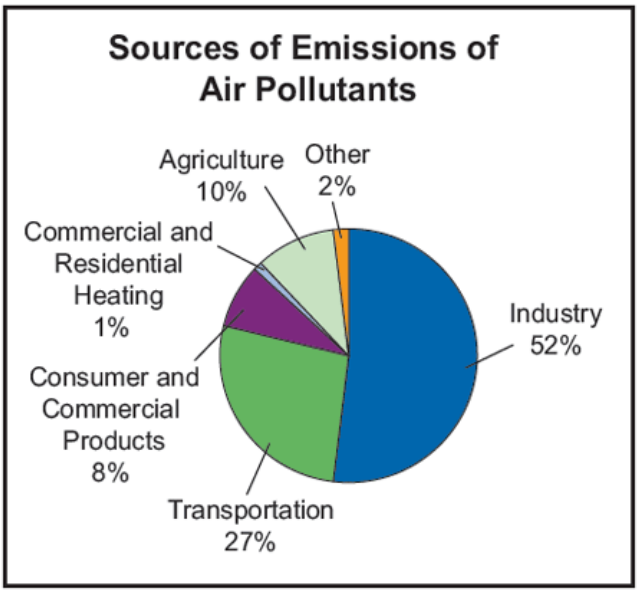
Pollution as Devolution of the Species?

A Mini eBook

By Mauro Di Pasquale, B.Sc. (Hons), M.D.
Updated: January 25, 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

Table of Contents

Trillions for Your Thoughts 2

Introduction 5

Pollution and Covid-19 Pandemic 5

Air quality and COVID-19 adverse outcomes: Divergent views and experimental findings 6

Evidence-Based Considerations Exploring Relations between SARS-CoV-2 Pandemic and Air Pollution: Involvement of PM2.5-Mediated Up-Regulation of the Viral Receptor ACE-2 6

Pollution and Climate Change - 7

The video - What Happens if Earth Gets 2°C Warmer? Is worth watching. 7

The Green Revolution? 8

Pollution Escalation and Other Information 8

Flame Retardants 8

Other Sources of Pollution 9

Reactivity-directed analysis - a novel approach for the identification of toxic organic electrophiles in drinking water 10

Abstract 10

Air Pollution 11

Air pollution exposure is associated with the gut microbiome as revealed by shotgun metagenomic sequencing 11

Longer commutes are associated with increased human exposure to tris(1,3-dichloro-2-propyl) phosphate. 12

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. 13

Ultrafine particles and children's health: Literature review. 13

Developmental impact of air pollution on brain function. 14

The Impact of Inhaled Ambient Ultrafine Particulate Matter on Developing Brain: Potential Importance of Elemental Contaminants. 14

The Brave New World of Pollution 15

Environmental Cadmium and Mortality from Influenza and Pneumonia in U.S. Adults 15

Low-level lead exposure and mortality in US adults: a population-based cohort study. 16

Heavy metal associated health hazards: An interplay of oxidative stress and signal transduction 17

Toxic metal exposure as a possible risk factor for COVID-19 and other respiratory infectious diseases 18

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 20

Does night-time aircraft noise trigger mortality? A case-crossover study on 24 886 cardiovascular deaths 20

Plastic Pollution 21

Microplastics 21

Environmental exposure enhances the internalization of microplastic particles into cells 22

