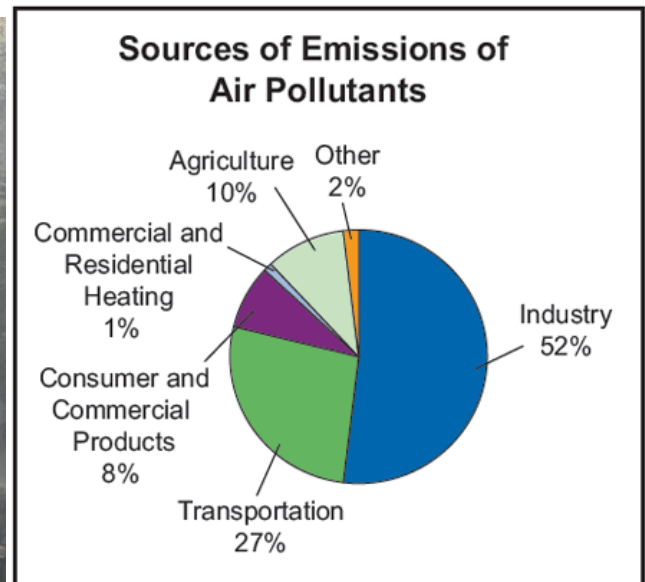
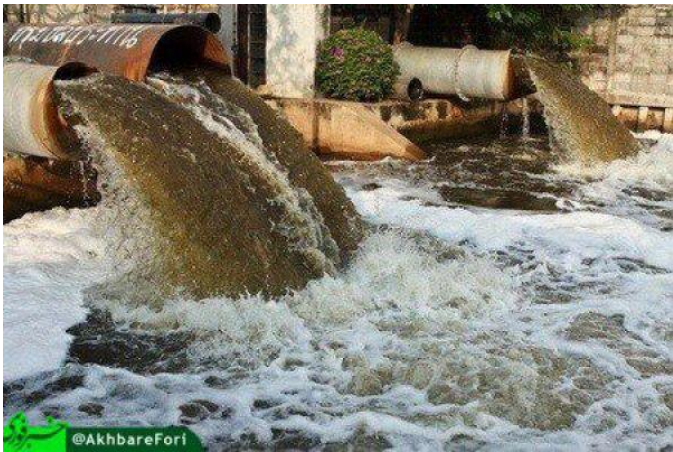


Pollution as Devolution of the Species?

An eBook

By Mauro Di Pasquale, B.Sc. (Hons), M.D.

Updated: February 18, 2021



The Green Solution – Slash and Burn forests for crops

I WANT AND POLLUTE THEREFORE I EXIST

Trillions for Your Thoughts

A poem by Mauro – for another poem go to the end of this article before the references.

The rain that pours down
Is not what it seems
It's not wet but dry
Not like in our dreams

You can look up and see
The sky is bright and blue
But you're told that what you seek
Is no longer looking for you

Finding what we're searching for
Something to believe
If we look where we should look
We'll be told that we must leave

What we're told is disguised
As what we should believe
Leaving us forsaken
Because we're being deceived

The answer is so simple
Or at least it's what they say
Just tune into the confusion
You'll surely find their way

Exciting discoveries uncovered
It's like Hollywood every day
Promising major changes
But they fail along the way

They revel in our domain
Never leaving it to chance
Consuming is their game
It's the only way to dance

A penny for your thoughts
Trillions for your greed
Never ending profit is only fair
Damn the rest the rich don't care

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Introduction

The common consensus, and one fostered by Darwin, is that evolution is progressive in that it is directed to improve the species both physically and mentally. However, considering the way that the world is progressing, devolution of the species may be just as probable.

Both evolution and devolution can be viewed as simply reactions to environmental change. However, if the changes in the environment are extreme, then it can negatively impact on the viability of the species by allowing humanity to devolve into a prior state of social and personal development. Evolutionary changes on the organism of humanity is variable and genetic/epigenetic adjustments can tend towards both positive (evolution) and negative (devolution) changes to our makeup.

This update (or eBook as you prefer), further describes the increasing pervasiveness and negative impact of pollution on our planet and every living entity. Because of its importance to humanity's present and future, I feel compelled to update this information from time to time for both my purposes to keep the information up to date for my readers.

While this article is about pollution and its many adverse effects on morbidity (both physical and mental) and mortality, it can even be argued that modern medicine, by allowing deleterious mutations to survive and be propagated to future generations, can lead to devolution rather than beneficial progressive evolution. As well, devolution is also a possibility if the chaos caused by world-wide malaise continues. But those discussions are for a future articles.

Unfortunately, there is a significant disconnect between perception and reality about pollution, global warming and the dire consequences that are coming our way. This disconnect also exists about the present pandemic and others that we will certainly be facing in the near future.

The bottom line is that we've killing our planet and ourselves and if we don't react appropriately, it will be too late to save us. It's time to make all nations accountable to their levels of pollution and enforce economic penalties on non-compliant nations and even uncompliant people.

Pollution and Covid-19 Pandemic

The Lancet Commission on pollution and health published in October, 2017 stated that **“pollution is the largest environmental cause of disease and death in the world today.”**¹ For more information go to [http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736\(17\)32345-0.pdf](http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(17)32345-0.pdf).

The current coronavirus pandemic is partly being fueled by pollution since pollution increases inflammation in our bodies, lowers our resistance and makes us more susceptible to serious and potentially fatal infestation by many pathogens including bacteria, fungi, and viruses. There are many research papers and articles that show the association of pollution and the present Covid-19 pandemic.

The following references below, in somewhat random order, are just a small sampling showing the association of pollution as a modifiable factor contributing to differential SARS-CoV-2 spread.²³⁴⁵⁶⁷⁸⁹¹⁰¹¹¹²¹³¹⁴ Most of these reference are also presently available in full text. For example,

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7198142/pdf/main.pdf>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7395654/pdf/main.pdf>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7711169/pdf/main.pdf>.

Pollution is also responsible for climate change which in turn may potentiate viral infections as wildlife habitats are being destroyed meaning that wildlife and their pathogens are able to infect us either directly or through increased contacts with domestic animals that are closer to wildlife than ever before.

As an example, a recent study suggests that shifts in global bat diversity suggest a possible role of climate change in the emergence of SARS-CoV-1 and SARS-CoV-2.¹⁵

COVID and Air Pollution: Cardio Societies Call for Urgent Action

Megan Brooks

January 29, 2021

Editor's note: Find the latest COVID-19 news and guidance in Medscape's [Coronavirus Resource Center](#).

Four leading cardiovascular organizations — the World Heart Federation (WHF), American College of Cardiology (ACC), American Heart Association (AHA), and European Society of Cardiology (ESC) — have jointly called for the medical community and health authorities to take action to mitigate the harmful effects of air pollution on health.

The statement was published simultaneously January 28 in the [Journal of the American College of Cardiology](#), [Circulation](#), the [European Heart Journal](#), and [Global Heart](#).

Air pollution has been shown to increase the risk of cardiovascular disease (CVD), including [myocardial infarction](#) and [stroke](#), as well as diabetes and respiratory diseases.

Recent evidence also suggests air pollution increases the risk of more severe COVID-19 and death from COVID-19, they note. Air pollution may also increase the risk of novel coronavirus transmission due to its impact on more frequent coughing.

This dangerous "triple threat" of air pollution, COVID-19, and CVD needs urgent action, the statement authors say.

"Even before the COVID-19 pandemic, air pollution was an issue of growing concern due to its impact on people's health, although it was frequently overlooked as a risk factor for cardiovascular disease," Michael Brauer, ScD, chair of the WHF air pollution expert group and co-author of the statement, said in a news release.

"COVID-19 has brought a new, deadly factor to the equation, and the time has come for the health community to speak up and take action," said Brauer.

The authors call for structural actions to reduce emissions of air pollutants and harmful exposure. They also call on healthcare providers to:

- Advocate for air pollution mitigation as a health measure, further research on air quality and its impact on CVD, and interventions to reduce air pollution and its effect on noncommunicable disease.
- Provide patients with personal measures to reduce exposure, such as room air filtration systems.
- Integrate air pollution into disease management approaches, for example through the use of air quality indices.
- Participate in the development of guidelines on air pollution and CVD.
- Support ministries of environment, energy, and transportation in their mitigation efforts.
- Work to educate and raise awareness on the cardiovascular benefits of clean air.
- Collaborate with senior decision-makers in national, regional, and global governmental institutions to make air pollution-related heart disease a priority.

"Clinicians have a responsibility to educate their patients, their colleagues and their communities at large on the connection between air pollution and cardiovascular disease risk," Richard Kovacs, MD, senior author of the joint statement and immediate past-president of the ACC, said in the news release.

"By advocating for recognition of air pollution as a health factor, working individually with our patients to reduce exposure and associated risks, and integrating air pollution into broader disease management approaches, the healthcare community can provide support for larger pollution mitigation efforts," said Kovacs.

"Poor air quality can harm heart and brain health, with a disproportionate impact on low-income and poor communities located near sources of air pollution," added co-author Robert Harrington, MD, immediate past-president of the AHA.

"We must address this problem as a global community to equitably reduce exposure to air pollution and reverse the health harms of poor air quality for all," said Harrington.

ESC President Stephan Achenbach, MD, noted that air pollution is "one of the most underestimated causes of heart disease and stroke."

"Air pollution needs to be recognized as a major modifiable risk factor in the prevention and management of cardiovascular disease, and measures to reduce its detrimental short-term and long-term influence on cardiovascular health, potentially over generations, are urgently required," said Achenbach.

Kovacs reports other from Cook Research Inc, other from Clintrex, personal fees from Eli Lilly, grants and personal fees from Gen InCode, personal fees from Prilenia, other from Pfizer, outside the submitted work. Brauer, Harrington, and Achenbach have disclosed no relevant financial relationships.

J Am Coll Cardiol. Published online January 28, 2021. [Full text](#)

Circulation. Published online January 28, 2021. [Abstract](#)

Eur Heart J. 2021;ehaa1025. [Abstract](#)

Global Heart. 2021;16:8. [Full text](#)

For more from *TheHeart.org*, follow us on [Twitter](#) and [Facebook](#).

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Environ Res. . 2020 Dec 3;193:110556. doi: 10.1016/j.envres.2020.110556. Online ahead of print.

Air quality and COVID-19 adverse outcomes: Divergent views and experimental findings

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Abstract

Background: The questioned link between air pollution and coronavirus disease 2019 (COVID-19) spreading or related mortality represents a hot topic that has immediately been regarded in the light of divergent views. A first "school of thought" advocates that what matters are only standard epidemiological variables (i.e. frequency of interactions in proportion of the viral charge). A second school of thought argues that co-factors such as quality of air play an important role too.

Methods: We analyzed available literature concerning the link between air quality, as measured by different pollutants and a number of COVID-19 outcomes, such as number of positive cases, deaths, and excess mortality rates. We reviewed several studies conducted worldwide and discussing many different methodological approaches aimed at investigating causality associations.

Results: Our paper reviewed the most recent empirical researches documenting the existence of a huge evidence produced worldwide concerning the role played by air pollution on health in general and on COVID-19 outcomes in particular. These results support both research hypotheses, i.e. long-term exposure effects and short-term consequences (including the hypothesis of particulate matter acting as viral "carrier") according to the two schools of thought, respectively.

Conclusions: The link between air pollution and COVID-19 outcomes is strong and robust as resulting from many different research methodologies. Policy implications should be drawn from a "rational" assessment of these findings as "not taking any action" represents an action itself.

Evidence-Based Considerations Exploring Relations between SARS-CoV-2 Pandemic and Air Pollution: Involvement of PM_{2.5}-Mediated Up-Regulation of the Viral Receptor ACE-2

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Full Text available at: <https://www.mdpi.com/1660-4601/17/15/5573/htm>.

Abstract

The COVID-19/SARS-CoV-2 pandemic struck health, social and economic systems worldwide, and represents an open challenge for scientists -coping with the high inter-individual variability of COVID-19, and for policy makers -coping with the responsibility to understand environmental factors affecting its severity across different geographical areas. Air pollution has been warned of as a modifiable factor contributing to differential SARS-CoV-2 spread but the biological mechanisms underlying the phenomenon are still unknown. Air quality and COVID-19 epidemiological data from 110 Italian provinces were studied by correlation analysis, to evaluate the association between particulate matter (PM)_{2.5} concentrations and incidence, mortality rate and case fatality risk of COVID-19 in the period 20 February-31 March 2020. Bioinformatic analysis of the DNA sequence encoding the SARS-CoV-2 cell receptor angiotensin-converting enzyme 2 (ACE-2) was performed to identify consensus motifs for transcription factors mediating cellular response to pollutant insult. Positive correlations between PM_{2.5} levels and the incidence ($r = 0.67$, $p < 0.0001$), the mortality rate ($r = 0.65$, $p < 0.0001$) and the case fatality rate ($r = 0.7$, $p < 0.0001$) of COVID-19 were found. The bioinformatic analysis of the ACE-2 gene identified nine putative consensus motifs for the aryl hydrocarbon receptor (AHR). Our results confirm the supposed link between air pollution and the rate and outcome of SARS-CoV-2 infection and support the hypothesis that pollution-induced over-expression of ACE-2 on human airways may favor SARS-CoV-2 infectivity.

Pollution has also been implicated in the dangerous trend in antibiotic resistance, which has become a major health problem. The usual reason is the overuse of antibiotics in both humans and animals, allowing genetic and epigenetic changes to nullify the antibiotics antimicrobial effects.¹⁶¹⁷¹⁸¹⁹²⁰²¹²²²³²⁴²⁵ However, studies also implicate pollution in antibiotic resistance.²⁶²⁷²⁸

Pollution, Global Warming, and Climate Change

Pollution, climate change and global warming are intertwined and result in palpable threats to us and our planet. Limiting global warming is urgently needed to avoid disastrous consequences that include excessive heat, drought, extreme weather and sea level rise.

Limiting global warming to an increase of two degrees Celsius is currently the goal that has a common consensus as how to mitigate the consequence of global warming.²⁹ However, current agreements, such as the Paris Agreement, are first of all not enough to achieve this goal and secondly not being followed by most nations anyway³⁰³¹ (see below on my comments as far as commitments being mainly lip service).

A recent study looked at what would be required to meet emissions cuts that would result in the goal of limiting global warming to 2 degrees centigrade, considered a threshold for climate stability and climate-related risks.³² The study found that emissions reductions of 80% more ambitious than those in the Paris Agreement would be needed to stay within the 2 degrees threshold.

The video - What Happens if Earth Gets 2°C Warmer? Is worth watching.

https://www.youtube.com/watch?v=KeX7x5NtNFg&feature=emb_logo

As pollution increases, the earth gets warmer. The questions and models in this video address what will happen to us and the planet earth as the earth warms and why we must address climate change and pollution to assure that catastrophic warming doesn't happen.

The video explains how the 2 degrees Celsius threshold in the average global temperature is important and how it was arrived at. It also explains why the earth is warming. It also explains what the threshold increase could mean for us including disasters to land, water, weather, species existence and human life.

The video also explains how air pollution, primarily the increase in CO2 and other heat trapping gases, resulting mainly as a result of burning fossil fuels, causes global warming, which in turn will result in catastrophic events if it's allowed to continue.

There's no doubt in my mind that global warming is due to vehicle, industrial, agricultural, personal and other contributions to air pollution. For example, The World Health Organization (WHO) recently ranked air pollution as the major environmental cause of premature death -

<https://www.who.int/teams/environment-climate-change-and-health/air-quality-and-health/ambient-air-pollution>.

And on December 15, 2020. For the first time, in a landmark judgement, a UK coroner has said that air pollution was the cause of death of a 9-year-old London schoolgirl Ella Kissi-Debrah. This official ruling, and its legal and moral consequences, hopefully will spur countries to seriously tackle the task of significantly decreasing the many deadly sources of pollution in the world and decreasing the threat of global warming.

However, while goals, limits, and national and international agendas have been put in place, it seems to me that most of it is just lip service and that nothing significant has been done to control pollution and deal with the threats of global warming.

Perhaps more persuasion is needed, and it certainly is coming as inaction further threatens us and our world.³³

Deforestation

Another example of that is the decimation of forest throughout the world and especially the extensive Amazon rainforest in South America, the largest rainforest in the world that represents over half of the world's rainforests.

More and more forests are cut down and the wood usually burned (which by itself increase pollution and climate change) by using the slash and burn method, to make room mainly for cattle but also for developments, agriculture, and other domesticated animals.

There are several ramifications of the deforestation including the real possibility that as the forest area declines, there will come a time when it will not be able to sustain itself.

One of the main ramification of deforestation is the decrease in the world's trees since trees buffer the pollution that drives climate change.³⁴ One of the authors of this study stated "forests consume large amounts of carbon dioxide globally. Without that, more carbon dioxide would go into the air and build up in the atmosphere even more than it already is, which could exacerbate climate change. Our work shows yet another important reason to preserve and maintain our forests and keep them healthy."

The Green Revolution?

As I mentioned above, while there's a lot of fuss and noise on how we're all concerned about pollution and climate change, there is very little being done to reverse the trend. Most of the promises and excuses by those who could make a difference is mostly just enough talk to assuage us, but little action.

Take the so-called green revolution, which is touted to decrease pollution, provide clean energy, and cure all that's going wrong in our world. Another pack of lies by those who profit the most from fooling us all into believing they really care. Actually, they do care but it's about their bottom line and all they say is fueled by greed.

You likely know my views from other articles about the capitalistic elite and the non-capitalistic elite, which boils down to them all bowing before the altar of the almighty dollar. Putting into motion effective measures to reduce pollution and other factors contributing to climate change doesn't sit well with their bottom line.

Flame Retardants

But day by day more evidence accumulates on how increasingly polluted this planet is and how our exposure is continually escalating. For example, a recent study found that our vehicles can be a significant source of pollution but not in the usual way we think of vehicle pollution. It's been known that chemicals originating from the inside of vehicles and those introduced (for example smoking cigarettes and e-cigs) are significant sources of body pollution.³⁵

While there are many forms of pollution originating from the inside of a vehicle one of the new kids on the block is the flame retardant used mostly in the foam of vehicle seats. The study published this year found that the longer you drive, even if it's only a week, the more you're exposed to this carcinogenic chemical flame retardant – see abstract below and other abstracts of interest.³⁶

But flame retardants are ubiquitous, and most people aren't aware that they're all around us. They are used on clothes, furniture, almost all construction material including the increasingly popular insulated concrete forms, electronics, transportation products in all forms of transportation besides vehicles as mentioned above.

