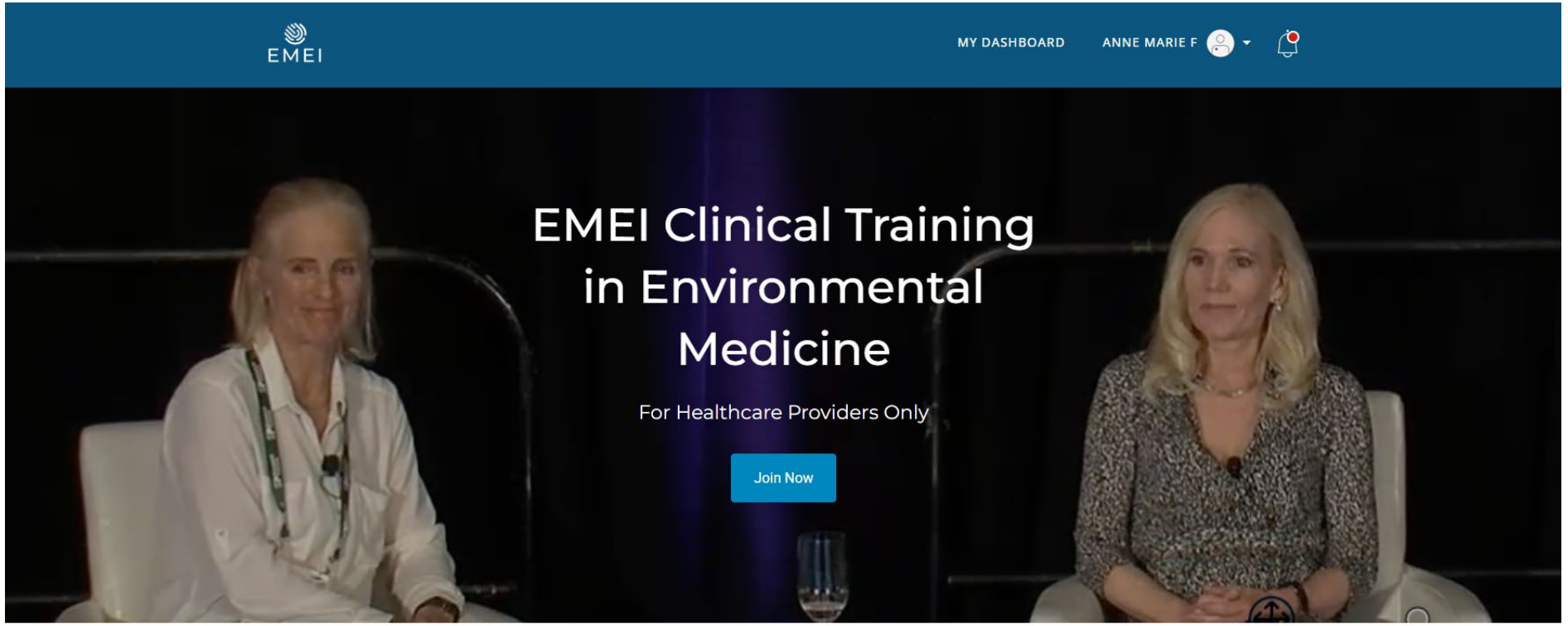

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