Great Pacific Garbage Patch growing rapidly, study shows 22
New analysis reveals the region contains as much as sixteen times more plastic than previously estimated, with pollution levels increasing exponentially 22
Researchers estimate 10,000 metric tons of plastic enter Great Lakes every year 24
Study inventories movement of plastic and microplastic debris throughout lake system 24
Air Pollution 26
News 26
Introduction 26
Indoor Air Pollution 27
American Thoracic Society 2017 INTERNATIONAL CONFERENCE 27
Environmental exposures and sleep outcomes: A review of evidence, potential mechanisms, and implications 28
Genomic, Epigenomic, and Phenotypic Effects of Pollution 29
Air pollution and molecular changes in age-related diseases 29
Prenatal Particulate Air Pollution and DNA Methylation in Newborns: An Epigenome-Wide Meta-Analysis 29
Air pollution associated epigenetic modifications: Transgenerational inheritance and underlying molecular mechanisms. 30
Epigenetics as a mechanism linking developmental exposures to long-term toxicity. 31
Epigenetic memory in response to environmental stressors. 31
Transgenerational epigenetic inheritance: adaptation through the germline epigenome? 32
Elusive inheritance: Transgenerational effects and epigenetic inheritance in human environmental disease. 32
Environmental stress and epigenetic transgenerational inheritance. 33
Environmental epigenetics and its implication on disease risk and health outcomes. 33
MicroRNAs expression in relation to particulate matter exposure: A systematic review. 34
MicroRNA expression profiling in human acute organophosphorus poisoning and functional analysis of dysregulated miRNAs 34
Organic Food? 35
Body Pollution 36
Other Underappreciated Sources of Body Pollution 37
Cigarettes and Electronic Cigarettes 38
What Can Be Done? 38
Some Easy Ways to Decrease Exposure to Pollutants Including Endocrine Disrupting Chemicals 39
Decreasing Body Pollution with Specific Nutritional Supplementation 41
MetabolicDiet.com Nutritional Supplement Products for Decreasing the Adverse Effects of Pollution 41
Coenzyme Q10 (CoQ10) 42
Antioxidant CoQ10 Restores Fertility by Rescuing Bisphenol A-Induced Oxidative DNA Damage in the *Caenorhabditis elegans* Germline. 42
L-carnitine and Acetyl-L-carnitine 43
Environmental Stressors and Their Impact on Health and Disease with Focus on Oxidative Stress. . 43

Reports, Citations and Abstracts of Interest **44**
Respiratory and cardiovascular responses to walking down a traffic-polluted road compared with walking in a traffic-free area **44**
Impact of ambient air pollution on physical activity among adults: a systematic review and meta-analysis. **45**
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 **46**
Environmental pollution is associated with increased risk of psychiatric disorders in the US and Denmark. **47**
Effect of Sunscreen Application Under Maximal Use Conditions on Plasma Concentration of Sunscreen Active Ingredients: A Randomized Clinical Trial **47**
2 Poems by Mauro **49**
References **53**

Introduction

The common consensus, and one fostered by Darwin, is that evolution is progressive in that it's 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.

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.

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.

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**.

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.

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

Leonardo Becchetti[1], Gabriele Beccari[2], Gianluigi Conzo[2], Pierluigi Conzo[3], Davide De Santis[2], Francesco Salustri[4]

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 PM2.5-Mediated Up-Regulation of the Viral Receptor ACE-2

Marina Borro[1], Paolo Di Girolamo[2], Giovanna Gentile[1], Ottavia De Luca[3], Robert Preissner[4], Adriano Marcolongo[5], Stefano Ferracuti[6], Maurizio Simmaco[1]

Int J Environ Res Public Health 2020 Aug 2;17(15):E5573. doi: 10.3390/ijerph17155573.
PMID: 32748812

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.[15161718192021222324] However, studies also implicate pollution in antibiotic resistance.[252627]

Pollution and Climate Change -

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

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

Pollution and climate change are intertwined. 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 Organisation (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.[28]

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.

Pollution Escalation and Other Information

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.[29]

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.[30]

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,

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.[31323334353637] 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.[3839]

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.[40]

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.[41]

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.

Environ Sci Process Impacts
. 2021 Jan 12. Online ahead of print. PMID: 33432313. DOI: 10.1039/d0em00471e

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.[42] 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=Zk11vI-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..[4344]

Environ Int. 2020 May;138:105604. doi: 10.1016/j.envint.2020.105604. Epub 2020 Mar 2.