The problem with flame retardants and similar compounds as found in insulation, urethane and polyurethane solids and foams, is that they persist for long periods of time,

Pollution Escalation and Other Information

Other Sources of Pollution

There have been several recent studies confirming the chronic long-term effects of water and soil pollution, especially the effects of how pervasive and long-term pollution by these toxic compounds affects us including development in children.³⁷³⁸³⁹⁴⁰⁴¹⁴²⁴³ Water pollution has a dire toxic effect on all living entities.

Red Tide

One form of water pollution results in the occurrence of toxic algal blooms commonly called red tide, mainly in coastal waters around the world. Red tide results from excessive phytoplankton proliferate secondary to nutrient or sewage flow into seawater and results in damage to the fisheries industry and the marine ecosystem.⁴⁴⁴⁵

Gyms, Indoor, and Other Spaces

Gyms and indoor exercise facilities, while offering needed resources to improve health can actually be counterproductive as they present significant levels of pollutants. The same goes for all under vented indoor spaces, especially those that use disinfectants which because of the pandemic includes all public places and even all popular modes of transportation such as airplanes, trains, and busses.

Finewax Z, Pagonis D, Claflin MS, Handschy AV, Brown WL, Jenks O, Nault BA, Day DA, Lerner BM, Jimenez JL, Ziemann PJ, de Gouw JA. Quantification and source characterization of volatile organic compounds from exercising and application of chlorine-based cleaning products in a university athletic center. *Indoor Air*. 2020 Dec 18. doi: 10.1111/ina.12781. Epub ahead of print. PMID: 33337567.

Abstract

Humans spend approximately 90% of their time indoors, impacting their own air quality through occupancy and activities. Human VOC emissions indoors from exercise are still relatively uncertain, and questions remain about emissions from chlorine-based cleaners. To investigate these and other issues, the ATHLETic center study of Indoor Chemistry (ATHLETIC) campaign was conducted in the weight room of the Dal Ward Athletic Center at the University of Colorado Boulder. Using a Vocus Proton-Transfer-Reaction Time-of-Flight Mass Spectrometer (Vocus PTR-TOF), an Aerodyne Gas Chromatograph (GC), an Iodide-Chemical Ionization Time-of-Flight Mass Spectrometer (I-CIMS), and Picarro cavity ringdown spectrometers, we alternated measurements between the weight room and supply air, allowing for determination of VOC, NH₃, H₂O, and CO₂ emission rates per person (emission factors). Human-derived

emission factors were higher than previous studies of measuring indoor air quality in rooms with individuals at rest and correlated with increased CO₂ emission factors. Emission factors from personal care products (PCPs) were consistent with previous studies and typically decreased throughout the day. In addition, N-chloraldimines were observed in the gas phase after the exercise equipment was cleaned with a dichlor solution. The chloraldimines likely originated from reactions of free amino acids with HOCl on gym surfaces.

Gasoline and Service Stations

Gas stations, service stations and combination gas stations and service stations contribute to both air and water pollution. Pollution comes from fluid seepage, spilled gas and diesel, antifreeze and oil, and also other potentially hazardous materials such as copper and brass found in engine degreasers, lead, oil and grease can leak when radiators are flushed, working on brakes may release asbestos. These pollutants often end up in the air, soil, and water, and as time passes much of these pollutants end up in storm drains where they are carried to waterways.

Carwash Stations

The most common scenario is that with every vehicle washed, a large volume of otherwise drinkable water is turned into heavily polluted water laden with grime, oils and grease, heavy metals and detergents, which ends up as wastewater in waterways and increasing water pollution world-wide.⁴⁶

In a small minority of cases wastewater recycling is practiced. Although there are a number of ways of decreasing pollution and recycling wastewater, this practice could be dramatically enhanced by offering incentives and putting in place counter incentives by instituting a tax system that rewards stations that recycle wastewater and surcharges those wasting freshwater.⁴⁷

Drinking Water

Most drinking water from the tap is treated and while the treatment doesn't remove many of the contaminants, it adds contaminants such as chlorine and other disinfectants in the process. However, you're told by municipal water companies that their "treated" water is safe to drink. They know that their testing is inadequate and that their tap water for the most part contains many chemicals, with varying degrees of toxicity and effects on our metabolism and hormones.

Several studies have tested tap water in several areas of the US and found many dangerous chemicals in treated tap water, including drugs such as antidepressants and antibiotics, and chemicals used as herbicides and pesticides.

The use of bottled water has increased dramatically over the past few decades. In many countries it has outpaced the drinking of tap water.

Bottled water may seem to be a better alternative to tap water that has been treated as the treatment processes usually pollutes the treated water even as it clears up some of the pollution and infectious agents in the untreated water.

However, bottled water, has a significant adverse effects on pollution both in the water itself , the method of bottled water production, and the plastic bottles themselves.

While new methods are being developed to identify pollutants in water their use is limited and is rarely being used.

Reactivity-directed analysis - a novel approach for the identification of toxic organic electrophiles in drinking water

[Carsten Prasse](#)

Abstract

Drinking water consumption results in exposure to complex mixtures of organic chemicals, including natural and anthropogenic chemicals and compounds formed during drinking water treatment such as disinfection by-products. The complexity of drinking water contaminant mixtures has hindered efforts to assess associated health impacts. Existing approaches focus primarily on individual chemicals and/or the evaluation of mixtures, without providing information about the chemicals causing the toxic effect. Thus, there is a need for the development of novel strategies to evaluate chemical mixtures and provide insights into the species responsible for the observed toxic effects. This critical review introduces the application of a novel approach called Reactivity-Directed Analysis (RDA) to assess and identify organic electrophiles, the largest group of known environmental toxicants. In contrast to existing in vivo and in vitro approaches, RDA utilizes in chemico methodologies that investigate the reaction of organic electrophiles with nucleophilic biomolecules, including proteins and DNA. This review summarizes the existing knowledge about the presence of electrophiles in drinking water, with a particular focus on their formation in oxidative treatment systems with ozone, advanced oxidation processes, and UV light, as well as disinfectants such as chlorine, chloramines and chlorine dioxide. This summary is followed by an overview of existing RDA approaches and their application for the assessment of aqueous environmental matrices, with an emphasis on drinking water. RDA can be applied beyond drinking water, however, to evaluate source waters and wastewater for human and environmental health risks. Finally, future research demands for the detection and identification of electrophiles in drinking water via RDA are outlined.

Air Pollution

As well, a new studies on the adverse effects of air pollution appear almost daily. For example, a recent study, found that even short exposure to ultrafine particles, which come mostly from automobile exhaust, for even a few hours can cause serious heart disease in the form of non-fatal heart attacks.⁴⁸ You can read this research paper in PDF format for free at <https://ehp.niehs.nih.gov/doi/pdf/10.1289/EHP5478>.

There is an escalating amount of research that shows the havoc pollution causes to the earth and all of its inhabitants.

I'll mention some relevant ones, but you can see so many more simply by searching Medline and over the Internet. For example, this is worth watching: <https://www.youtube.com/watch?v=Zk11vl-7czE>.

Recent studies have found that breathing polluted air adversely affects the microbiome increasing morbidity and mortality, including increasing the risk of various detrimental parameters such as diabetes, obesity, cardiovascular disease, cancer, gastrointestinal disorders, and infections including the current coronavirus pandemic.⁴⁹⁵⁰

Air pollution exposure is associated with the gut microbiome as revealed by shotgun metagenomic sequencing.

[Fouladi F](#)¹, [Bailey MJ](#)², [Patterson WB](#)², [Sioda M](#)¹, [Blakley IC](#)¹, [Fodor AA](#)¹, [Jones RB](#)³, [Chen Z](#)⁴, [Kim JS](#)⁴, [Lurmann F](#)⁵, [Martino C](#)⁶, [Knight R](#)⁷, [Gilliland FD](#)⁴, [Alderete TL](#)⁸.

Abstract

Animal work indicates exposure to air pollutants may alter the composition of the gut microbiota. This study examined relationships between air pollutants and the gut microbiome in young adults residing in Southern California. Our results demonstrate significant associations between exposure to air pollutants and the composition of the gut microbiome using whole-genome sequencing. Higher exposure to 24-hour O₃ was associated with lower Shannon diversity index, higher *Bacteroides caecimuris*, and multiple gene pathways, including L-ornithine de novo biosynthesis as well as pantothenate and coenzyme A biosynthesis I. Among other pollutants, higher NO₂ exposure was associated with fewer taxa, including higher Firmicutes. The percent variation in gut bacterial composition that was explained by air pollution exposure was up to 11.2% for O₃ concentrations, which is large compared to the effect size for many other covariates reported in healthy populations. This study provides the first evidence of significant associations between exposure to air pollutants and the compositional and functional profile of the human gut microbiome. These results identify O₃ as an important pollutant that may alter the human gut microbiome.

Longer commutes are associated with increased human exposure to tris(1,3-dichloro-2-propyl) phosphate.

[Reddam A](#)¹, [Tait G](#)², [Herkert N](#)², [Hammel SC](#)², [Stapleton HM](#)², [Volz DC](#)³.

Abstract

Organophosphate esters (OPEs) are a class of semi-volatile organic compounds (SVOCs) used as flame retardants, plasticizers, and anti-foaming agents. Due to stringent flammability standards in vehicles and the ability of OPEs to migrate out of end-use products, elevated concentrations of OPEs have been found in car dust samples around the world. As many residents of Southern California spend a significant amount of time in their vehicles, there is potential for increased exposure to OPEs associated with longer commute times. As approximately 70% of the University of California, Riverside's undergraduate population commutes, the objective of this study was to use silicone wristbands to monitor personal exposure to OPEs and determine if exposure was associated with commute time in a subset of these students. Participants were asked to wear wristbands for five continuous days and complete daily surveys about the amount of time spent commuting. Data were then used to calculate a participant-specific total commute score. Components of Firemaster 550 (triphenyl phosphate, or TPHP, and isopropylated triaryl phosphate isomers) and Firemaster 600 (TPHP and tert-butylated triaryl phosphate isomers) - both widely used commercial flame retardant formulations - were strongly correlated with other OPEs detected within participant wristbands. Moreover, the concentration of tris(1,3-dichloro-2-propyl) phosphate (TDCIPP) was significantly correlated with the concentration of several Firemaster 550 components and tris(2-chloroisopropyl) phosphate (TCIPP). Finally, out of all OPEs measured, TDCIPP was significantly and positively correlated with total commute score, indicating that longer commutes are associated with increased human exposure to TDCIPP. Overall, our findings raise concerns about the potential for

chronic TDCIPP exposure within vehicles and other forms of transportation, particularly within densely populated and traffic-congested areas such as Southern California.

To read the full study in PDF format go to

<https://reader.elsevier.com/reader/sd/pii/S0160412019340644?token=882BD470BC63DC34BC61046AE4F60C779FCB778C14910EA5CF87BDE4ACE30F5CF71D76DC33AB64C6D32DBCA032920EA8>.

A recent study found that even in Africa pollution is escalating to the point where it affects visibility. The study, "Visibility as a proxy for air quality in East Africa" **Ajit Singh et al, 2020, Environ. Res. Lett. in press** Full text available at <https://doi.org/10.1088/1748-9326/ab8b12>.

The authors looked at PM air pollution in three East African cities: Addis Ababa, Nairobi and Kampala, from 1974 to 2018. They found that these cities have undergone rapid increases in population and national GDP growth (driven predominantly by study city's economies) which has resulted in increased rates of citywide fuel use and motorization, which provides a direct link to increased PM emissions and thus visibility loss.

Studies are also showing that pollution has an endocrine disrupting effects lowering testosterone on men and women and thus adversely impacting body composition and both mental and physical performance. For more information have a look at the full PDFs on [TestoBoost](#) and [GHboost](#) and other articles found in this newsletter and on www.MetabolicDiet.com/Shop under the individual nutritional supplement products.

However, it also seems that pollution is affecting children in several ways even to the point of epigenetically or mutationally altering children's hormonal status, behavior, cognitive function and potentials later in life.⁵¹⁵²⁵³⁵⁴⁵⁵

[Neurotoxicology](#). 2018 Sep;68:203-211. doi: 10.1016/j.neuro.2018.08.009. Epub 2018 Aug 23.

Developmental exposures to ultrafine particle air pollution reduces early testosterone levels and adult male social novelty preference: Risk for children's sex-biased neurobehavioral disorders.

[Sobolewski M](#)¹, [Anderson T](#)¹, [Conrad K](#)¹, [Marvin E](#)¹, [Klocke C](#)¹, [Morris-Schaffer K](#)¹, [Allen JL](#)¹, [Cory-Slechta DA](#)².

Abstract

Epidemiological studies have reported associations of air pollution exposures with various neurodevelopmental disorders such as autism spectrum disorder (ASD), attention deficit and schizophrenia, all of which are male-biased in prevalence. Our studies of early postnatal exposure of mice to the ultrafine particle (UFP) component of air pollution, considered the most reactive component, provide support for these epidemiological associations, demonstrating male-specific or male-biased neuropathological changes and cognitive and impulsivity deficits consistent with these disorders. Since these neurodevelopmental disorders also include altered social behavior and communication, the current study examined the ability of developmental UFP exposure to reproduce these social behavior deficits and to determine whether any observed alterations reflected changes in steroid hormone concentrations. Elevated plus maze,

social conditioned place preference, and social novelty preference were examined in adult mice that had been exposed to concentrated (10-20x) ambient UFPs averaging approximately 45 ug/m³ particle mass concentrations from postnatal day (PND) 4-7 and 10-13 for 4 h/day. Changes in serum testosterone (T) and corticosterone were measured at postnatal day (P)14 and approximately P120. UFP exposure decreased serum T concentrations on PND 14 and social nose-to-nose sniff rates with novel males in adulthood, suggesting social communication deficits in unfamiliar social contexts. Decreased sniff rates were not accounted for by alterations in fear-mediated behaviors and occurred without overt deficits in social preference, recognition or communication with a familiar animal or alterations in corticosterone. Adult T serum concentrations were positively correlated with nose to nose sniff rates. Collectively, these studies confirm another feature of male-biased neurodevelopmental disorders following developmental exposures to even very low levels of UFP air pollution that could be related to alterations in sex steroid programming of brain function.

[Paediatr Respir Rev.](#) 2019 Nov;32:73-81. doi: 10.1016/j.prrv.2019.06.003. Epub 2019 Jun 26.

Ultrafine particles and children's health: Literature review.

[da Costa E Oliveira JR](#)¹, [Base LH](#)², [de Abreu LC](#)³, [Filho CF](#)⁴, [Ferreira C](#)², [Morawska L](#)⁵.

Abstract

The aim of this study was to review and synthesize the existing knowledge of the effects of ultrafine particles [UFPs] with a specific focus on children's health. An extensive literature search identified 16 studies fulfilling the criteria set for the review. One of the most important findings of the review was that, in general, there is an association between children's health and exposure to UFPs, especially among children with respiratory diseases, who commonly experience alterations in inflammatory biomarkers and deterioration in lung function as a result of UFP exposure. Notably, the health effects of UFPs are related to their ability to penetrate through different systems of the body due to their small size.

[Neurochem Int.](#) 2019 Dec;131:104580. doi: 10.1016/j.neuint.2019.104580. Epub 2019 Oct 15.

Developmental impact of air pollution on brain function.

[Costa LG](#)¹, [Cole TB](#)², [Dao K](#)³, [Chang YC](#)³, [Garrick JM](#)³.

Abstract

Air pollution is an important contributor to the global burden of disease, particularly to respiratory and cardiovascular diseases. In recent years, evidence is accumulating that air pollution may adversely affect the nervous system as shown by human epidemiological studies and by animal models. Age appears to play a relevant role in air pollution-induced neurotoxicity, with growing evidence suggesting that air pollution may contribute to neurodevelopmental and neurodegenerative diseases. Traffic-related air pollution (e.g. diesel exhaust) is an important contributor to urban air pollution, and fine and ultrafine particulate matter (PM) may possibly be its more relevant component. Air pollution is associated with increased oxidative stress and inflammation both in the periphery and in the nervous system, and fine and ultrafine PM can directly access the central nervous system. This short review focuses on the adverse effects of air pollution on the developing brain; it discusses some characteristics that make the developing brain more susceptible to toxic effects, and summarizes the animal and human evidence suggesting that exposure to elevated air pollution is associated with a number of behavioral and biochemical adverse effects. It also discusses more in detail the emerging evidence of an association between perinatal exposure to air pollution and increased risk of autism spectrum disorder.

Some of the common mechanisms that may underlie the neurotoxicity and developmental neurotoxicity of air pollution are also discussed. Considering the evidence presented in this review, any policy and legislative effort aimed at reducing air pollution would be protective of children's well-being.

[Toxicol Pathol.](#) 2019 Dec;47(8):976-992. doi: 10.1177/0192623319878400. Epub 2019 Oct 14.

The Impact of Inhaled Ambient Ultrafine Particulate Matter on Developing Brain: Potential Importance of Elemental Contaminants.

[Cory-Slechta DA](#)¹, [Sobolewski M](#)¹, [Marvin E](#)¹, [Conrad K](#)¹, [Merrill A](#)¹, [Anderson T](#)¹, [Jackson BP](#)², [Oberdorster G](#)¹.

Abstract

Epidemiological studies report associations between air pollution (AP) exposures and several neurodevelopmental disorders including autism, attention deficit disorder, and cognitive delays. Our studies in mice of postnatal (human third trimester brain equivalent) exposures to concentrated ambient ultrafine particles (CAPs) provide biological plausibility for these associations, producing numerous neuropathological and behavioral features of these disorders, including male-biased vulnerability. These findings raise questions about the specific components of AP that underlie its neurotoxicity, which our studies suggest could involve trace elements as candidate neurotoxicants. X-ray fluorescence analyses of CAP chamber filters confirm contamination of AP exposures by multiple elements, including iron (Fe) and sulfur (S). Correspondingly, laser ablation inductively coupled plasma mass spectrometry of brains of male mice indicates marked postexposure elevations of Fe and S and other elements. Elevations of brain Fe and S in particular are consistent with potential ferroptotic, oxidative stress, and altered antioxidant capacity-based mechanisms of CAPs-induced neurotoxicity, supported by observations of increased serum oxidized glutathione and increased neuronal cell death in nucleus accumbens with no corresponding significant increase in caspase-3, in male brains following postnatal CAP exposures. Understanding the role of trace element contaminants of particulate matter AP as a source of neurotoxicity is critical for public health protection.