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 O_3 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 NO_2 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 O_3 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 O_3 as an important pollutant that may alter the human gut microbiome.

Environ Int. 2020 Jan 27;136:105499. doi: 10.1016/j.envint.2020.105499. [Epub ahead of print]

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 500 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.[45464748]

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/m^3 particle mass concentrations from postnatal day (PND) 4-7 and 10-13 for 4 h/day. Changes in serum testosterone (T) and corticosterone where 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".[49] 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.[50]

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 years[51] – **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.[5253]

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**.[54]

Environ Health Perspect. 2020 Dec;128(12):127004. doi: 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 ≥45≥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.

Free full text available at http://www.thelancet.com/pdfs/journals/lanpub/PIIS2468-2667(18)30025-2.pdf.

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

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-κB, NRF2, JAK-STAT, JNK, FOXO, and HIF.

Food Chem Toxicol. 2020 Dec;146:111809. doi: 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 (PM2.5) 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".[55]

In the Introduction of this paper (the full paper is available at t**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

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.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.[56] 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.[57]

Another recent study found that environmentally exposed microplastic particles enter the body much more than pristine microplastic particles with significant toxic and endocrine disrupting effects[58] (see abstract below).

Sci Adv 2020 Dec 9;6(50):eabd1211. doi: 10.1126/sciadv.abd1211. Print 2020 Dec.

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.[59,60]

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.

Share:

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 km^2 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 km^2, 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

---

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.

Share:

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 metrics 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

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.[61] A recent study, while mentioning the WHO announcement, found that traffic-related air pollution is adversely affecting mental health. [62]

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.[6364656667686970]

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.[71]

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.[72] 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.[73]

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.[747576777879808182] Exposure to pollution, both indoor and outdoor, at any time but especially during gestation, results in adverse epigenetic and transgenerational epigenetic changes that adversely affect both short and long term physical and mental processes.[838485868788899091929394959697 98]

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.[99100101102103104105106107108109110111]

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, including airborne spores, both dead and alive.

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.[112113114115116117118119120]

As well, a recent paper found that environmental pollution is associated with increased risk of psychiatric disorders.[121]

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.2020.110406j 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.[122123124125]

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.[126127128]

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

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. 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.

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 A[1], Bunkar N[1], Kumar R[1], Bhargava A[1], Tiwari R[1], Chaudhury K[2], Goryacheva IY[3], Mishra PK[4].

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 R[1], Melén E[2], Herceg Z[3], Beckers J[4], Chen J[5], Karagas M[6], Puga A[7], Xia Y[7], Chadwick L[8], Yan W[9], Audouze K[10], Slama R[11], Heindel J[12], Grandjean P[13], Kawamoto T[14], Nohara K[15].

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.

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 SM[1], 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 hydroxylmethylation 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.[129130131]

Environ Pollut. 2020 May;260:113961. doi: 10.1016/j.envpol.2020.113961. Epub 2020 Jan 21.

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 quantitive 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."[132] (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.[133] (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.[134][135][136]

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,[137] 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.[138]

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.[139]

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.[140]

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.[141] 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.[142] 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.[143]

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.[144145146147] 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.[148149150151152153154]

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.

- 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 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).[155]

- 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.[156] 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.[157][158]

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.[159][160][161][162]

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."[163]

- 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.[164][165]

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**.[166][167][168][169][170][171][172][173][174][175][176][177][178]

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.[179][180][181][182]

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.[183]

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.[184185186187188189190191192]

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."**[193] **In a study published this year the authors concluded that** "CoQ10 may counteract BPA-induced reprotoxicity."[194]

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 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.[195196]

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.[197198199200201202203]

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.