Besides the information in this article under the heading **Decreasing Body Pollution with Specific Nutritional Supplementation** I've added the potential beneficial effects of Coenzyme-Q10 (CoQ10 - found in several products in my line of nutritional supplements, on mitigating some aspects of pollution. For example, a recent paper published in early February 2020 proposes that "CoQ10 may counteract BPA-induced reprotoxicity".⁵⁶ More on that below but for a more comprehensive look on the benefits of CoQ10 go to the article on CoQ10 that is also in this newsletter.

I've also added and revised several section in this article including a new section on "Some Easy Ways to Decrease Exposure to Pollutants Including Endocrine Disrupting Chemicals."

The Brave New World of Pollution

Since the Industrial Revolution in the 1700s, the world has increasingly become a cesspool of pollution. Pollution existed for millennia before then but the toxicity of the pollution, mostly organic, wasn't the same in that disease-causing inadequate sanitation was the main problem then and not the toxic pollution that permeates our lives today.

There are many forms of pollution besides the obvious ones of air, water and soil pollution by industrial and other chemicals including drugs, insecticides, herbicides, and vehicular pollution with even low levels of pollutants will impact morbidity and mortality.⁵⁷

For example, even though lead pollution has declined since the advent of lead-free gasoline and other methods used to decrease lead contamination, a recent study found that even low levels of lead exposure can increase the risk for premature death. The study estimates that as many as 256,000 premature deaths from cardiovascular disease and 185,000 deaths from ischemic heart disease in the United States each year are linked to lead exposure in people over age 44 years⁵⁸ – **see citation and abstract, as well as a link to free access of the full text below.**

Other heavy metals, such as cadmium and lead, even at low levels, can adversely affect our immune system and health, and make us more susceptible to infections including COVID-19 infections.⁵⁹⁶⁰

For example, a recent study found that high levels of cadmium (found in cigarettes and in contaminated vegetables, are associated with higher mortality in patients with influenza or pneumonia. It's also likely to exacerbate infections by respiratory tract viruses **including the severity of COVID-19 infections.**⁶¹

Environ Health Perspect. 2020 Dec;128(12):127004. doi: [10.1289/EHP7598](https://doi.org/10.1289/EHP7598). Epub 2020 Dec 16.
PMID: 33325772.

Environmental Cadmium and Mortality from Influenza and Pneumonia in U.S. Adults
[Sung Kyun Park](#), [Coralynn Sack](#), [Matti J Sirén](#), [Howard Hu](#).

Abstract

Background: Environmental cadmium exposure is widespread. In humans, cadmium is poorly excreted, triggers pulmonary inflammation, reduces pulmonary function, and enhances lung injury by respiratory syncytial virus.

Objectives: We examined the association of cadmium burden with mortality related to influenza or pneumonia.

Methods: This prospective analysis of the National Health and Nutrition Examination Survey (NHANES) included 7,173 and 8,678 participants $\geq 45 \geq 45$ years of age enrolled in NHANES-III and NHANES 1999-2006, respectively. Associations were evaluated between cadmium and mortality from influenza or pneumonia during a median follow-up of 17.3 y (NHANES-III, based on creatinine-corrected urine cadmium) and 11.4 y (NHANES 1999-2006, based on blood cadmium). Survey-weighted Cox proportional hazard models were used to compute hazard ratios (HRs) comparing the mortality of individuals at the 80th vs. the 20th percentile of cadmium concentrations.

Results: In NHANES-III, after adjustment for sex, race/ethnicity, education, body mass index, serum cholesterol, hypertension, and NHANES phase (or cycle), the HR comparing influenza or pneumonia mortality among participants with creatinine-corrected urinary cadmium in the 80th vs. 20th percentile was 1.15 (95% CI: 1.05, 1.26; $p=0.002$ $p=0.002$) in the population as a whole and 1.27 (95% CI: 1.12, 1.43; $p=0.002$ $p=0.002$) among never smokers. In NHANES 1999-2006, adjusted HRs for the 80th vs. 20th percentile of blood cadmium were 1.14 (95% CI: 0.96, 1.36; $p=0.15$ $p=0.15$) for the overall population and 1.71 (95% CI: 0.95, 3.09; $p=0.07$ $p=0.07$) in never smokers.

Discussion: Among middle-aged and older adults in the United States, higher cadmium burdens are associated with higher mortality from influenza or pneumonia. This raises the possibility that cadmium may worsen outcomes from COVID-19 infections. **Full text in PDF format available at -**

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7739956/pdf/ehp7598.pdf>.

Low-level lead exposure and mortality in US adults: a population-based cohort study.

Lancet Public Health. 2018 Apr;3(4):e177-e184. doi: 10.1016/S2468-2667(18)30025-2. PMID: 29544878.

[Lanphear BP](#)¹, [Rauch S](#)², [Auinger P](#)³, [Allen RW](#)⁴, [Hornung RW](#)⁵.

Abstract

Lead exposure is a risk factor for cardiovascular disease mortality, but the number of deaths in the USA attributable to lead exposure is poorly defined. We aimed to quantify the relative contribution of environmental lead exposure to all-cause mortality, cardiovascular disease mortality, and ischaemic heart disease mortality.

METHODS:

Our study population comprised a nationally representative sample of adults aged 20 years or older who were enrolled in the Third National Health and Nutrition Examination Survey (NHANES-III) between 1988 and 1994 and followed up to Dec 31, 2011. Participants had completed a medical examination and home interview and had results for concentrations of lead in blood, cadmium in urine, and other relevant covariates. Individuals were linked with the National Death Index. This study presents extended follow-up of an earlier analysis.

FINDINGS:

We included 14 289 adults in our study. The geometric mean concentration of lead in blood was 2.71 µg/dL (geometric SE 1.31). 3632 (20%) participants had a concentration of lead in blood of at least 5 µg/dL (≥ 0.24 µmol/L). During median follow-up of 19.3 years (IQR 17.6-21.0), 4422 people died, 1801 (38%) from cardiovascular disease and 988 (22%) from ischaemic heart disease. An increase in the concentration of lead in blood from 1.0 µg/dL to 6.7 µg/dL (0.048 µmol/L to 0.324 µmol/L), which represents the tenth to 90th percentiles, was associated with all-cause mortality (hazard ratio 1.37, 95% CI 1.17-1.60), cardiovascular disease mortality (1.70, 1.30-2.22), and ischaemic heart disease mortality (2.08, 1.52-2.85). The population attributable fraction of the concentration of lead in blood for all-cause mortality was 18.0% (95% CI 10.9-26.1), which is equivalent to 412 000 deaths annually. Respective fractions were 28.7% (15.5-39.5) for cardiovascular disease mortality and 37.4% (23.4-48.6) for ischaemic heart disease mortality, which correspond to 256 000 deaths a year from cardiovascular disease and 185 000 deaths a year from ischaemic heart disease.

INTERPRETATION:

Low-level environmental lead exposure is an important, but largely overlooked, risk factor for cardiovascular disease mortality in the USA. A comprehensive strategy to prevent deaths from cardiovascular disease should include efforts to reduce lead exposure.

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Chemosphere. 2021 Jan;262:128350. doi: 10.1016/j.chemosphere.2020.128350. Epub 2020 Sep 16.

PMID: 33182141. DOI: [10.1016/j.chemosphere.2020.128350](https://doi.org/10.1016/j.chemosphere.2020.128350)

Heavy metal associated health hazards: An interplay of oxidative stress and signal transduction

[Jagdish Gopal Paithankar](#)¹, [Sanjay Saini](#)², [Shiwangi Dwivedi](#)¹, [Anurag Sharma](#)³, [Debapratim Kar Chowdhuri](#)⁴

Abstract

Heavy metal-induced cellular and organismal toxicity have become a major health concern in biomedical science. Indiscriminate use of heavy metals in different sectors, such as, industrial-, agricultural-, healthcare-, cosmetics-, and domestic-sectors has contaminated environment matrices and poses a severe health concern. Xenobiotics mediated effect is a ubiquitous cellular response. Oxidative stress is one such prime cellular response, which is the result of an imbalance in the redox system. Further, oxidative stress is associated with macromolecular damages and activation of several cell survival and cell death pathways. Epidemiological as well as laboratory data suggest that oxidative stress-induced cellular response following heavy metal exposure is linked with an increased risk of neoplasm, neurological disorders, diabetes, infertility, developmental disorders, renal failure, and cardiovascular disease. During the recent past, a relation among heavy metal exposure, oxidative stress, and signaling pathways have been explored to understand the heavy metal-induced toxicity. Heavy metal-induced oxidative stress and its connection with different signaling pathways are complicated; therefore, the systemic summary is essential. Herein, an effort has been made to decipher the interplay among heavy metals/metalloids (Arsenic, Chromium, Cadmium, and Lead) exposures, oxidative stress, and signal transduction, which are essential to mount the cellular and organismal response. The signaling pathways involved in this interplay include NF- κ B, NRF2, JAK-STAT, JNK, FOXO, and HIF.

Food Chem Toxicol. 2020 Dec;146:111809. doi: [10.1016/j.fct.2020.111809](https://doi.org/10.1016/j.fct.2020.111809). Epub 2020 Oct 16.

Toxic metal exposure as a possible risk factor for COVID-19 and other respiratory infectious diseases

[Anatoly V Skalny](#)¹, [Thania Rios Rossi Lima](#)², [Tao Ke](#)³, [Ji-Chang Zhou](#)⁴, [Julia Bornhorst](#)⁵, [Svetlana I Alekseenko](#)⁶, [Jan Aaseth](#)⁷, [Ourania Anesti](#)⁸, [Dimosthenis A Sarigiannis](#)⁹, [Aristides Tsatsakis](#)¹⁰, [Michael Aschner](#)¹¹, [Alexey A Tinkov](#)¹²

Abstract

Multiple medical, lifestyle, and environmental conditions, including smoking and particulate pollution, have been considered as risk factors for COroNaVIrus Disease 2019 (COVID-19) susceptibility and severity. Taking into account the high level of toxic metals in both particulate matter (PM_{2.5}) and tobacco smoke, the objective of this review is to discuss recent data on the role of heavy metal exposure in development of respiratory dysfunction, immunotoxicity, and severity of viral diseases in epidemiological and experimental studies, as to demonstrate the potential crossroads between heavy metal exposure and COVID-19 severity risk. The existing data demonstrate that As, Cd, Hg, and Pb exposure is associated with respiratory dysfunction and respiratory diseases (COPD, bronchitis). These observations corroborate laboratory findings on the role of heavy metal exposure in impaired mucociliary clearance, reduced barrier function, airway inflammation, oxidative stress, and apoptosis. The association between heavy metal exposure and severity of viral diseases, including influenza and respiratory syncytial virus has been also demonstrated. The latter may be considered a consequence of adverse effects of metal exposure on adaptive immunity. Therefore, reduction of toxic metal exposure may be considered as a potential tool for reducing susceptibility and severity of viral diseases affecting the respiratory system, including COVID-19.

Another example is a recent study that shows that chemicals in lavender and tea tree oil are endocrine disrupters, as are many other essential oils that are freely available on the market and not regulated by the FDA – see <https://www.endocrine.org/news-room/2018/chemicals-in-lavender-and-tea-tree-oil-appear-to-be-hormone-disruptors>.

There are many chemicals that are endocrine disrupters in that they disrupt the body's hormones acutely and their effects may extend beyond more than one generation. While I'll cover some of the sources and effects of endocrine disrupters below, a recent review looked at the effect of Bisphenol A (BPA) on mortality and concluded that BPA “increased risk of all-cause mortality”.⁶²

In the Introduction of this paper (the full paper is available at https://jamanetwork.com/journals/jamanetworkopen/fullarticle/2769313?utm_source=silverchair&utm_medium=email&utm_campaign=article_alert-jamanetworkopen&utm_term=mostread&utm_content=olf-widget_09112020) the authors stated: **“Widespread exposure to bisphenol A (BPA) has emerged as a major public health concern.^{1,2} Bisphenol A is a high-volume industrial chemical produced primarily for the manufacturing of polycarbonate plastics and epoxy resins. Polycarbonate plastics based on BPA are used in many consumer products, such as plastic bottles, sports equipment, compact discs, some medical devices, and dental sealants and composites. Epoxy resins that contain BPA are used to line water pipes, coat the inside of food and beverage cans, and make thermal paper such as that used in sales receipts.^{3,4} As a result, BPA exposure to humans is ubiquitous from a variety of sources ranging from consumer products, food, and water to dust.⁵ National biomonitoring data in the United States show that BPA is detectable in more than 90% of urine samples in the general population.^{6,7} Currently in the United States, 12 states and Washington, DC have restrictions in place against BPA. In Europe, the European Chemical Agency has added BPA to the Candidate List of substances of very high concern. Evidence from animal studies has shown that BPA has endocrine-disrupting effects.^{8,9} Exposure to BPA can disrupt endocrine function and metabolism, inducing the development of obesity and metabolic disorders.^{10,11} Exposure to BPA can also induce cardiac arrhythmias, accelerate atherosclerosis, decrease atrial contraction rate and force, and lead to cardiac remodeling in animal models.¹²⁻¹⁷ Moreover, previous epidemiologic studies have shown that BPA exposure is associated with an increased risk of obesity,¹⁸⁻²¹ diabetes,²²⁻²⁴ hypertension,²⁵ and cardiovascular disease (CVD).^{22,26}”**

Other forms of pollution that impact our health and quality of life include noise, light, various kinds of radiation and electromagnetic pollution, pollution from various methods producing and disposal of just about everything that's manufactured today, including littering, plastics, computers, cell phones, wireless/wired electronics, etc.

For example, noise pollution can impact our environment and health as much as other forms of pollution. The most common sources of noise pollution are from traffic (including trains) and aircrafts and studies have shown that both impact morbidity and mortality.

In 2018 the World Health Organization published environmental noise guidelines, which is available at https://www.euro.who.int/_data/assets/pdf_file/0008/383921/noise-guidelines-eng.pdf.

Below is the Foreword of that publication.

Noise is one of the most important environmental risks to health and continues to be a growing concern among policy-makers and the public alike. Based on the assessment threshold specified in the Environmental Noise Directive of the European Union (EU), at least 100 million people in the EU are affected by road traffic noise, and in western Europe alone at least 1.6 million healthy years of life are lost as a result of road traffic noise. At the

request of Member States at the Fifth Ministerial Conference on Environment and Health in Parma, Italy, in March 2010, the WHO Regional Office for Europe has developed these guidelines, based on the growing understanding of the health impacts of exposure to environmental noise. They provide robust public health advice, which is essential to drive policy action that will protect communities from the adverse effects of noise. These WHO guidelines – the first of their kind globally – provide recommendations for protecting human health from exposure to environmental noise originating from various sources. They not only offer robust public health advice but also serve as a solid basis for future updates, given the growing recognition of the problem and the rapid advances in research on the health impacts of noise. The comprehensive process of developing the guidelines has followed a rigorous methodology; their recommendations are based on systematic reviews of evidence that consider more health outcomes of noise exposure than ever before. Through their potential to influence urban, transport and energy policies, these guidelines contribute to the 2030 Agenda for Sustainable Development and support WHO's vision of creating resilient communities and supportive environments in the European Region. Following the publication of WHO's community noise guidelines in 1999 and night noise guidelines for Europe in 2009, these latest guidelines represent the next evolutionary step, taking advantage of the growing diversity and quality standards in this research domain. Comprehensive and robust, and underpinned by evidence, they will serve as a sound basis for action. While these guidelines focus on the WHO European Region and provide policy guidance to Member States that is compatible with the noise indicators used in the EU's Environmental Noise Directive, they still have global relevance. Indeed, a large body of the evidence underpinning the recommendations was derived not only from noise effect studies in Europe but also from research in other parts of the world – mainly in Asia, Australia and the United States of America. I am proud to present these guidelines as another leading example of the normative work undertaken in our Region in the area of environment and health. On behalf of the WHO Regional Office for Europe and our European Centre for Environment and Health in Bonn, Germany, which coordinated the development of the guidelines, I would like to express my gratitude to the large network of experts, partners, colleagues and consultants who have contributed to this excellent publication. I would also like to thank Switzerland and Germany for providing financial support to this complex project, and look forward to following the influence of the guidelines on policy and research in the years to come. Dr Zsuzsanna Jakab WHO Regional Director for Europe

Environ Sci Pollut Res Int. 2020 Oct 23. doi: 10.1007/s11356-020-11190-4. Online ahead of print. PMID: 33094463

Exposure to the road traffic noise in an urban complex in Greece: the quantification of healthy life years lost due to noise-induced annoyance and noise-induced sleep disturbances

[Paraskevi Begou](#)¹, [Pavlos Kassomenos](#)²

Abstract

In the recent years, the environmental noise is a global issue of great concern. Especially, it is considered to be one of the most deleterious environmental risk factors for the human health and well-being in urban areas. In this study, we focus on the major source of the transportation noise in the modern-day urbanized societies, which is the road traffic noise. The study was performed in the urban complex in Thessaloniki-Neapoli in Greece, and the estimation of the road traffic noise levels was based on the CoRTN (Calculation of Road Traffic Noise) prediction method. In addition, we estimated the EBD (environmental burden of disease), in terms of DALYs (disability-adjusted life years), due to the annoyance and sleep disturbances associated with the exposure to road traffic noise. The estimation of the EBD was based on the strategic noise mapping in Thessaloniki-Neapoli as well as on the guidance document produced by the WHO (World Health Organization) for the quantitative assessment on the humans' health consequences of the environmental noise. Above all, the results revealed the magnitude of the health damage caused by the transportation noise.

Eur Heart J . 2020 Nov 27;ehaa957. doi: 10.1093/eurheartj/ehaa957. Online ahead of print.

Does night-time aircraft noise trigger mortality? A case-crossover study on 24 886 cardiovascular deaths

[Apolline Saucy](#)^{1,2}, [Beat Schäffer](#)³, [Louise Tangermann](#)^{1,2}, [Danielle Vienneau](#)^{1,2}, [Jean-Marc Wunderli](#)³, [Martin Röösli](#)^{1,2} PMID: 33245107 - DOI: [10.1093/eurheartj/ehaa957](https://doi.org/10.1093/eurheartj/ehaa957)

Abstract

Aims: It is unclear whether night-time noise events, including from aeroplanes, could trigger a cardiovascular death. In this study, we investigate the potential acute effects of aircraft noise on mortality and the specific role of different night-time exposure windows by means of a case-crossover study design.

Methods and results: We selected 24 886 cases of death from cardiovascular disease (CVD) from the Swiss National Cohort around Zürich Airport between 2000 and 2015. For night-time deaths, exposure levels 2 h preceding death were significantly associated with mortality for all causes of CVD [OR = 1.44 (1.03-2.04) for the highest exposure group (LAeq > 50 dB vs. <20 dB)]. Most consistent associations were observed for ischaemic heart diseases, myocardial infarction, heart failure, and arrhythmia. Association were more pronounced for females (P = 0.02) and for people living in areas with low road and railway background noise (P = 0.01) and in buildings constructed before 1970 (P = 0.36). We calculated a population attributable fraction of 3% in our study population.

Conclusion: Our findings suggest that night-time aircraft noise can trigger acute cardiovascular mortality. The association was similar to that previously observed for long-term aircraft noise exposure.

Plastic Pollution

Over the last century the escalating use of plastics has resulted in the ubiquitous plastic pollution of our planet. Because plastics are relatively inexpensive and slow to degrade the accumulating pollution adversely affects all living entities, including wildlife and humans.

Plastics contain and leach several toxic and hazardous chemicals. Among these are endocrine-disrupting chemicals (EDCs) that significantly impact health and are involved in a variety of diseases including cancer, cardiovascular disease, diabetes, and disruptions of metabolic and hormonal processes.

A new report, [Plastics, EDCs, & Health](#), from the Endocrine Society and the IPEN (International Pollutants Elimination Network), presents the relevant research and details the harmful effects of EDCs contained in plastics.

The first two paragraphs of the Foreword of this report are as follows”

Chemical additives in plastic and the threat they pose to human health and the environment is an emerging issue of global concern that is garnering increasing attention as society is beginning to address the world-wide plastic pollution problem. The publication *Plastics, EDCs and Health*, produced by the Endocrine Society and authored by a leading international group of scientists and professors, is an authoritative and comprehensive resource. The report details the endocrine disrupting chemicals

(EDCs) in plastics and the hazards that these chemicals pose to human health throughout the life-cycle of plastics.

Many plastic additives are known to interfere with hormone functioning and are, by definition, endocrine disrupting chemicals. This publication provides clear and extensive evidence of the human health impacts of many chemicals in common plastics. The health impacts of these widely used chemicals can be profound and life threatening. Cancers, diabetes, kidney, liver, and thyroid impacts, metabolic disorders, neurological impacts, inflammation, alterations to both male and female reproductive development, infertility, and impacts to future generations as a result of germ cell alterations are the consequence of many EDC exposures, EDCs that are integral to plastics.

Microplastics

An example of pollution that many are unaware of is microplastics, tiny fragments of plastic that are ubiquitous in all waters, including water we drink from all sources. For more information go [http://www.thelancet.com/journals/lanplh/article/PIIS2542-5196\(17\)30121-3](http://www.thelancet.com/journals/lanplh/article/PIIS2542-5196(17)30121-3), <http://www.uni-bayreuth.de/en/university/press/press-releases/2018/037-microplastics-report/index.html> and <https://www.sciencedaily.com/releases/2016/12/161219151752.htm>.

A recent study found the nanoscale plastics are ubiquitous in water everywhere including all the water we drink.⁶³ Not only that but the increase in coastal populations worldwide leads to increases in the contamination of seafood and can cause potential health risks to humans, even large distances from pollution sources.⁶⁴

Another recent study found that environmentally exposed microplastic particles enter the body much more than pristine microplastic particles with significant toxic and endocrine disrupting effects⁶⁵ (see abstract below).

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Environmental exposure enhances the internalization of microplastic particles into cells

[A F R M Ramsperger](#)^{1,2}, [V K B Narayana](#)¹, [W Gross](#)², [J Mohanraj](#)³, [M Thelakkat](#)³, [A Greiner](#)⁴, [H Schmalz](#)⁴, [H Kress](#)⁵, [C Laforsch](#)⁶

Abstract

Microplastic particles ubiquitously found in the environment are ingested by a huge variety of organisms. Subsequently, microplastic particles can translocate from the gastrointestinal tract into the tissues likely by cellular internalization. The reason for cellular internalization is unknown, since this has only been shown for specifically surface-functionalized particles. We show that environmentally exposed microplastic particles were internalized significantly more often than pristine microplastic particles into macrophages. We identified biomolecules forming an eco-corona on the surface of microplastic particles, suggesting that environmental exposure promotes the cellular internalization of microplastics. Our findings further indicate that cellular internalization is a key route by which microplastic particles translocate into tissues, where they may cause toxicological effects that have implications for the environment and human health.

Full text PDF is available at - <https://advances.sciencemag.org/content/6/50/eabd1211/tab-pdf>.

It's estimated that just cleaning up surface plastic, which by current technologies could take several decades, does not take into account the plastic that is in the deeper part of the oceans, with that plastic being essentially lost and uncleanable, and affecting marine life more than the surface plastics.⁶⁶⁶⁷

Great Pacific Garbage Patch growing rapidly, study shows

New analysis reveals the region contains as much as sixteen times more plastic than previously estimated, with pollution levels increasing exponentially

March 22, 2018

A new study shows that 1.8 trillion pieces of plastic weighing 80,000 metric tons are currently afloat in an area known as the Great Pacific Garbage Patch -- and the problem is rapidly getting worse.

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Plastic samples collected during The Ocean Cleanup's Mega Expedition, 2015.

Credit: Image courtesy of The Ocean Cleanup

1.8 trillion pieces of plastic weighing 80,000 metric tons are currently afloat in an area known as the Great Pacific Garbage Patch -- and it is rapidly getting worse. These are the main conclusions of a three-year mapping effort conducted by an international team of scientists affiliated with The Ocean Cleanup Foundation, six universities and an aerial sensor company. Their findings were published today in the journal *Scientific Reports*.

The Great Pacific Garbage Patch (GPGP), located halfway between Hawaii and California, is the largest accumulation zone for ocean plastics on Earth. Conventionally, researchers have used single, fine-meshed nets, typically less than a meter in size, in an attempt to quantify the problem. However, this method yields high uncertainty because of the small

surface area that is covered. Additionally, these methods could not measure the magnitude of the problem to its fullest extent, because all sampling nets -- small and large -- were unable to capture objects greater than the size of the net.

In order to analyze the full extent of the GPGP, the team conducted the most comprehensive sampling effort of the GPGP to date by crossing the debris field with 30 vessels simultaneously, supplemented by two aircraft surveys. Although most vessels were equipped with standard surface sampling nets, the fleet's mothership *RV Ocean Starr* also trawled two six-meter-wide devices, which allowed the team to sample medium to large-sized objects.

To increase the surface area surveyed, and quantify the largest pieces of plastic -- objects that include discarded fishing nets several meters in size -- a C-130 Hercules aircraft was fitted with advanced sensors to collect multispectral imagery and 3D scans of the ocean garbage. The fleet collected a total of 1.2 million plastic samples, while the aerial sensors scanned more than 300 km² of ocean surface.

The results, published today in *Scientific Reports*, reveal that the GPGP, defined as the area with more than 10 kg of plastic per km², measures 1.6 million square kilometers, three times the size of continental France. Accumulated in this area are 1.8 trillion pieces of plastic, weighing 80,000 metric tons, the equivalent of 500 Jumbo Jets. These figures are four to sixteen times higher than previous estimates. 92% of the mass is represented by larger objects; while only 8% of the mass is contained in microplastics, defined as pieces smaller than 5 mm in size.

"We were surprised by the amount of large plastic objects we encountered," said Dr. Julia Reisser, Chief Scientist of the expeditions. "We used to think most of the debris consists of small fragments, but this new analysis shines a new light on the scope of the debris."

By comparing the amount of microplastics with historical measurements of the GPGP, the team found that plastic pollution levels within the GPGP have been growing exponentially since measurements began in the 1970s. Laurent Lebreton, lead author of the study, explains: "Although it is not possible to draw any firm conclusions on the persistency of plastic pollution in the GPGP yet, this plastic accumulation rate inside the GPGP, which was greater than in the surrounding waters, indicates that the inflow of plastic into the patch continues to exceed the outflow."

Boyan Slat, Founder of The Ocean Cleanup and co-author of the study, elaborated on the relevance of the findings for his organisation's cleanup plans: "To be able to solve a problem, we believe it is essential to first understand it. These results provide us with key data to develop and test our cleanup technology, but it also underlines the urgency of dealing with the plastic pollution problem. Since the results indicate that the amount of hazardous microplastics is set to increase more than tenfold if left to fragment, the time to start is now."

More information:

Great Pacific Garbage Patch page: <https://www.theoceancleanup.com/great-pacific-garbage-patch/>

Video links:

The Great Pacific Garbage Patch -- Explainer: <https://youtu.be/0EyaTqezSzs>

Boyan Slat -- The New Picture of the Great Pacific Garbage Patch (2018): <https://youtu.be/VxMATP5oRx4>

Story Source:

[Materials](#) provided by [The Ocean Cleanup](#). Note: Content may be edited for style and length.

Journal Reference:

1. L. Lebreton, B. Slat, F. Ferrari, B. Sainte-Rose, J. Aitken, R. Marthouse, S. Hajbane, S. Cunsolo, A. Schwarz, A. Levivier, K. Noble, P. Debeljak, H. Maral, R. Schoeneich-Argent, R. Brambini, J. Reisser. **Evidence that the Great Pacific Garbage Patch is rapidly accumulating plastic.** *Scientific Reports*, 2018; 8 (1) DOI: [10.1038/s41598-018-22939-w](https://doi.org/10.1038/s41598-018-22939-w)
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The Ocean Cleanup. "Great Pacific Garbage Patch growing rapidly, study shows: New analysis reveals the region contains as much as sixteen times more plastic than previously estimated, with pollution levels increasing exponentially." ScienceDaily. ScienceDaily, 22 March 2018.
<www.sciencedaily.com/releases/2018/03/180322123755.htm>.

Researchers estimate 10,000 metric tons of plastic enter Great Lakes every year

Study inventories movement of plastic and microplastic debris throughout lake system

December 19, 2016

Rochester Institute of Technology

A new study that inventories and tracks high concentrations of plastic in the Great Lakes could help inform cleanup efforts and target pollution prevention. Researchers found that nearly 10,000 metric tons -- or 22 million pounds -- of plastic debris enter the Great Lakes every year from the United States and Canada.

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FULL STORY



Plastic pollution in Lake Michigan is approximately the equivalent of 100 Olympic-sized pools full of plastic bottles dumped into the lake every year, say researchers.

A recent study by Rochester Institute of Technology that inventories and tracks high concentrations of plastic in the Great Lakes could help inform cleanup efforts and target pollution prevention.

Researchers found that nearly 10,000 metric tons -- or 22 million pounds -- of plastic debris enter the Great Lakes every year from the United States and Canada. Matthew Hoffman, assistant professor in RIT's School of Mathematical Sciences, is the lead author of "Inventory and transport of plastic debris in the Laurentian Great Lakes," which will run in an upcoming issue of *Marine Pollution Bulletin*.

"This study is the first picture of the true scale of plastic pollution in the Great Lakes," Hoffman said. Hoffman used computer simulations to follow the volume of plastic debris moving across state and international boundaries -- from Illinois to Michigan and from Canada to the United States.

Earlier studies estimate 40,000 to 110,000 metric tons of plastics enter the oceans along the U.S. coastline, Hoffman said.

In their study, Hoffman and co-author Eric Hittinger, assistant professor of public policy at RIT, report that half of the plastic pollution entering the Great Lakes -- 5,000 metric tons per year -- goes into Lake Michigan, followed by Lake Erie with 2,500 metric tons and Lake Ontario with 1,400 metric tons. Lake Huron receives 600 metric tons of plastic and Lake Superior, 32 metric tons per year.

Estimates of surface microplastics entering the lakes each year show 4.41 metric tons in Lake Erie, 1.44 metric tons in Lake Huron and .0211 metric tons in Lake Superior.

Plastic pollution in Lake Michigan is approximately the equivalent of 100 Olympic-sized pools full of plastic bottles dumped into the lake every year, Hittinger said, whereas the yearly amount of plastic in Lake Ontario equates to 28 Olympic-sized pools full of plastic bottles.

Prior observational studies measured localized concentrations of plastic pollution in the open water, tributaries and along the shorelines. The new study applied mathematical modeling for the first time to extend the scope of the problem over time and spatial scales.

The inventory gives full mass estimates on the entire connected lake system and maps plastic debris moving between lakes and across interstate and international borders. The results provide environmentally realistic concentrations of plastic in the Great Lakes.

Findings of the study show debris travels differently in the Great Lakes than in the ocean. Instead of the floating "garbage patches" found in the ocean, plastic in the Great Lakes are carried by persistent winds and lake currents to the shore -- often washing up in another state or country, Hoffman said.

Plastic accounts for approximately 80 percent of the litter on the shorelines of the Great Lakes. The study quantifies dense plastic that quickly sinks and surface plastics like microbeads, fragments and pellets, plastic line and Styrofoam, which could be consumed by wildlife and potentially enter the food chain.

Major population centers are the primary sources of plastic pollution in the Great Lake system, with Chicago, Toronto, Cleveland and Detroit releasing more plastic particles than accumulate on their shorelines.

"Most of the particles from Chicago and Milwaukee end up accumulating on the eastern shores of Lake Michigan, while the particles from Detroit and Cleveland end up along the southern coast of the eastern basin of Lake Erie," Hoffman said. "Particles released from Toronto appear to accumulate on the southern coast of Lake Ontario, including around Rochester and Sodus Bay."

Estimates of plastic pollution throughout the Great Lakes were derived using population dynamics within 100 kilometers, or 62 miles, of the shores and hydrodynamic modeling to simulate the distribution of plastic debris throughout the Great Lakes from 2009 to 2014. Data from the National Oceanic and Atmospheric Administration Great Lakes Coastal Forecast System were used to simulate currents transporting plastic debris throughout the lake system.

Story Source:

[Materials](#) provided by [Rochester Institute of Technology](#). Original written by Susan Gawlowicz. *Note: Content may be edited for style and length.*

Journal Reference:

1. Matthew J. Hoffman, Eric Hittinger. **Inventory and transport of plastic debris in the Laurentian Great Lakes.** *Marine Pollution Bulletin*, 2016; DOI: [10.1016/j.marpolbul.2016.11.061](https://doi.org/10.1016/j.marpolbul.2016.11.061)

Rochester Institute of Technology. "Researchers estimate 10,000 metric tons of plastic enter Great Lakes every year: Study inventories movement of plastic and microplastic debris throughout lake system." ScienceDaily. ScienceDaily, 19 December 2016. <www.sciencedaily.com/releases/2016/12/161219151752.htm>.

Air Pollution

News

The World Health Organization (WHO) ranks air pollution as the major environmental cause of premature death.⁶⁸ A recent study, while mentioning the WHO announcement, found that traffic-related air pollution is adversely affecting mental health.⁶⁹

And on December 15, 2020. For the first time, in a landmark judgement, a UK coroner has said that air pollution was the cause of death of a 9-year-old London schoolgirl Ella Kissi-Debrah. This official ruling, and its legal and moral consequences, hopefully will spur countries to do more than just give lip service and seriously tackle the task of significantly decreasing the many deadly sources of pollution in the world.

Introduction

Besides all the bad effects of air pollution, both outdoor and indoor, exposure to any air pollution, even short-term and low levels, increases morbidity including cardiopulmonary and cardiometabolic disease, strokes, all-cause mortality, and universally decreases lifespan.⁷⁰⁷¹⁷²⁷³⁷⁴⁷⁵⁷⁶⁷⁷

A recent study looking at 60 million Americans found that long term exposure to even low levels of fine particulate air pollution (levels below what the National Ambient Air Quality Standards consider safe) significantly increased the risk of premature death.⁷⁸

A later study looking at nearly 53 million older Medicare beneficiaries in the US found that long-term ambient fine particulate (PM2.5) exposure was associated with mortality from CVD, respiratory, and all cancer.⁷⁹ This study found that blacks, urban, and younger beneficiaries were most vulnerable to the long-term impacts of PM2.5 on mortality.

Air pollution overall is the leading environmental cause of global disease and premature death and causes adverse cardiorespiratory effects, and increased risk of cerebrovascular and neuropsychiatric disorders.⁸⁰

It's also been shown that air pollution affects the fetus and young children to the extent that it is detrimental to cognitive development, accelerated aging, and detrimental changes in the brain that have lifelong effects.⁸¹⁸²⁸³⁸⁴⁸⁵⁸⁶⁸⁷⁸⁸⁸⁹ Exposure to pollution, both indoor and outdoor, at any time but especially during gestation (the time between conception and birth), results in adverse epigenetic and transgenerational epigenetic changes that adversely affect both short and long term physical and mental processes.⁹⁰⁹¹⁹²⁹³⁹⁴⁹⁵⁹⁶⁹⁷⁹⁸⁹⁹¹⁰⁰¹⁰¹¹⁰²¹⁰³¹⁰⁴¹⁰⁵

A recent study found a small association between exposure to higher levels of air pollution in 1935, when participants were in utero, and a worse cognitive trajectory from age 11 years to age 70 years.¹⁰⁶

Indoor Air Pollution

Although most of us are aware of outdoor air pollution given the ubiquitous coverage of global warming due to vehicle, industrial, agricultural, personal and other contributions to air pollution, many of us are not as aware of indoor air pollution, both in our homes and indoor public places such as casinos, bars, restaurants, and other indoor establishments, that can be due to first, second, and third hand smoke, indoor use of chemicals, pesticides, building materials, mold, and even natural sources from the ground up such as radon gas.¹⁰⁷¹⁰⁸¹⁰⁹¹¹⁰¹¹¹¹¹²¹¹³¹¹⁴¹¹⁵¹¹⁶¹¹⁷¹¹⁸¹¹⁹

While some sources of indoor pollution are easily understood, many of which I've named above, others are not. For example, even the best of homes have unacceptable levels of airborne dust and particulate matter from many sources such as skin, insect matter and building material, as well as pollution from mold (present in almost all buildings and houses), including airborne spores, both dead and alive.¹²⁰¹²¹¹²²

American Thoracic Society 2017 INTERNATIONAL CONFERENCE

There were dozens of presentations on the adverse effects of pollution on health and disease at the American Thoracic Society 2017 INTERNATIONAL CONFERENCE held on MAY 19-24, 2017 in WASHINGTON, D.C.