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 (NO_2) 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, NO_2, PM_{10}, $PM_{2.5}$, 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 [FEV_1] 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 FEV_1 and FVC, and an increase in R5-20 were associated with an increase in during-walk exposure to NO_2, ultrafine particles and $PM_{2.5}$, and an increase in PWV and augmentation index with NO_2 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 $PM_{2.5}$, PM_{10}, O_3, 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 ($\mu g/m^3$) 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 PM_{10}, NO_2, SO_2, CO, O_3, 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 PM_{10}, NO_2, SO_2, CO, O_3, 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, PhD[1]; Robbert Zusterzeel, MD, PhD, MPH[1]; Nageswara R. Pilli, PhD[1]; et alVikram Patel, PhD[1]; Donna A. Volpe, PhD[1]; Jeffry Florian, PhD[1]; Luke Oh, PhD[2]; Edward Bashaw, PharmD[2]; Issam Zineh, PharmD, MPH[2]; Carlos Sanabria, MD[3]; Sarah Kemp, RN[3]; Anthony Godfrey, PharmD[3]; Steven Adah, PhD[4]; Sergio Coelho, PhD[4]; Jian Wang, PhD[5]; Lesley-Anne Furlong, MD[5]; Charles Ganley, MD[5]; Theresa Michele, MD[4]; David G. Strauss, MD, PhD[1]
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 cm^2 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.

---

2 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

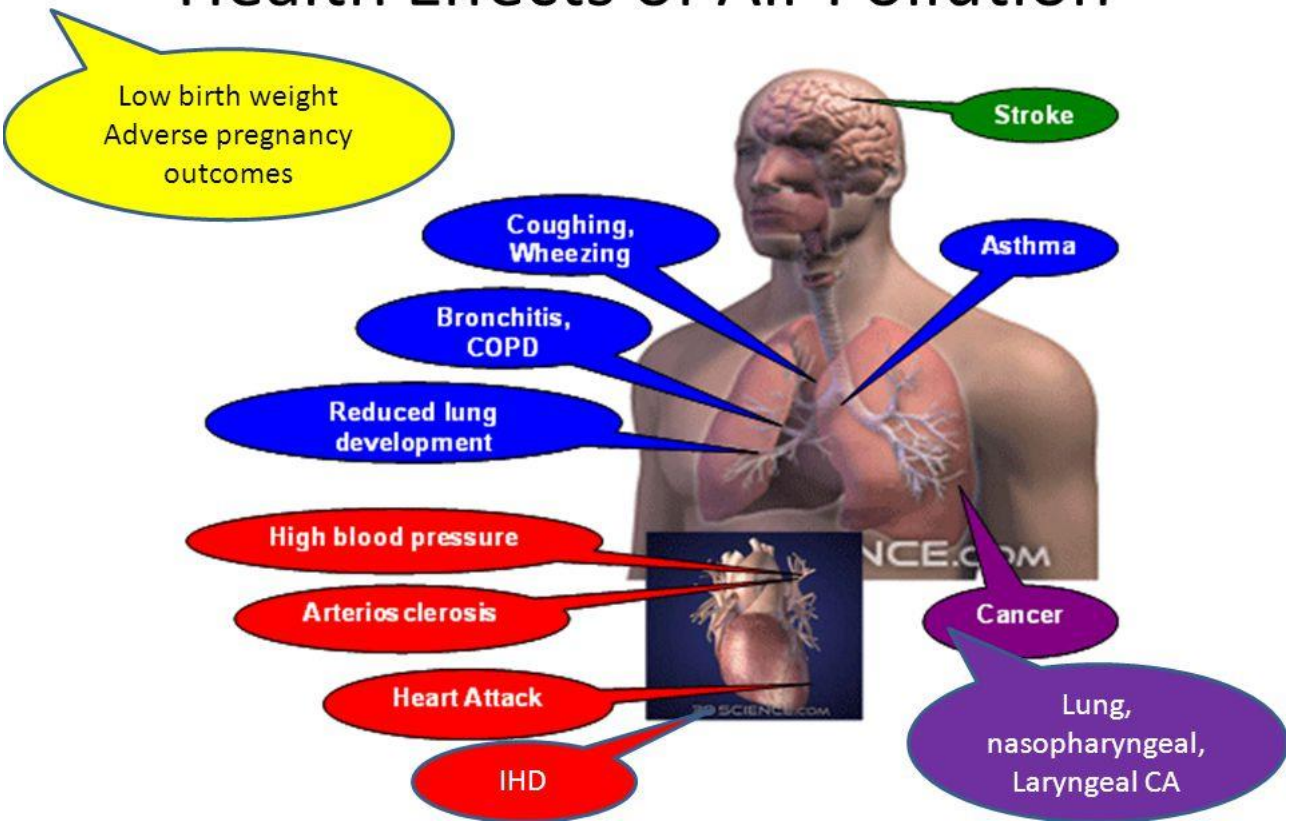
They've got your number
They follow who you are
Their ghosts surround you
You're stuck from afar