Presentations and Topics included:

- Outdoor Air Pollution and Environmental Mitochondriomics Program Respiratory Health in Childhood R. Wright, MD, MPH, New York, NY.
- Biological Aging and Air Pollution A. Peters, MD, Munich, Germany.
- Relationship of Air Pollution to Sleep Disruption: The Multi-Ethnic Study of Atherosclerosis (MESA) Sleep and MESA-Air Studies/M.E. Billings, D.R. Gold, P.J. Leary, A. Szpiro, C.P. Aaron, J.D. Kaufman, S.S. Redline, Seattle, WA, p.A2930.
- Effect Modification by Differing Omega-6(n-6) to Omega-3 (n-3) Ratios on the Adverse Changes in Fibrinogen Associated with Ambient Particulate Air Pollution/D.P. Croft, S.J. Cameron, C.N. Morrell, C.J. Lowenstein, F. Ling, W. Zareba, P.K. Hopke, K. Thevenet-Morrison, S.W. Thurston, K. Evans, M.J. Utell, D. Chalupa, R. Block, D.Q. Rich, Rochester, NY, p.A6831.
- Several presentations under the umbrella: POLLUTION EFFECTS ON THE EPIGENOME IN THE DEVELOPMENT OF CHRONIC LUNG DISEASE Assemblies on Respiratory Cell and Molecular Biology; Allergy, Immunology and Inflammation; Environmental, Occupational and Population Health; Respiratory Structure and Function.
- Several presentations under the umbrella: AIR POLLUTION AND PSYCHOSOCIAL STRESS with an emphasis on recognizing how psychosocial stressors impact negative health outcomes associated with air pollution exposure.
- Epidemiologic and Epigenetic Evidence of Links Between Air Pollution, Stress and Negative Respiratory Health Outcomes J. Clougherty, MSc, ScD, Pittsburgh, PA.

One of the presentations, the Relationship of Air Pollution to Sleep Disruption, was on how air pollution disturbs sleep. The researchers analyzed data from 1,863 participants and found in the words of one of the authors of the study "that commonly experienced levels of air pollution not only affect heart and lung disease, but also sleep quality. Improving air quality may be one way to enhance sleep health and perhaps reduce health disparities."

Disrupted sleep patterns are counterproductive for those wanting to improve health, body composition, and physical and mental performance.¹²³¹²⁴¹²⁵¹²⁶¹²⁷¹²⁸¹²⁹¹³⁰¹³¹

As well, a recent paper found that environmental pollution is associated with increased risk of psychiatric disorders.¹³²

And then of course our lives are polluted by ubiquitous and addicting consumerism, sociopathic and corrupt politics, media sources including all forms of advertising that relentlessly inundate us with misinformation, cons, and lies, much of it in the name of marketing, but almost all of it in the name of greed.

Environ Res. 2020 Oct 29;110406. doi: [10.1016/j.envres.2020.110406](https://doi.org/10.1016/j.envres.2020.110406) PMID: 33130170. Online ahead of print.

Environmental exposures and sleep outcomes: A review of evidence, potential mechanisms, and implications

[Jianghong Liu](#)¹, [Lea Ghastine](#)², [Phoebe Um](#)², [Elizabeth Rovit](#)³, [Tina Wu](#)³

Abstract

Environmental exposures and poor sleep outcomes are known to have consequential effects on human health. This integrative review first seeks to present and synthesize existing literature investigating the relationship between exposure to various environmental factors and sleep health. We then present potential mechanisms of action as well as implications for policy and future research for each environmental exposure. Broadly, although studies are still emerging, empirical evidence has begun to show a positive association between adverse effects of heavy metal, noise pollution, light pollution, second-hand smoke, and air pollution exposures and various sleep problems. Specifically, these negative sleep outcomes range from subjective sleep manifestations, such as general sleep quality, sleep duration, daytime dysfunction, and daytime sleepiness, as well as objective sleep measures, including difficulties with sleep onset and maintenance, sleep stage or circadian rhythm interference, sleep arousal, REM activity, and sleep disordered breathing. However, the association between light exposure and sleep is less clear. Potential toxicological mechanisms are thought to include the direct effect of various environmental toxicants on the nervous, respiratory, and cardiovascular systems, oxidative stress, and inflammation. Nevertheless, future research is required to tease out the exact pathways of action to explain the associations between each environmental factor and sleep, to inform possible therapies to negate the detrimental effects, and to increase efforts in decreasing exposure to these harmful environmental factors to improve health.

Genomic, Epigenomic, and Phenotypic Effects of Pollution

Not only are we negatively affected by air pollution throughout our lives but it affects us even before we were born, as shown by a recent study.- <https://doi.org/10.1016/j.biopsych.2018.01.016>.

But even this doesn't go far enough because inherited epigenetic changes secondary to our parents and even grand-parents exposure to pollution can affect us both in a negative way but also contributes to the detrimental effects that pollution ordinarily affects us throughout our lifetime.¹³³¹³⁴¹³⁵¹³⁶

So, not only does pollution have detrimental effects on us both physically and mentally, but so does the detrimental effects of pollution suffered by our ancestors through transgenerational epigenetic changes.

But there's more. Studies have shown that the nuclear DNA and even mitochondrial DNA are also directly affected by pollution (besides the epigenetic changes as mentioned above) leading to mutations that negatively affects us in many ways.¹³⁷¹³⁸¹³⁹

Air pollution and molecular changes in age-related diseases

[B Hermanova](#)^{1,2}, [P Riedlova](#)^{1,2}, [A Dalecka](#)^{1,2}, [V Jirik](#)^{1,2}, [V Janout](#)¹, [R J Sram](#)¹

Int J Environ Health Res 2020 Jul 29;1-19. doi: 10.1080/09603123.2020.1797643. Online ahead of print.

PMID: 32723182. DOI: [10.1080/09603123.2020.1797643](https://doi.org/10.1080/09603123.2020.1797643)

Abstract

Assessment of the impact that air contaminants have on health is difficult as this is a complex mixture of substances that varies depending on the time and place. There are many studies on the association between air pollution and increased morbidity and mortality. Before the effect of polluted air is manifested at the level of the organs, an impact can be observed at the molecular level. These include some new biomarkers, like a shortening of the mean telomere length in DNA, dysregulation of gene expression caused by microRNA levels or a variation in the copy number of mitochondrial DNA. These changes may predispose individuals to premature development of age-related diseases and consequently to shortening of life. The common attribute, shared by changes at the molecular level and the development of diseases, is the presence of oxidative stress.

Environ Health Perspect. 2019 May;127(5):57012. doi: [10.1289/EHP4522](https://doi.org/10.1289/EHP4522). Epub 2019 May 31.

Prenatal Particulate Air Pollution and DNA Methylation in Newborns: An Epigenome-Wide Meta-Analysis

[Olena Gruzieva](#)^{1,2}, [Cheng-Jian Xu](#)^{3,4,5}, [Paul Yousefi](#)^{6,7}, [Caroline Relton](#)^{6,7}, [Simon Kebede Merid](#)¹, [Carrie V Breton](#)⁸, [Lu Gao](#)⁸, [Heather E Volk](#)^{9,10}, [Jason I Feinberg](#)⁹, [Christine Ladd-Acosta](#)¹¹, [Kelly Bakulski](#)¹¹, [Charles Auffray](#)¹², [Nathanaël Lemonnier](#)^{12,13}, [Michelle Plusquin](#)^{14,15}, [Akram Ghantous](#)¹⁶, [Zdenko Herceg](#)¹⁶, [Tim S Nawrot](#)^{14,17}, [Costanza Pizzi](#)¹⁸, [Lorenzo Richiardi](#)¹⁸, [Franca Rusconi](#)¹⁹, [Paolo Vineis](#)¹⁵, [Manolis Kogevinas](#)^{20,21}, [Janine F Felix](#)^{22,23}, [Liesbeth Duijts](#)^{22,24}, [Herman T den Dekker](#)^{22,23}, [Vincent W V Jaddoe](#)^{22,23}, [José L Ruiz](#)^{25,26}, [Mariona Bustamante](#)^{20,21,25}, [Josep Maria Antó](#)^{20,21,27}, [Jordi Sunyer](#)^{20,21,27}, [Martine Vrijheid](#)^{20,21}, [Kristine B Gutzkow](#)²⁸, [Regina Grazuleviciene](#)²⁹, [Carles Hernandez-Ferrer](#)^{20,30}, [Isabella Annesi-Maesano](#)³¹, [Johanna Lepeule](#)³², [Jean Bousquet](#)^{33,34}, [Anna Bergström](#)^{1,2}, [Inger Kull](#)^{1,35,36}, [Cilla Söderhäll](#)^{37,38}, [Juha Kere](#)^{38,39}, [Ulrike Gehring](#)⁴⁰, [Bert Brunekreef](#)^{40,41}, [Allan C Just](#)⁴², [Rosalind J Wright](#)⁴³, [Cheng Peng](#)⁴⁴, [Diane R Gold](#)^{44,45}, [Itai Kloog](#)⁴⁶, [Dawn L DeMeo](#)⁴⁴, [Göran Pershagen](#)^{1,2}, [Gerard H Koppelman](#)^{3,4}, [Stephanie J London](#)⁴⁷, [Andrea A Baccarelli](#)⁴⁸, [Erik Melén](#)^{1,36}

Abstract

Background: Prenatal exposure to air pollution has been associated with childhood respiratory disease and other adverse outcomes. Epigenetics is a suggested link between exposures and health outcomes.

Objectives: We aimed to investigate associations between prenatal exposure to particulate matter (PM) with diameter [Formula: see text] ([Formula: see text]) or [Formula: see text] ([Formula: see text]) and DNA methylation in newborns and children.

Methods: We meta-analyzed associations between exposure to [Formula: see text] ([Formula: see text]) and [Formula: see text] ([Formula: see text]) at maternal home addresses during pregnancy and newborn DNA methylation assessed by Illumina Infinium HumanMethylation450K BeadChip in nine European and American studies, with replication in 688 independent newborns and look-up analyses in 2,118 older children. We used two approaches, one focusing on single cytosine-phosphate-guanine (CpG) sites and another on differentially methylated regions (DMRs). We also related PM exposures to blood mRNA expression.

Results: Six CpGs were significantly associated [false discovery rate (FDR) [Formula: see text]] with prenatal [Formula: see text] and 14 with [Formula: see text] exposure. Two of the [Formula: see text] CpGs mapped to FAM13A (cg00905156) and NOTCH4 (cg06849931) previously associated with lung

function and asthma. Although these associations did not replicate in the smaller newborn sample, both CpGs were significant ([Formula: see text]) in 7- to 9-y-olds. For cg06849931, however, the direction of the association was inconsistent. Concurrent [Formula: see text] exposure was associated with a significantly higher NOTCH4 expression at age 16 y. We also identified several DMRs associated with either prenatal [Formula: see text] and or [Formula: see text] exposure, of which two [Formula: see text] DMRs, including H19 and MARCH11, replicated in newborns.

Conclusions: Several differentially methylated CpGs and DMRs associated with prenatal PM exposure were identified in newborns, with annotation to genes previously implicated in lung-related outcomes.

Full article in PDF format available at: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6792178/pdf/ehp-127-057012.pdf>.

[Sci Total Environ.](#) 2019 Mar 15;656:760-777. doi: 10.1016/j.scitotenv.2018.11.381. Epub 2018 Nov 28.

Air pollution associated epigenetic modifications: Transgenerational inheritance and underlying molecular mechanisms.

[Shukla A¹](#), [Bunkar N¹](#), [Kumar R¹](#), [Bhargava A¹](#), [Tiwari R¹](#), [Chaudhury K²](#), [Goryacheva IY³](#), [Mishra PK⁴](#).

Abstract

Air pollution is one of the leading causes of deaths in Southeast Asian countries including India. Exposure to air pollutants affects vital cellular mechanisms and is intimately linked with the etiology of a number of chronic diseases. Earlier work from our laboratory has shown that airborne particulate matter disturbs the mitochondrial machinery and causes significant damage to the epigenome. Mitochondrial reactive oxygen species possess the ability to trigger redox-sensitive signaling mechanisms and induce irreversible epigenomic changes. The electrophilic nature of reactive metabolites can directly result in deprotonation of cytosine at C-5 position or interfere with the DNA methyltransferases activity to cause alterations in DNA methylation. In addition, it also perturbs level of cellular metabolites critically involved in different epigenetic processes like acetylation and methylation of histone code and DNA hypo or hypermethylation. Interestingly, these modifications may persist through downstream generations and result in the transgenerational epigenomic inheritance. This phenomenon of subsequent transfer of epigenetic modifications is mainly associated with the germ cells and relies on the germline stability of the epigenetic states. Overall, the recent literature supports, and arguably strengthens, the contention that air pollution might contribute to transmission of epimutations from gametes to zygotes by involving mitochondrial DNA, parental allele imprinting, histone withholding and non-coding RNAs. However, larger prospective studies using innovative, integrated epigenome-wide metabolomic strategy are highly warranted to assess the air pollution induced transgenerational epigenetic inheritance and associated human health effects.

[Environ Int.](#) 2018 Feb 27;114:77-86. doi: 10.1016/j.envint.2018.02.014. [Epub ahead of print]

Epigenetics as a mechanism linking developmental exposures to long-term toxicity.

[Barouki R¹](#), [Melén E²](#), [Herceg Z³](#), [Beckers J⁴](#), [Chen J⁵](#), [Karagas M⁶](#), [Puga A⁷](#), [Xia Y⁷](#), [Chadwick L⁸](#), [Yan W⁹](#), [Audouze K¹⁰](#), [Slama R¹¹](#), [Heindel J¹²](#), [Grandjean P¹³](#), [Kawamoto T¹⁴](#), [Nohara K¹⁵](#).

Abstract

A variety of experimental and epidemiological studies lend support to the Developmental Origin of Health and Disease (DOHaD) concept. Yet, the actual mechanisms accounting for mid- and long-term effects of early-life exposures remain unclear. Epigenetic alterations such as changes in DNA methylation, histone modifications and the expression of certain RNAs have been suggested as possible mediators of long-term health effects of environmental stressors. This report captures discussions and conclusions debated during the last Prenatal Programming and Toxicity meeting held in Japan. Its first aim is to propose a number of criteria that are critical to support the primary contribution of epigenetics in DOHaD and intergenerational transmission of environmental stressors effects. The main criteria are the full characterization of the stressors, the actual window of exposure, the target tissue and function, the specificity of the epigenetic changes and the biological plausibility of the linkage between those changes and health outcomes. The second aim is to discuss long-term effects of a number of stressors such as smoking, air pollution and endocrine disruptors in order to identify the arguments supporting the involvement of an epigenetic mechanism. Based on the developed criteria, missing evidence and suggestions for future research will be identified. The third aim is to critically analyze the evidence supporting the involvement of epigenetic mechanisms in intergenerational and transgenerational effects of environmental exposure and to particularly discuss the role of placenta and sperm. While the article is not a systematic review and is not meant to be exhaustive, it critically assesses the contribution of epigenetics in the long-term effects of environmental exposures as well as provides insight for future research.

Full text in PDF format at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5899930/pdf/nihms947551.pdf>.

[FASEB J.](#) 2017 Jun;31(6):2241-2251. doi: 10.1096/fj.201601059RR. Epub 2017 Mar 9.

Epigenetic memory in response to environmental stressors.

[Vineis P](#)¹, [Chatziioannou A](#)², [Cunliffe VT](#)³, [Flanagan JM](#)⁴, [Hanson M](#)⁵, [Kirsch-Volders M](#)⁶, [Kyrtopoulos S](#)².

Abstract

Exposure to environmental stressors, toxicants, and nutrient deficiencies can affect DNA in several ways. Some exposures cause damage and alter the structure of DNA, but there is increasing evidence that the same or other environmental exposures, including those that occur during fetal development *in utero*, can cause epigenetic effects that modulate DNA function and gene expression. Some epigenetic changes to DNA that affect gene transcription are at least partially reversible (*i.e.*, they can be enzymatically reversed after cessation of exposure to environmental agents), but some epigenetic modifications seem to persist, even for decades. To explain the effects of early life experiences (such as famine and exposures to other stressors) on the long-term persistence of specific patterns of epigenetic modifications, such as DNA methylation, we propose an analogy with immune memory. We propose that an epigenetic memory can be established and maintained in self-renewing stem cell compartments. We suggest that the observations on early life effects on adult diseases and the persistence of methylation changes in smokers support our hypothesis, for which a mechanistic basis, however, needs to be further clarified. We outline a new model based on methylation changes. Although these changes seem to be mainly adaptive, they are also implicated in the pathogenesis and onset of diseases, depending on individual genotypic background and types of subsequent exposures. Elucidating the relationships between the adaptive and maladaptive consequences of the epigenetic modifications that result from complex environmental exposures is a major challenge for current and future research in

epigenetics.-Vineis, P., Chatziioannou, A., Cunliffe, V. T., Flanagan, J. M., Hanson, M., Kirsch-Volders, M., Kyrtopoulos, S. Epigenetic memory in response to environmental stressors.

[Epigenomics](#). 2015 Aug;7(5):829-46. doi: 10.2217/epi.15.36. Epub 2015 Sep 14.

Transgenerational epigenetic inheritance: adaptation through the germline epigenome?

[Prokopuk L](#)^{1,2}, [Western PS](#)^{1,2}, [Stringer JM](#)^{1,2}.

Abstract

Epigenetic modifications direct the way DNA is packaged into the nucleus, making genes more or less accessible to transcriptional machinery and influencing genomic stability. Environmental factors have the potential to alter the epigenome, allowing genes that are silenced to be activated and vice versa. This ultimately influences disease susceptibility and health in an individual. Furthermore, altered chromatin states can be transmitted to subsequent generations, thus epigenetic modifications may provide evolutionary mechanisms that impact on adaptation to changed environments. However, the mechanisms involved in establishing and maintaining these epigenetic modifications during development remain unclear. This review discusses current evidence for transgenerational epigenetic inheritance, confounding issues associated with its study, and the biological relevance of altered epigenetic states for subsequent generations.

[Prog Biophys Mol Biol](#). 2015 Jul;118(1-2):44-54. doi: 10.1016/j.pbiomolbio.2015.02.011. Epub 2015 Mar 16.

Elusive inheritance: Transgenerational effects and epigenetic inheritance in human environmental disease.

[Martos SN](#)¹, [Tang WY](#)², [Wang Z](#)³.