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

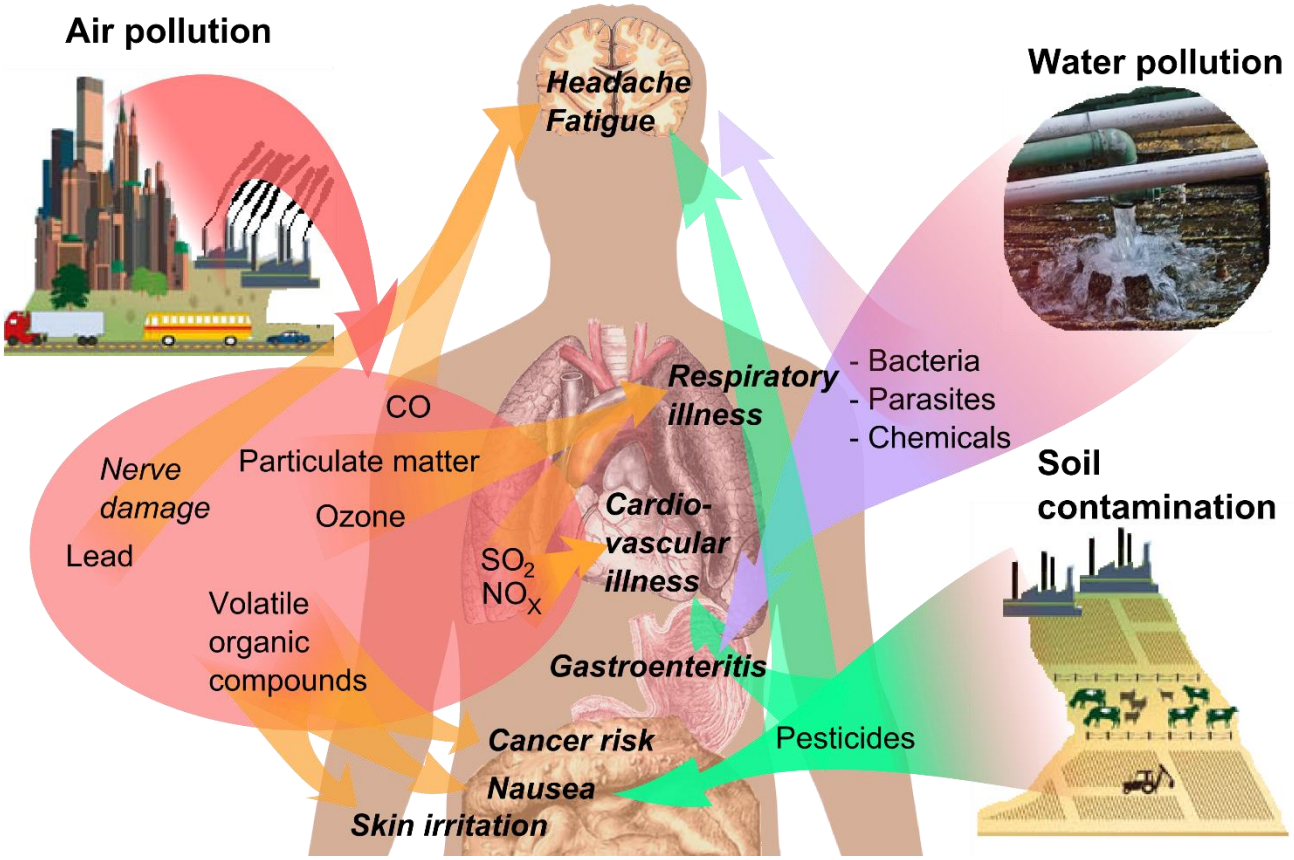
They’re forcing you to feel
The things they plant inside
And those feelings won't go away
Not until they no longer sway

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

Health Effects of Air Pollution



Health effects of pollution



References

1 Landrigan PJ, Fuller R, Acosta NJR, Adeyi O, Arnold R, Basu NN, Baldé AB, Bertollini R, Bose-O'Reilly S, Boufford JI, Breysse PN, Chiles T, Mahidol C, Coll-Seck AM, Cropper ML, Fobil J, Fuster V, Greenstone M, Haines A, Hanrahan D, Hunter D, Khare M, Krupnick A, Lanphear B, Lohani B, Martin K, Mathiasen KV, McTeer MA, Murray CJL, Ndahimananjara JD, Perera F, Potocnik J, Preker AS, Ramesh J, Rockström J, Salinas C, Samson LD, Sandilya K, Sly PD, Smith KR, Steiner A, Stewart RB, Suk WA, van Schayck OCP, Yadama GN, Yumkella K, Zhong M. The Lancet Commission on pollution and health. LLancet. 2018 Feb 3;391(10119):462-512.

2 Fattorini D, Regoli F. Role of the chronic air pollution levels in the Covid-19 outbreak risk in Italy. Environ Pollut. 2020 Sep;264:114732. doi: 10.1016/j.envpol.2020.114732. Epub 2020 May 4. PMID: 32387671.

3 Frontera A, Cianfanelli L, Vlachos K, Landoni G, Cremona G. Severe air pollution links to higher mortality in COVID-19 patients: The "double-hit" hypothesis. J Infect. 2020 Aug;81(2):255-259. doi: 10.1016/j.jinf.2020.05.031. Epub 2020 May 21. PMID: 32447007.

4 Moelling K, Broecker F. Air Microbiome and Pollution: Composition and Potential Effects on Human Health, Including SARS Coronavirus Infection. J Environ Public Health. 2020 May 28;2020:1646943. doi: 10.1155/2020/1646943. eCollection 2020. PMID: 32565838.

5 Comunian S, Dongo D, Milani C, Palestini P. Air Pollution and Covid-19: The Role of Particulate Matter in the Spread and Increase of Covid-19's Morbidity and Mortality. Int J Environ Res Public Health. 2020 Jun 22;17(12):4487. doi: 10.3390/ijerph17124487. PMID: 32580440.

6 Borro M, Di Girolamo P, Gentile G, De Luca O, Preissner R, Marcolongo A, Ferracuti S, Simmaco M. Evidence-Based Considerations Exploring Relations between SARS-CoV-2 Pandemic and Air Pollution: Involvement of PM2.5-Mediated Up-Regulation of the Viral Receptor ACE-2. Int J Environ Res Public Health. 2020 Aug 2;17(15):E5573. doi: 10.3390/ijerph17155573. PMID: 32748812.

7 Reyes MSS, Medina PMB. Environmental pollutant exposure can exacerbate COVID-19 neurologic symptoms. Med Hypotheses. 2020 Jul 26;144:110136. doi: 10.1016/j.mehy.2020.110136. Online ahead of print. PMID: 3277194.

8 Adhikari A, Yin J. Short-Term Effects of Ambient Ozone, PM(2.5,) and Meteorological Factors on COVID-19 Confirmed Cases and Deaths in Queens, New York. Int J Environ Res Public Health. 2020 Jun 5;17(11):4047. doi: 10.3390/ijerph17114047. PMID: 32517125.