Abstract

Epigenetic mechanisms involving DNA methylation, histone modification, histone variants and nucleosome positioning, and noncoding RNAs regulate cell-, tissue-, and developmental stage-specific gene expression by influencing chromatin structure and modulating interactions between proteins and DNA. Epigenetic marks are mitotically inherited in somatic cells and may be altered in response to internal and external stimuli. The idea that environment-induced epigenetic changes in mammals could be inherited through the germline, independent of genetic mechanisms, has stimulated much debate. Many experimental models have been designed to interrogate the possibility of transgenerational epigenetic inheritance and provide insight into how environmental exposures influence phenotypes over multiple generations in the absence of any apparent genetic mutation. Unexpected molecular evidence has forced us to reevaluate not only our understanding of the plasticity and heritability of epigenetic factors, but of the stability of the genome as well. Recent reviews have described the difference between transgenerational and intergenerational effects; the two major epigenetic reprogramming events in the mammalian lifecycle; these two events making transgenerational epigenetic inheritance of environment-induced perturbations rare, if at all possible, in mammals; and mechanisms of transgenerational epigenetic inheritance in non-mammalian eukaryotic organisms. This paper briefly introduces these topics and mainly focuses on (1) transgenerational phenotypes and epigenetic effects in mammals, (2) environment-induced intergenerational epigenetic effects, and (3) the inherent difficulties in establishing a role for epigenetic inheritance in human environmental disease.

[BMC Med.](#) 2014 Sep 5;12:153. doi: 10.1186/s12916-014-0153-y.

Environmental stress and epigenetic transgenerational inheritance.

[Skinner MK.](#)

Abstract

Previous studies have shown a wide variety of environmental toxicants and abnormal nutrition can promote the epigenetic transgenerational inheritance of disease. More recently a number of studies have indicated environmental stress can also promote epigenetic alterations that are transmitted to subsequent generations to induce pathologies. A recent study by Yao and colleagues demonstrated gestational exposure to restraint stress and forced swimming promoted preterm birth risk and adverse newborn outcomes generationally. This ancestral stress promoted the epigenetic transgenerational inheritance of abnormalities in the great-grand offspring of the exposed gestating female. Several studies now support the role of environmental stress in promoting the epigenetic transgenerational inheritance of disease. Observations suggest ancestral environmental stress may be a component of disease etiology in the current population.

[ILAR J.](#) 2012;53(3-4):289-305. doi: 10.1093/ilar.53.3-4.289.

Environmental epigenetics and its implication on disease risk and health outcomes.

[Ho SM¹](#), [Johnson A](#), [Tarapore P](#), [Janakiram V](#), [Zhang X](#), [Leung YK](#).

Abstract

This review focuses on how environmental factors through epigenetics modify disease risk and health outcomes. Major epigenetic events, such as histone modifications, DNA methylation, and microRNA expression, are described. The function of dose, duration, composition, and window of exposure in remodeling the individual's epigenetic terrain and disease susceptibility are addressed. The ideas of lifelong editing of early-life epigenetic memories, transgenerational effects through germline transmission, and the potential role of hydroxymethylation of cytosine in developmental reprogramming are discussed. Finally, the epigenetic effects of several major classes of environmental factors are reviewed in the context of pathogenesis of disease. These include endocrine disruptors, tobacco smoke, polycyclic aromatic hydrocarbons, infectious pathogens, particulate matter, diesel exhaust particles, dust mites, fungi, heavy metals, and other indoor and outdoor pollutants. We conclude that the summation of epigenetic modifications induced by multiple environmental exposures, accumulated over time, represented as broad or narrow, acute or chronic, developmental or lifelong, may provide a more precise assessment of risk and consequences. Future investigations may focus on their use as readouts or biomarkers of the totality of past exposure for the prediction of future disease risk and the prescription of effective countermeasures.

And other recent studies, showing the effects of pollution on the genome and on genetic expression, something we already know about epigenetics, and how the genome is affected by the environment and thus changes genomic expression including causing epigenetic changes via alterations in microRNAs.¹⁴⁰¹⁴¹¹⁴²

MicroRNAs expression in relation to particulate matter exposure: A systematic review.

[Cheng M](#)¹, [Wang B](#)¹, [Yang M](#)¹, [Ma J](#)¹, [Ye Z](#)¹, [Xie L](#)¹, [Zhou M](#)¹, [Chen W](#)².

Abstract

MicroRNAs (miRNAs) are a class of small, non-coding RNAs with a post-transcriptional regulatory function on gene expression and cell processes, including proliferation, apoptosis and differentiation. In recent decades, miRNAs have attracted increasing interest to explore the role of epigenetics in response to air pollution. Air pollution, which always contains kinds of particulate matters, are able to reach respiratory tract and blood circulation and then causing epigenetics changes. In addition, extensive studies have illustrated that miRNAs serve as a bridge between particulate matter exposure and health-related effects, like inflammatory cytokines, blood pressure, vascular condition and lung function. The purpose of this review is to summarize the present knowledge about the expression of miRNAs in response to particulate matter exposure. Epidemiological and experimental studies were reviewed in two parts according to the size and source of particles. In this review, we also discussed various functions of the altered miRNAs and predicted potential biological mechanism participated in particulate matter-induced health effects. More rigorous studies are worth conducting to understand contribution of particulate matter on miRNAs alteration and the etiology between environmental exposure and disease development.

MicroRNA expression profiling in human acute organophosphorus poisoning and functional analysis of dysregulated miRNAs

[Haijun Yuan](#)¹, [Mei Yuan](#)², [Yonghong Tang](#)², [Biao Wang](#)¹, [Xiangyang Zhan](#)¹

Afr Health Sci 2018 Jun;18(2):333-342. doi: 10.4314/ahs.v18i2.18.

Abstract

Objective: Acute organophosphorus(OP) pesticide poisoning is associated with dysfunctions in multiple organs, especially skeletal muscles, the nervous system and the heart. However, little is known about the specific microRNA (miRNA) changes that control the pathophysiological processes of acute OP poisoning damage. We aimed to explore miRNA expression profiles and gain insight into molecular mechanisms of OP toxic effects.

Methods: MicroRNA expression was analyzed by TaqMan Human MicroRNA Array analysis and subsequent validated with quantitative PCR. The targets of the significantly different miRNAs were predicted with miRNA prediction databases, and pathway analysis of the predicted target genes was performed using bioinformatics resources.

Results: 37 miRNAs were significantly different in the sera of poisoned patients compared to the healthy controls, including 29 miRNAs that were up-regulated and 8 miRNAs that were down-regulated. Functional analysis indicated that many pathways potentially regulated by these miRNAs are involved in skeletal muscle, nervous system and heart disorders.

Conclusion: This study mapped changes in the serum miRNA expression profiles of poisoning patients and predicted functional links between miRNAs and their target genes in the regulation of acute OP

poisoning. Our findings are an important resource for further understanding the role of these miRNAs in the regulation of OP-induced injury.

To read the full study in PDF format go to:

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6306958/pdf/AFHS1802-0333.pdf>.

Organic Food?

Everything we consume is polluted to some extent. The problem is made worse by people who abuse terms like pesticide/insecticide free, organic, free range, grass fed, etc. none of which is true in the absolute sense, misleading at best and self-serving and outright lies at worst.

Commonly used advertising terms such as grass-fed beef and cows, free range chickens and eggs, non-GMO, natural ingredients and many others. While many are taken in by these terms to mean clean, unadulterated, and pollution free foods and products, such is not the case. However, it's impossible to verify that what they're saying about their products is true or is any better than other, less expensive products that don't use any of these terms.

For example, a recently published study concluded that "the consumption of organically produced meat does not diminish this carcinogenic risk, but on the contrary, it seems to be even higher, especially that associated with lamb consumption meat that the consumption of meat is labelled as organic."¹⁴³ (Hernández ÁR, Boada LD, Mendoza Z, et al. Consumption of organic meat does not diminish the carcinogenic potential associated with the intake of persistent organic pollutants (POPs). *Environ Sci Pollut Res Int*. 2017 Feb;24(5):4261-4273.)

Another recent study found that organic meat contained significantly higher levels of "Dioxins, PCBs, HBCD, Zn, Cu, Cd, Pb, As" than conventional meat.¹⁴⁴ (Dervilly-Pinel G, Guérin T, Minvielle B, et al. Micropollutants and chemical residues in organic and conventional meat. *Food Chem*. 2017 Oct 1;232:218-228.)

I've personally visited farms that advertise grass fed beef and dairy products but whose lands are close to major highways and polluting industries and whose lands still contain persistent pesticides and herbicides.

The biggest problem with farms that are close to heavy traffic (also all buildings, close to trafficked roads), is that even if the farms are able to decrease the use of pesticides, herbicides, and other obvious sources of pollution, there is significant pollution from many sources besides the obvious vehicle particulate and other air born emissions. These include air born emissions from wear and tear on brakes, tires and road surfaces, resuspended dust, and heavy metals.¹⁴⁵¹⁴⁶¹⁴⁷

I've also visited farms that claim free range chickens and eggs when in fact all that is provided is a maze leading to the outside, but which is never used by the chickens, making these chickens and eggs no better than any other products.

And just what does organic really mean. Does using animal waste for fertilizer really produce plants that are organic? It all depends on whether the animals were organic over several generations otherwise organic fertilizers can still be heavily polluted.

All the confusion surrounding the organic movement is mostly about greed and corruption because the providers, growers and manufacturers can charge more for the products since they can appeal to the increasing number of people who are trying to decrease the effects of pollution in their lives.

The only option that guarantees less pollution is having your environment tested and if everything is relatively free of pollution you can produce your own food. But that's an option that not many people can or wish to make as our lives are so hectic that it precludes us spending all the time and effort to do so.

Our environment is worsening by the minute but for the individual who is astute enough not to get caught up in it all, there is a solution, even an antidote, and that's opting out, feeling, thinking, and then acting on what you feel is right for you, cutting out all the negative energy, doing what you can that's reasonable about all the various forms of pollution and the state of the world.

It's really all about having control over your life, realizing that absolute control is impossible but having enough that you're comfortable with what you can control, and not bothered about what other people think about your choices. Once you've done that then you may even be able to be a rather lonely voice for change in the world, being involved in things you're comfortable with doing.

Body Pollution

While all these forms of pollution are important, the most important is what I call body pollution, pollution that directly affects our health, lifespan, healthspan, and not in the least, our anabolicspan.

Anabolicspan is a term that I coined to indicate that healthspan, being free of significant disease, is not enough. We should be aiming to retaining the ability to function mentally and physically as close as possible to when we were in our prime, working and playing without significant impairment, and staying vital and strong as close to your lifespan as possible.

Body pollution, to one extent or another, is inescapable as it's all around us, in the air we breathe, the food we eat, the water we drink and as importantly the choices we make in polluting our bodies by choice (alcohol, smoking in all its forms including second and third hand smoke,¹⁴⁸ drugs, etc.).

All the earth is polluted to one extent or another. For example, all of earth's air is polluted, although there's a difference in the type and amount of pollution depending on where you live. The effects of air pollution on our physical and mental health also depends on where you live but also how you live.

Some forms of pollution are much more prevalent in third world countries where for example potable water may be scarce and can be contaminated by waste that are high in pollutants, and disease-causing organisms such as bacteria and parasites.

Some forms are more prevalent in industrialized countries such as chemical pollution of our water, soil contamination by industrial wastes and other chemicals such as pesticides and herbicides, and air pollution by industry and more importantly by motor vehicle emissions.

Pollution of water, soil and air are connected in that pollution from one source pollutes the others. For example, pollutants in the air contribute to water and soil pollution as the pollutants settle and especially when driven by rain. Pollution in the soil can be driven into the air by wind, and into water by rain or irrigation.

Pollution is an escalating phenomenon in concert with the earth's rising carbon footprint. There are many reasons why, including the relentless increase in earth's population and consumption, especially in the emerging third world countries.

Can we solve the pollution problem before it overwhelms us? Unfortunately, it's likely that pollution will increase despite the convoluted and politically complex attempts to decrease it. After all we're a

consumer society and there's more of us every day that demand goods and services that now are mostly consumed by the industrial "first world" countries, consisting of a smaller percentage of the world's population as compared to third world countries.

How can it be otherwise when capitalist globalization is taking over the world? The world population and consumerism are increasing by the minute and along with it, the rising and over the next decade the exponential need for essential and non-essential goods and services.

And there's very little good news in studies that document decreased pollution in some US cities, for example Los Angeles, as it matters little if small pockets of the earth decrease pollution, and even this so-called lowered levels of pollution may be illusionary than real. Decreasing one form of pollution, such as nitric oxide, may in fact lead to the increase in other forms of pollution.¹⁴⁹

The bottom line of course is that what's important is the global pollution load, which is constantly increasing, rather than decreases in the relatively small regional, mostly urban, areas of the world.

Other Underappreciated Sources of Body Pollution

There are several sources of body pollution that are under appreciated. For example, a recent study found that our vehicles can be a significant source of pollution but not in the usual way we think of vehicle pollution. It's been known that chemicals originating from the inside of vehicles and those introduced (for example smoking cigarettes and e-cigs) are significant sources of body pollution.¹⁵⁰

While there are many forms of pollution originating from the inside of a vehicle one of the underappreciated are flame retardants. For example, their use in many areas of vehicles, although mostly in the foam of vehicle seats. A study published this year found that the longer you drive, even if it's only a week, the more you're exposed to flame retardant, which is carcinogenic and increases morbidity and mortality – see info and abstract below and other abstracts of interest.¹⁵¹

A recent study found that we are not even all that safe when using sunscreen, something most of us have used and some use almost daily. It seems that the ingredients of sunscreens don't just stay on the skin but are absorbed into the body. The systemic absorption is sure to cause body pollution in one way or another.¹⁵² I've copied the full text of that study below for those interested in the details and discussions on the good, bad and the ugly of sunscreens.

But it doesn't stop there. I'm sure that anything we use on our hair or skin, especially potential toxins and preservatives that are widely used in these products, gets absorbed to one extent or another into our bodies.¹⁵³ We've yet to know how these chemicals found in insect repellants, and various shampoos, soaps, deodorant products, etc. affect body pollution but I'm sure that it can't be good for us.

For example, a study published this year found that parabens (endocrine disrupting compounds with estrogenic effects used as preservatives in cosmetics including creams and body lotions) if used by pregnant women can lead to overweight in their children.¹⁵⁴

Another source of increased exposure to air pollution is in all areas that are subject to increased air pollution but are relatively closed off and under ventilated. An example is the significant increased exposure to air pollution in subways systems.¹⁵⁵

Cigarettes and Electronic Cigarettes

Cigarettes are a significant form of body pollution that leads to increased morbidity and mortality. Electronic cigarettes are touted as a better solution to smoking although it's questionable that they are

given the amount of pollutants in the super-heated e-cigs. But mostly that's the greed talking and trying to convince us that e-cigs are OK to use as they're relatively harmless. But it's all lies.

E-cigs are toxic for those that smoke them, due not only to the nicotine but also the pollutants and toxins involved in inhaling flavor components and thermal decomposition products even if nicotine is absent.¹⁵⁶¹⁵⁷¹⁵⁸¹⁵⁹ E-cigs are also toxic for those who are exposed to secondhand e-cig smoke and even those that are exposed to third hand e-cig smoke on the clothes and surroundings of the smokers.¹⁶⁰¹⁶¹¹⁶²¹⁶³¹⁶⁴¹⁶⁵¹⁶⁶

What Can Be Done?

Not much on a grand scale. Most plans and treatments for decreasing environmental pollution are meant for dealing with the present problems and may have had merit if population, technology along with their use of resources and the resulting increased pollution and waste, wasn't dramatically escalating. But as it is no matter what any local or even world governments and institutions plan to deal with today's pollution, it won't be enough to stop the increase in the level of pollution that's out of control on a global scale.

It might be possible if we could all just consume and buy what we need rather than what we are made to want by the overwhelming ubiquitous marketing and advertising that is fueled by consumer capitalistic greed. But that's not going to happen as any measures to control and even decrease pollution will be tainted by political and personal agendas. See my newly updated article [Lies, Lies and Damn Lies](#) for more info.

However, we can do a lot on an individual level if we have the conviction to make the small and large changes that need to be made.

Most of us are destined to be passive nihilist as far as the environment and give up personal responsibility to a higher power, be it government, science, religion, industry, or money hoarding multi-millionaires and billionaires. Refusing to accept responsibility for pollution and all that it represents can even be considered stoic. After all there's nothing you can do as one person, so let's let it go its course and deal with issues that we can do something about.

But negating pollution as a personal issue, with token participation for example by separating recycling and waste in the garbage you put curbside every week, doesn't make a dent on global pollution and its malignant consequence which are surely coming. Having an uncaring attitude about the way the world is evolving is morally unacceptable even if your philosophy on life and death mirror the existentialist and nihilists view that life is essentially meaningless and absurd (Nietzsche, Sartre, Camus, and many other existentialist philosophers).

You can accept what's happening or you can make whatever changes you can make to protect yourself against the effects of the rising tide of pollution. As well, you can be mad as hell and not take it anymore and go against the flow. One of the biggest impacts you can make is to deny rampant consumerism by simply not buying anything without first considering whether you really need it or need it as a fix.

Everything and everyone around us encourages, compels and manipulates us to buy, buy, buy, and use more and more when in fact we need to resist the urges of rampant capitalistic consumerism that is mostly fueled by the raw and pathological greed (inspired by the survive at all costs Darwinian curse) of the already rich.

Your motto should be to simply buy and consume what's absolutely essential and no more, regardless of the relentless exposure and pressure, subtle or otherwise, that we're subjected to in all facets of our lives.

But to deal with present levels of pollution you need to also get your own universe in order by making personal changes that will moderate the adverse epigenetic and subsequent metabolic, life-changing effects caused by pollution.

For example, you can filter your drinking and even bathing water, use natural ingredients for dealing with your personal space and property, buy organic, natural food as much as possible (even though, as mentioned above, there is inevitably some degree of pollution in anything labelled organic, if indeed it is organic, simply because of the ubiquitous prevalence of pollution) or even better produce as much of your own food as you're comfortable doing, avoid cities as much as possible, and drive only when you have to in order to decrease as much as you can the use of motor vehicles, which pollute both our external and internal environments (see above about the inherent internal pollution in our vehicles).

You don't have to be a minimalist, a stoic, an anti-consumer or adopt any form of extremist lifestyle. After all, to live you must consume. It's excessive consumerism that's the problem and it's fueled by the ubiquitous marketing that makes us buy not what we really need but what the marketing that fuels not our needs but our wants.