9 Bornstein SR, Voit-Bak K, Schmidt D, Morawietz H, Bornstein AB, Balanzew W, Julius U, Rodionov RN, Biener AM, Wang J, Schulte KM, Krebs P, Vollmer G, Straube R. Is There a Role for Environmental and Metabolic Factors Predisposing to Severe COVID-19? Horm Metab Res. 2020 Jul;52(7):540-546. doi: 10.1055/a-1182-2016. Epub 2020 Jun 29. PMID: 32599638.

10 Urrutia-Pereira M, Mello-da-Silva CA, Solé D. COVID-19 and air pollution: A dangerous association? Allergol Immunopathol (Madr). 2020 Jul 1:S0301-0546(20)30109-9. doi: 10.1016/j.aller.2020.05.004. Online ahead of print. PMID: 32636082.

11 In 't Veen JCCM, Kappen JH, van Schayck OCP. [Air pollution: a determinant for COVID-19?]. Ned Tijdschr Geneeskd. 2020 May 28;164:D5153. PMID: 32749825.

12 Manoj MG, Satheesh Kumar MK, Valsaraj KT, Sivan C, Vijayan SK. Potential link between compromised air quality and transmission of the novel corona virus (SARS-CoV-2) in affected areas. Environ Res. 2020 Aug 1:110001. doi: 10.1016/j.envres.2020.110001. Online ahead of print. PMID: 32750327.

13 Borro M, Di Girolamo P, Gentile G, De Luca O, Preissner R, Marcolongo A, Ferracuti S, Simmaco M. Evidence-Based Considerations Exploring Relations between SARS-CoV-2 Pandemic and Air Pollution: Involvement of PM2.5-Mediated Up-Regulation of the Viral Receptor ACE-2. Int J Environ Res Public Health. 2020 Aug 2;17(15):E5573. doi: 10.3390/ijerph17155573. PMID: 32748812.