All that's required is that you are aware of all the forces that try to manipulate you to take on a lifestyle that really isn't you. You can experiment with ridding yourself of excessive consumerism simply to relieve an itch created by manipulative marketing.

Some Easy Ways to Decrease Exposure to Pollutants Including Endocrine Disrupting Chemicals

****change or put elsewhere**** As we saw above, flame retardants are ubiquitous in our environment. While we may not be able as much to as much to decrease pollution in our external environment, we can decrease levels of pollutants in our homes.

There are some pretty basic steps you can take to avoid pollutants in our homes including chemicals that cause havoc to our endocrine system and disrupt the proper functioning of gonadal and other hormones, including testosterone, estrogen, thyroid and adrenal hormones. The disruption of these hormones can lead to all manner of diseases as well as adversely impacting body composition and mental and physical performance.

Here are some of them:

- Don't purchase anything that contains toxic flame retardants, regardless of the type used as they're all dangerous pollutants.
- Since all types of organic pollutants from our furniture, electronics, and numerous other products, and even radon gas, accumulate in our homes I suggest you air out your home as often as it's feasible including crawlspaces. As well, since household dust contain these pollutants including possible mold spores, vacuum your home as often as is feasible, preferably using a HEPA filtered vacuum cleaner, and also mop the floors to get rid of residual dust and other particles that your vacuum cleaner doesn't pick up.

- Use an efficient furnace air filter and clean or change the filter as required for your particular system.
- Look for all areas that may have mold and remove mold as well as making sure to correct any problems that encourage the formation of mold.
- Don't use anything but cast iron or stainless-steel cookware or utensils. Especially avoid nonstick cookware which contain perfluoroalkyl substances (PFAS). Those that state that their products are "PFAS free" will invariably contain similar compounds that are just as dangerous.
- Be careful of any foods that are in contact with plastics (phthalates are commonly found in plastic or plastic based food packaging and containers and can have adverse effects on body composition) and don't use plastics of any kind in a microwave even if the manufacturer states that it's "microwave safe."
- Avoid canned foods since they all contain bisphenols. If the manufacturer says it's BPA free you can be sure it contains other bisphenols which are just as dangerous.
- Be careful of any liquids, including water, that you drink to make sure they're as pollutant free as possible, including chlorine treated water in your home that can pollute not just in drinking the water but using the water for personal hygiene.
- While many people think eating organic will help decrease pollution both in ourselves and in our environment, that may not be the case as discussed in this article under organic foods.
- Since vehicles also pollute internally (see above), keep your use of them as much as is possible.
- Keep away as much as possible from spaces where air pollution builds up and is not properly vented away, such as subways.
- Keep noise pollution to a minimum in any way you can including making changes to your house to decrease outside noise levels, buying household items that make less noise – for example fans that operate relatively silent, and if necessary using earplugs at night and even during the day.
- There are several other sources of indoor air pollution that you should be aware of and that are under your control. For example, jumping on beds, not airing out your home, and indoor pets, especially furry pets (grooming them frequently is important).¹⁶⁷
- Also using a laser printer can expose you to microscopic toner nanoparticles that increase inflammation and can increase your susceptibility to various diseases. waft from laser printers may change our genetic and metabolic profiles in ways that make disease more likely.¹⁶⁸ The solution is to air out your home, especially your office, whenever possible. Also, you may want to get a wireless printer that is in a room that you're not in very often.

Decreasing Body Pollution with Specific Nutritional Supplementation

Pollution, and especially particulate matter in air pollution (most coming from motor vehicle exhaust and especially diesel exhaust mainly from trucks), is counter-productive for physiological and psychological health, body composition, and exercise/athletic performance.¹⁶⁹¹⁷⁰

Aerobic training in urban environments, both indoor and outdoor, and in rural environments under certain conditions, such as living or training close to pollution heavy manufacturing facilities and roads with moderate to high traffic and major highways, increases inflammation and decreases the beneficial effects of exercise on health, body composition, and performance.¹⁷¹¹⁷²¹⁷³¹⁷⁴

A recent meta-analysis study found that all of the relevant studies “found air pollution level to be negatively associated with physical activity and positively associated with leisure-time physical inactivity. Study participants, and particularly those with respiratory disease, self-reported a reduction in outdoor activities to mitigate the detrimental impact of air pollution.”¹⁷⁵

- On the other hand, studies have shown that regular exercise can mitigate some of the harmful effects of pollution and that even exercising in a polluted environment can be better for you than not exercising at all. For more information on when and where pollution overrides the beneficial effects of exercise see:
- <https://www.theguardian.com/cities/2017/feb/13/tipping-point-cities-exercise-more-harm-than-good>,
- <https://www.smithsonianmag.com/smart-news/walking-polluted-areas-reduces-benefits-exercise-180967434/>
- <https://www.mayoclinic.org/healthy-lifestyle/fitness/expert-answers/air-pollution-and-exercise/faq-20058563>
- <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4666455/>,

MetabolicDiet.com Nutritional Supplement Products for Decreasing the Adverse Effects of Pollution

Many of MVM’s ingredients, and dosages, are useful for decreasing the adverse effects of pollution on our health, body composition, and physical and mental performance.

For example, recent papers have found that high doses of certain B vitamins (as found in MVM but not in most of other vitamin mineral supplements) decrease the adverse effects of both short term and long term fine particle pollution on the cardiovascular, neurological, and immune systems by modulating the epigenetic modifications that occur secondary to environmental pollution.¹⁷⁶¹⁷⁷

As well, several papers have documented the beneficial cardiovascular, neurological and immune systems, and effects on reducing inflammation (from pollution and other causes) of other ingredients in MVM including **vitamin D, green tea polyphenol (-)-epigallocatechin-3-gallate, resveratrol (in grape seed extract), curcumin, quercetin, lipoic acid, astaxanthin, lutein, coenzyme Q10, L-carnitine, and many other flavonoids, polyphenols and extracts.**¹⁷⁸¹⁷⁹¹⁸⁰¹⁸¹¹⁸²¹⁸³¹⁸⁴¹⁸⁵¹⁸⁶¹⁸⁷¹⁸⁸¹⁸⁹¹⁹⁰

EFA+ has a host of beneficial effects including decreasing inflammation and improving body composition and performance. It also helps to protect against the adverse effects of air pollution, including from industrial pollution, motor vehicle exhaust, and cigarette smoke.¹⁹¹¹⁹²¹⁹³¹⁹⁴

Antiox is the prime supplement for combating oxidative stress, especially against the effects of pollution and intensive exercise. I’ll cover that topic in detail in an upcoming article.

Click on the relevant links for complete information on [MVM](#), [EFA+](#), and [Antiox](#). Each product is available in the shop at <https://metabolicdiet.com/shop/>.

Most of the other nutritional supplements in my lineup also help in decreasing the burden of world pollution. For example, the resveratrol, B vitamins, and CoQ10 (see below) in TestoBoost can counteract some of the detrimental effects of environmental pollution including first, second, and third

hand cigarette smoke and air pollution. You can download my eBook on [TestoBoost](#) for more information.

Coenzyme Q10 (CoQ10)

I recently wrote a detailed article on CoQ10 that is available here. For this article I'll just mention a few specifics on how it reduces pollution's harmful effects.

Coenzyme Q10 (CoQ10), a coenzyme that is ubiquitous in animals, including humans, is a lipid-soluble antioxidant and acting as an electron carrier is a key component of the mitochondrial electron transport chain for adenosine triphosphate (ATP) production.¹⁹⁵

CoQ10 is also one of the key antioxidant nutrients that protect us from pollution induced damage (such as free radical-induced oxidative damage and inflammatory cytokines) to our systems including mitochondrial membrane lipids and proteins and mitochondrial DNA.

CoQ10 supplementation has been shown to have anti-aging and beneficial effects on semen parameters, fertility, testicular damage, and reproductive hormones including testosterone.¹⁹⁶¹⁹⁷¹⁹⁸¹⁹⁹²⁰⁰²⁰¹²⁰²²⁰³²⁰⁴

In a recent study CoQ10 while not found to directly increase testosterone, CoQ10 supplementation **“was found to ameliorate the reduction in testosterone induced by chemical reproductive toxicants, mainly by neutralizing the damaging effect of the generated free radicals.”**²⁰⁵ **In a study published this year the authors concluded that “CoQ10 may counteract BPA-induced reprotoxicity.”**²⁰⁶

[Genetics](#). 2020 Feb;214(2):381-395. doi: 10.1534/genetics.119.302939. Epub 2019 Dec 18.

Antioxidant CoQ10 Restores Fertility by Rescuing Bisphenol A-Induced Oxidative DNA Damage in the *Caenorhabditis elegans* Germline.

[Hornos Carneiro MF](#)^{#1,2}, [Shin N](#)^{#1}, [Karthikraj R](#)³, [Barbosa F Jr](#)², [Kannan K](#)^{3,4}, [Colaiácovo MP](#)⁵.

Abstract

Endocrine-disrupting chemicals are ubiquitously present in our environment, but the mechanisms by which they adversely affect human reproductive health and strategies to circumvent their effects remain largely unknown. Here, we show in *Caenorhabditis elegans* that supplementation with the antioxidant Coenzyme Q10 (CoQ10) rescues the reprotoxicity induced by the widely used plasticizer and endocrine disruptor bisphenol A (BPA), in part by neutralizing DNA damage resulting from oxidative stress. CoQ10 significantly reduces BPA-induced elevated levels of germ cell apoptosis, phosphorylated checkpoint kinase 1 (CHK-1), double-strand breaks (DSBs), and chromosome defects in diakinesis oocytes. BPA-induced oxidative stress, mitochondrial dysfunction, and increased gene expression of antioxidant enzymes in the germline are counteracted by CoQ10. Finally, CoQ10 treatment also reduced the levels of aneuploid embryos and BPA-induced defects observed in early embryonic divisions. We propose that CoQ10 may counteract BPA-induced reprotoxicity through the scavenging of reactive oxygen species and free radicals, and that this natural antioxidant could constitute a low-risk and low-cost strategy to attenuate the impact on fertility by BPA.

CoQ10 is an ingredient in several products in my nutritional supplement lineup at www.MetabolicDiet.com/Shop, including (in alphabetical order):

- [Amino](#)
- [Antiox](#)
- [Creatine Advantage](#)

- [GHboost](#)
- [InControl](#)
- [Joint Support](#)
- [LipoFlush](#)
- [MRP LoCarb](#)
- [MVM](#)
- [ReNew](#)
- [Resolve](#)
- [TestoBoost](#)

L-carnitine and Acetyl-L-carnitine

CoQ10 works additively or synergistically with other nutrients to counter the effects of pollution including those found in MVM and EFA+ (see above) as well as **L-carnitine** and **acetyl-l-carnitine** (the interchangeable acetyl form of L-carnitine). For example, studies have shown that under certain conditions CoQ10 plus L-carnitine significantly increases total antioxidant, LH and testosterone levels as well as improving semen parameters.²⁰⁷²⁰⁸

However, L-carnitine and acetyl-L-carnitine together or on their own (even though they are metabolically interchangeable they also have individual specific effects) also have significant anti-oxidative and anti-inflammatory effect as shown by their effects on optimizing testosterone levels and other anabolic effects.²⁰⁹²¹⁰²¹¹²¹²²¹³²¹⁴²¹⁵

Because of their beneficial effects on body composition, energy metabolism, and physical and mental performance, L-carnitine and acetyl-L-carnitine either on their own or together are found in almost all of the products in my nutritional supplement lineup.

I've included partial and full text of some interesting papers and articles below.

[Antioxid Redox Signal](#). 2018 Mar 20;28(9):735-740. doi: 10.1089/ars.2017.7488. Epub 2018 Feb 2.

Environmental Stressors and Their Impact on Health and Disease with Focus on Oxidative Stress.

[Münzel T](#)¹, [Daiber A](#)¹.

[Abstract](#)

Epidemiological, preclinical and interventional clinical studies have demonstrated that environmental stressors are associated with health problems, namely cardiovascular diseases. According to estimations of the World Health Organization (WHO), environmental risk factors account for an appreciable part of global deaths and life years spent with disability. This Forum addresses the impact of the environmental risk factors such as traffic noise exposure, air pollution by particulate matter (PM), mental stress/loneliness, and the life style risk factor (water-pipe) smoking on health and disease with focus on the cardiovascular system. We will critically discuss the use of observatory/modifiable biomarkers of oxidative stress and inflammation in environmental research on the aforementioned risk factors highlighting the need of exposome studies. Another focus will be on the epigenetic regulation via microRNAs in

environmental stress upon exposure to noise and toxins/heavy metals as well as mental stress conditions, providing mechanistic insights into the modulation of microRNA signaling by oxidative stress, and vice versa the contribution of microRNAs to oxidative stress conditions. We will also provide an in-depth overview on the mechanistic pathways that lead to health problems (e.g., cardiovascular diseases) in response to environmental psychosocial stress, air pollution exposure in the form of ambient PM and diesel exhaust, traffic noise exposure, and the life style drug (water-pipe) smoking. Almost all stressors share the activation of the hypothalamic-pituitary-adrenocortical axis and of the sympathetic nervous system with subsequent onset of inflammation and oxidative stress, defining the here proposed therapeutic (antioxidant and exercise) strategies. *Antioxid. Redox Signal.* 28, 735-740.

Click on the relevant links for complete information on [MVM](#), [EFA+](#), and [Antiox](#). Each product is available in the shop at <https://metabolicdiet.com/shop/>.

Reports, Citations and Abstracts of Interest

[Lancet](#). 2018 Jan 27;391(10118):339-349. doi: 10.1016/S0140-6736(17)32643-0. Epub 2017 Dec 5.

Respiratory and cardiovascular responses to walking down a traffic-polluted road compared with walking in a traffic-free area

in participants aged 60 years and older with chronic lung or heart disease and age-matched healthy controls: a randomised, crossover study.

[Sinharay R](#)¹, [Gong J](#)², [Barratt B](#)³, [Ohman-Strickland P](#)⁴, [Ernst S](#)⁵, [Kelly FJ](#)³, [Zhang JJ](#)², [Collins P](#)¹, [Cullinan P](#)¹, [Chung KF](#)⁶.

Erratum in - [Department of Error](#). [Lancet. 2018]

Abstract

BACKGROUND:

Long-term exposure to pollution can lead to an increase in the rate of decline of lung function, especially in older individuals and in those with chronic obstructive pulmonary disease (COPD), whereas shorter-term exposure at higher pollution levels has been implicated in causing excess deaths from ischaemic heart disease and exacerbations of COPD. We aimed to assess the effects on respiratory and cardiovascular responses of walking down a busy street with high levels of pollution compared with walking in a traffic-free area with lower pollution levels in older adults.

METHODS:

In this randomised, crossover study, we recruited men and women aged 60 years and older with angiographically proven stable ischaemic heart disease or stage 2 Global initiative for Obstructive Lung Disease (GOLD) COPD who had been clinically stable for 6 months, and age-matched healthy volunteers. Individuals with ischaemic heart disease or COPD were recruited from existing databases or outpatient respiratory and cardiology clinics at the Royal Brompton & Harefield NHS Foundation Trust and age-matched healthy volunteers using advertising and existing databases. All participants had abstained from smoking for at least 12 months and medications were taken as recommended by participants' doctors during the study. Participants were randomly assigned by drawing numbered disks at random from a bag to do a 2 h walk either along a commercial street in London (Oxford Street) or in an urban park (Hyde Park).

Baseline measurements of participants were taken before the walk in the hospital laboratory. During each walk session, black carbon, particulate matter (PM) concentrations, ultrafine particles, and nitrogen dioxide (NO₂) concentrations were measured.

FINDINGS:

Between October, 2012, and June, 2014, we screened 135 participants, of whom 40 healthy volunteers, 40 individuals with COPD, and 39 with ischaemic heart disease were recruited. Concentrations of black carbon, NO₂, PM₁₀, PM_{2.5}, and ultrafine particles were higher on Oxford Street than in Hyde Park. Participants with COPD reported more cough (odds ratio [OR] 1.95, 95% CI 0.96-3.95; p<0.1), sputum (3.15, 1.39-7.13; p<0.05), shortness of breath (1.86, 0.97-3.57; p<0.1), and wheeze (4.00, 1.52-10.50; p<0.05) after walking down Oxford Street compared with Hyde Park. In all participants, irrespective of their disease status, walking in Hyde Park led to an increase in lung function (forced expiratory volume in the first second [FEV₁] and forced vital capacity [FVC]) and a decrease in pulse wave velocity (PWV) and augmentation index up to 26 h after the walk. By contrast, these beneficial responses were attenuated after walking on Oxford Street. In participants with COPD, a reduction in FEV₁ and FVC, and an increase in R5-20 were associated with an increase in during-walk exposure to NO₂, ultrafine particles and PM_{2.5}, and an increase in PWV and augmentation index with NO₂ and ultrafine particles. In healthy volunteers, PWV and augmentation index were associated both with black carbon and ultrafine particles.

INTERPRETATION:

Short-term exposure to traffic pollution prevents the beneficial cardiopulmonary effects of walking in people with COPD, ischaemic heart disease, and those free from chronic cardiopulmonary diseases. Medication use might reduce the adverse effects of air pollution in individuals with ischaemic heart disease. Policies should aim to control ambient levels of air pollution along busy streets in view of these negative health effects.

[Perspect Public Health](#). 2018 Mar;138(2):111-121. doi: 10.1177/1757913917726567. Epub 2017 Aug 22.

Impact of ambient air pollution on physical activity among adults: a systematic review and meta-analysis.

[An R](#)¹, [Zhang S](#)², [Ji M](#)¹, [Guan C](#)³.

Abstract

AIMS:

This study systematically reviewed literature regarding the impact of ambient air pollution on physical activity among children and adults.

METHODS:

Keyword and reference search was conducted in PubMed and Web of Science to systematically identify articles meeting all of the following criteria - study designs: interventions or experiments, retrospective or prospective cohort studies, cross-sectional studies, and case-control studies; subjects: adults; exposures: specific air pollutants and overall air quality; outcomes: physical activity and sedentary behaviour; article types: peer-reviewed publications; and language: articles written in English. Meta-analysis was performed to estimate the pooled effect size of ambient PM_{2.5} air pollution on physical inactivity.

RESULTS:

Seven studies met the inclusion criteria. Among them, six were conducted in the United States, and one was conducted in the United Kingdom. Six adopted a cross-sectional study design, and one used a prospective cohort design. Six had a sample size larger than 10,000. Specific air pollutants assessed included PM_{2.5}, PM₁₀, O₃, and NO_x, whereas two studies focused on overall air quality. All studies found air pollution level to be negatively associated with physical activity and positively associated with leisure-time physical inactivity. Study participants, and particularly those with respiratory disease, self-reported a reduction in outdoor activities to mitigate the detrimental impact of air pollution. Meta-analysis revealed a one unit (µg/m³) increase in ambient PM_{2.5} concentration to be associated with an increase in the odds of physical inactivity by 1.1% (odds ratio = 1.011; 95% confidence interval = 1.001, 1.021; p-value < .001) among US adults.

CONCLUSIONS:

Existing literature in general suggested that air pollution discouraged physical activity. Current literature predominantly adopted a cross-sectional design and focused on the United States. Future studies are warranted to implement a longitudinal study design and evaluate the impact of air pollution on physical activity in heavily polluted developing countries.

[Environ Sci Pollut Res Int](#). 2019 Mar 22. doi: 10.1007/s11356-019-04874-z. [Epub ahead of print]

A comprehensive evaluation of the association between ambient air pollution and adverse health outcomes of major organ systems: a systematic review with a worldwide approach.

[Bazyar J](#)¹, [Pourvakhshoori N](#)¹, [Khankeh H](#)¹, [Farrokhi M](#)², [Delshad V](#)¹, [Rajabi E](#)¹.

Abstract

Ambient air pollution is nowadays one of the most crucial contributors to deteriorating health status worldwide. The components of air pollution include PM_{2.5} and PM₁₀, NO₂, SO₂, CO, O₃, and organic compounds. They are attributed to several health outcomes, for instance, cardiovascular diseases (CVD), respiratory diseases, birth outcomes, neurologic diseases, and psychiatric diseases. The objective of this study is to evaluate the association between different ambient air pollutants and the above-mentioned health outcomes. In this systematic review, a total of 76 articles was ultimately selected from 2653 articles, through multiple screening steps by the aid of a set of exclusion criteria as non-English articles, indoor air pollution assessment, work-related, occupational and home-attributed pollution, animal studies, tobacco smoking effects, letters to editors, commentaries, animal experiments, reviews, case reports and case series, out of 19,862 published articles through a systematic search in PubMed, Web of Science, and Scopus. Then, the associations between air pollution and different health outcomes were measured as relative risks and odds ratios. The association between air pollutants, PM_{2.5} and PM₁₀, NO₂, SO₂, CO, O₃, and VOC with major organ systems health was investigated through the gathered studies. Relative risks and/or odds ratios attributed to each air pollutant/outcome were ultimately reported. In this study, a thorough and comprehensive discussion of all aspects of the contribution of ambient air pollutants in health outcomes was proposed. To our knowledge up to now, there is no such comprehensive outlook on this issue. Growing concerns in concert with air pollution-induced health risks impose a great danger on the life of billions of people worldwide. Should we propose ideas and schemes to reduce ambient air pollutant, there will be dramatic reductions in the prevalence and occurrence of health-threatening conditions.

[PLoS Biol.](#) 2019 Aug 20;17(8):e3000353. doi: 10.1371/journal.pbio.3000353. eCollection 2019 Aug.

Environmental pollution is associated with increased risk of psychiatric disorders in the US and Denmark.

[Khan A](#)¹, [Plana-Ripoll O](#)^{2,3,4}, [Antonsen S](#)^{2,3,4}, [Brandt J](#)⁵, [Geels C](#)⁵, [Landecker H](#)⁶, [Sullivan PF](#)^{7,8}, [Pedersen CB](#)^{2,3,4,9}, [Rzhetsky A](#)^{1,10}.

Abstract

The search for the genetic factors underlying complex neuropsychiatric disorders has proceeded apace in the past decade. Despite some advances in identifying genetic variants associated with psychiatric disorders, most variants have small individual contributions to risk. By contrast, disease risk increase appears to be less subtle for disease-predisposing environmental insults. In this study, we sought to identify associations between environmental pollution and risk of neuropsychiatric disorders. We present exploratory analyses of 2 independent, very large datasets: 151 million unique individuals, represented in a United States insurance claims dataset, and 1.4 million unique individuals documented in Danish national treatment registers. Environmental Protection Agency (EPA) county-level environmental quality indices (EQIs) in the US and individual-level exposure to air pollution in Denmark were used to assess the association between pollution exposure and the risk of neuropsychiatric disorders. These results show that air pollution is significantly associated with increased risk of psychiatric disorders. We hypothesize that pollutants affect the human brain via neuroinflammatory pathways that have also been shown to cause depression-like phenotypes in **animal studies**.

Effect of Sunscreen Application Under Maximal Use Conditions on Plasma Concentration of Sunscreen Active Ingredients: A Randomized Clinical Trial

Murali K. Matta, PhD¹; Robbert Zusterzeel, MD, PhD, MPH¹; Nageswara R. Pilli, PhD¹; et al Vikram Patel, PhD¹; Donna A. Volpe, PhD¹; Jeffry Florian, PhD¹; Luke Oh, PhD²; Edward Bashaw, PharmD²; Issam Zineh, PharmD, MPH²; Carlos Sanabria, MD³; Sarah Kemp, RN³; Anthony Godfrey, PharmD³; Steven Adah, PhD⁴; Sergio Coelho, PhD⁴; Jian Wang, PhD⁵; Lesley-Anne Furlong, MD⁵; Charles Ganley, MD⁵; Theresa Michele, MD⁴; David G. Strauss, MD, PhD¹

Author Affiliations [Article Information](#)

JAMA. Published online May 6, 2019. doi:10.1001/jama.2019.5586

Key Points

Question What is the maximum plasma concentration of active ingredients of various types of sunscreen formulations under maximal use conditions?

Findings In this randomized clinical trial that included 24 healthy participants and application of 4 commercially available sunscreen formulations, maximum plasma concentrations (geometric mean [coefficient of variation]) for the active ingredient avobenzone were 4.0 (60.9%), 3.4 (77.3%), 4.3 (46.1%), and 1.8 (32.1%) ng/mL for 2 different sprays, a lotion, and a cream, respectively.

Meaning The systemic absorption of sunscreen active ingredients supports the need for further studies to determine the clinical significance of these findings.

Abstract

Importance The US Food and Drug Administration (FDA) has provided guidance that sunscreen active ingredients with systemic absorption greater than 0.5 ng/mL or with safety concerns should undergo nonclinical toxicology assessment including systemic carcinogenicity and additional developmental and reproductive studies.

Objective To determine whether the active ingredients (avobenzone, oxybenzone, octocrylene, and ecamsule) of 4 commercially available sunscreens are absorbed into systemic circulation.

Design, Setting, and Participants Randomized clinical trial conducted at a phase 1 clinical pharmacology unit in the United States and enrolling 24 healthy volunteers. Enrollment started in July 2018 and ended in August 2018.

Interventions Participants were randomized to 1 of 4 sunscreens: spray 1 (n = 6 participants), spray 2 (n = 6), a lotion (n = 6), and a cream (n = 6). Two milligrams of sunscreen per 1 cm² was applied to 75% of body surface area 4 times per day for 4 days, and 30 blood samples were collected over 7 days from each participant.

Main Outcomes and Measures The primary outcome was the maximum plasma concentration of avobenzone. Secondary outcomes were the maximum plasma concentrations of oxybenzone, octocrylene, and ecamsule.

Results Among 24 participants randomized (mean age, 35.5 [SD, 1.5] years; 12 [50%] women; 14 [58%] black or African American; 14 [58%]), 23 (96%) completed the trial. For avobenzone, geometric mean maximum plasma concentrations were 4.0 ng/mL (coefficient of variation, 6.9%) for spray 1; 3.4 ng/mL (coefficient of variation, 77.3%) for spray 2; 4.3 ng/mL (coefficient of variation, 46.1%) for lotion; and 1.8 ng/mL (coefficient of variation, 32.1%). For oxybenzone, the corresponding values were 209.6 ng/mL (66.8%) for spray 1, 194.9 ng/mL (52.4%) for spray 2, and 169.3 ng/mL (44.5%) for lotion; for octocrylene, 2.9 ng/mL (102%) for spray 1, 7.8 ng/mL (113.3%) for spray 2, 5.7 ng/mL (66.3%) for lotion, and 5.7 ng/mL (47.1%) for cream; and for ecamsule, 1.5 ng/mL (166.1%) for cream. Systemic concentrations greater than 0.5 ng/mL were reached for all 4 products after 4 applications on day 1. The most common adverse event was rash, which developed in 1 participant with each sunscreen.

Conclusions and Relevance In this preliminary study involving healthy volunteers, application of 4 commercially available sunscreens under maximal use conditions resulted in plasma concentrations that exceeded the threshold established by the FDA for potentially waiving some nonclinical toxicology studies for sunscreens. The systemic absorption of sunscreen ingredients supports the need for further studies to determine the clinical significance of these findings. These results do not indicate that individuals should refrain from the use of sunscreen.

Printable full text of this study is available at:

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6549296/?report=printable>.

3 Poems by Mauro

Green for the Innocents

We think they're going green
But just what does that mean
Is it helping the planet's health
Or is it just another dream

The innocents are gone
Disappeared into the past
Only whispers are left
What's there now can't last

We can't see as far as we could
Because the air isn't as good
As it once was before we went green
Since we all know what that means

What they tell us is a blatant lie
Just to hide that they don't comply
They do anything that they want to do
And we know it'll hurt me and you

Burn and destroy is their dream
Since they'll profit from their scheme
All the time saying that it's green
But that's not what they really mean

The green is not for us at all
It's the stuff that they can haul
Making only their pockets green
While the world becomes obscene

Wake up and take up the right call
Don't buy into their world and their drawl
Less is more if we abide
To buy less and stem the coming tide

Sell and buy your dreams online

We're asked to bow to their shrine
With no caring about what's to come
If we bow to their beating drum

Shop now for things you don't need
Creating a scene for their greed
It may be that we don't really want
But we're mesmerized by their détente

We don't need empty promises
And platitudes won't soothe us
What we need is action now
Not words to make us bow

We don't ask for much
Away from their touch
Just some protection
From their intention

In the Knowing

At one time before we knew
Secluded in the not knowing
Of the things we did not have
And those that weren't showing

The sirens only whispered
For the few that would listen
The rest were deaf and blind
It was a trick of the mind

We're shocked and confused
By the visions and noise
Created for us to know
How much we want our toys

It's all just a trick

Making us believe
The truth is what matters
Yet they deceive

They've got your number
They follow who you are
Their ghosts surround you
You're stuck from afar

Helping hands everywhere
To help you to decide
With incentives hidden
How can you abide

They're forcing you to feel
The things they plant inside
And those feelings won't go away
Not until they no longer sway

We need help but who to ask
Looking for truths is a lonely task

Blinded by the Blight

The lamp that I lit last night
The one that was so bright
Is now so dull it seems
Not like in my dreams

I wake up in the morning
The games are about to begin
The chants I hear are grim
They're not worthy of that hymn

The garden is before us
The trees no longer grow
As the days pass by

They shrink and then they die

Our lives won't be the same
Not like it's always been
Ideas of what was once right
Now seem like a blight

You stand behind me
All in tears just to say
Your clothes are all torn
But you're covered anyway

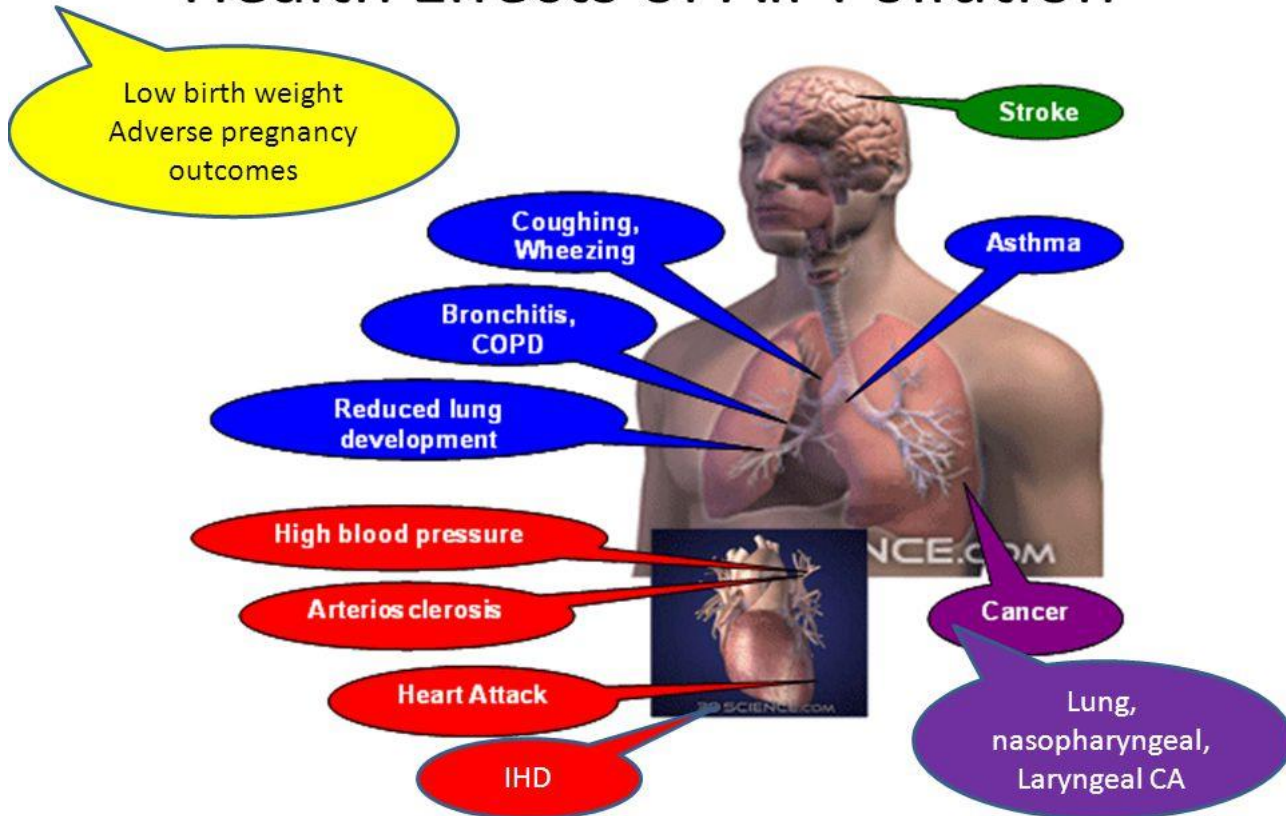
That tiny hand in mine
I see it like it's a shrine
To be one with all that is
Sinks into an abyss

I can't leave you again
The sun is still there
If my soul can't be found
I'll be seeing you around

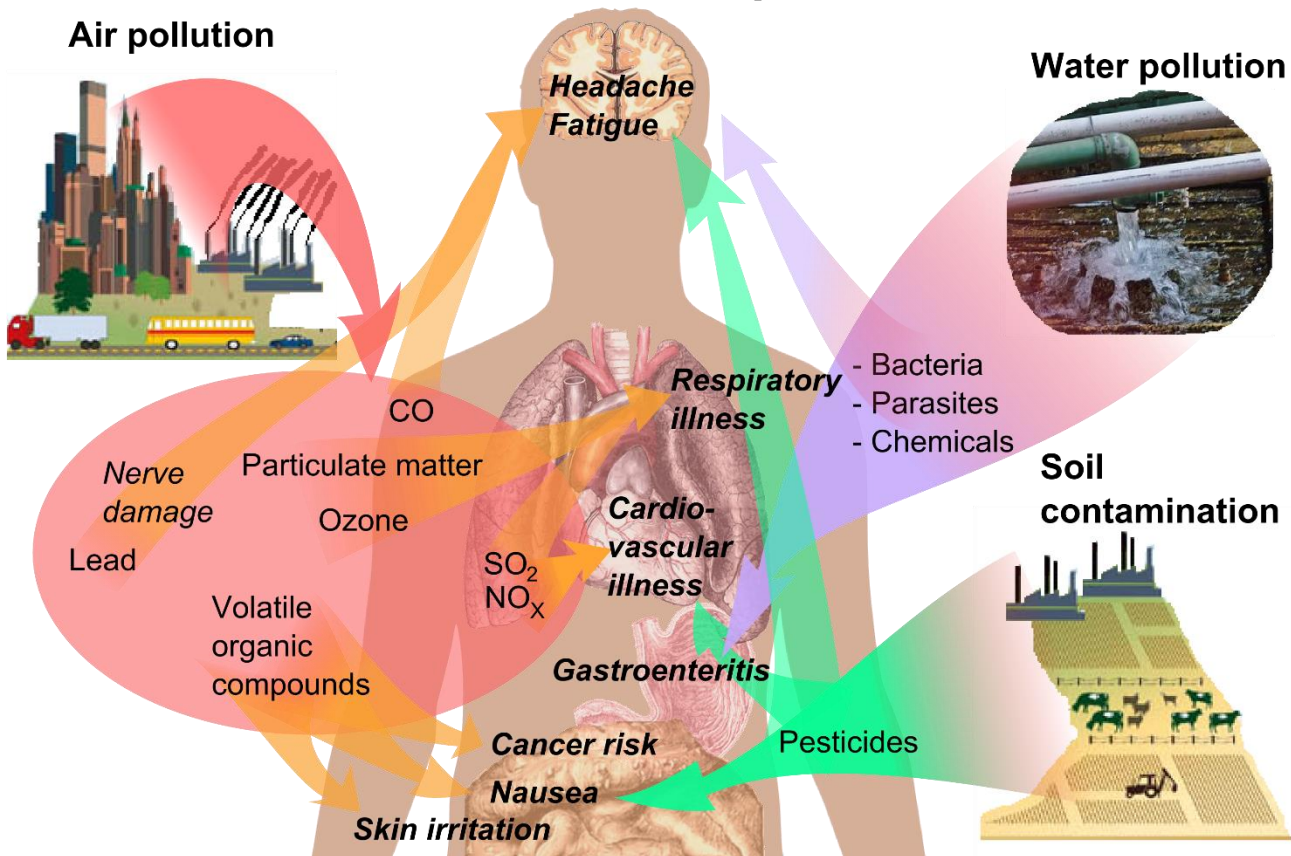
I still listen to the story
Of how we came to be
I listen but now I find
That I've always been blind

I'm crying all the time
The shame that is mine
Belongs to us all
As we begin our free fall

Health Effects of Air Pollution



Health effects of pollution



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