14 Becchetti L, Beccari G, Conzo G, Conzo P, De Santis D, Salustri F. Air quality and COVID-19 adverse outcomes: Divergent views and experimental findings. Environ Res. 2020 Dec 3;193:110556. doi: 10.1016/j.envres.2020.110556. Epub ahead of print. PMID: 33278470; PMCID: PMC7711169.
15 Wang J, Ben W, Yang M, Zhang Y, Qiang Z. Dissemination of veterinary antibiotics and corresponding resistance genes from a concentrated swine feedlot along the waste treatment paths. Environ Int. 2016 Jul-Aug;92-93:317-23.
16 Yang Y, Song W, Lin H, Wang W, Du L, Xing W. Antibiotics and antibiotic resistance genes in global lakes: A review and meta-analysis. Environ Int. 2018 Jul;116:60-73.
17 Ferroni L, Lovito C, Scoccia E, Dalmonte G, Sargenti M, Pezzotti G, Maresca C, Forte C, Magistrali CF. Antibiotic Consumption on Dairy and Beef Cattle Farms of Central Italy Based on Paper Registers. Antibiotics (Basel). 2020 May 25;9(5):273. doi: 10.3390/antibiotics9050273. PMID: 32466135.
18 Okubo Y, Michihata N, Uda K, Kinoshita N, Horikoshi Y, Miyairi I. Impacts of Primary Care Physician System on Healthcare Utilization and Antibiotic Prescription: Difference-in-Differences and Causal Mediation Analyses. Pediatr Infect Dis J. 2020 Jun 3. doi: 10.1097/INF.0000000000002762. Online ahead of print. PMID: 32502123.
19 Tang KL, Teoh TF, Ooi TT, Khor WP, Ong SY, Lim PP, Abdul Karim S, Tan SSA, Ch'ng PP, Choong YC, Foong WS, Ganesan S, Khan AH, Ming LC. Public Hospital Pharmacists' Perceptions and Knowledge of Antibiotic Use and Resistance: A Multicenter Survey. Antibiotics (Basel). 2020 Jun 9;9(6):311. doi: 10.3390/antibiotics9060311. PMID: 32526821.
20 Chang Y, Sangthong R, McNeil EB, Tang L, Chongsuvivatwong V. Effect of a computer network-based feedback program on antibiotic prescription rates of primary care physicians: A cluster randomized crossover-controlled trial. J Infect Public Health. 2020 Jun 15:S1876-0341(20)30494-9. doi: 10.1016/j.jiph.2020.05.027. Online ahead of print. PMID: 32554035.
21 Crayton E, Richardson M, Fuller C, Smith C, Liu S, Forbes G, Anderson N, Shallcross L, Michie S, Hayward A, Lorencatto F. Interventions to improve appropriate antibiotic prescribing in long-term care facilities: a systematic review. BMC Geriatr. 2020 Jul 9;20(1):237. doi: 10.1186/s12877-020-01564-1. PMID: 32646382.
22 Gebrehiwot Z, Tadiwos Y. Knowledge and Beliefs of Health Care Professionals Towards Antimicrobial Resistance in Hiwot Fana Specialized University Hospital, in Harar, Ethiopia. Infect Drug Resist. 2020 Jun 30;13:2027-2035. doi: 10.2147/IDR.S254237. eCollection 2020. PMID: 32636656.
23 Pieri A, Aschbacher R, Fasani G, Mariella J, Brusetti L, Pagani E, Sartelli M, Pagani L. Country Income Is Only One of the Tiles: The Global Journey of Antimicrobial Resistance among Humans, Animals, and Environment. Antibiotics (Basel). 2020 Aug 1;9(8):E473. doi: 10.3390/antibiotics9080473. PMID: 32752276.
24 Frenette C, Sperlea D, German GJ, Afra K, Boswell J, Chang S, Goossens H, Grant J, Lefebvre MA, McGeer A, Mertz D, The 2017 global point prevalence survey of antimicrobial consumption and resistance in Canadian hospitals. Science M, Versporten A, Thirion DJG. Antimicrob Resist Infect Control. 2020 Jul 11;9(1):104. doi: 10.1186/s13756-020-00758-x. PMID: 32653046.
25 Zhao Y, Cocerva T, Cox S, Tardif S, Su JQ, Zhu YG, Brandt KK. Evidence for co-selection of antibiotic resistance genes and mobile genetic elements in metal polluted urban soils. Sci Total Environ. 2019 Mar 15;656:512-520.
26 Bischofberger AM, Baumgartner M, Pfrunder-Cardozo KR, Allen RC, Hall AR. Associations between sensitivity to antibiotics, disinfectants and heavy metals in natural, clinical and laboratory isolates of Escherichia coli. Environ Microbiol. 2020 Jul;22(7):2664-2679. doi: 10.1111/1462-2920.14986. Epub 2020 Mar 25. PMID: 32162766.
27 Thomas JC 4th, Oladeinde A, Kieran TJ, Finger JW Jr, Bayona-Vásquez NJ, Cartee JC, Beasley JC, Seaman JC, McArthur JV, Rhodes OE Jr, Glenn TC. Co-occurrence of antibiotic, biocide, and heavy metal resistance genes in bacteria from metal and radionuclide contaminated soils at the Savannah River Site. Microb Biotechnol. 2020 Jul;13(4):1179-1200. doi: 10.1111/1751-7915.13578. Epub 2020 May 3. PMID: 32363769.
[28] https://www.nationalparkstraveler.org/sites/default/files/attachments/fcosc-01-615419.pdf.

29 Zulauf N, Dröge J, Klingelhöfer D, Braun M, Oremek GM, Groneberg DA. Indoor Air Pollution in Cars: An Update on Novel Insights. Int J Environ Res Public Health. 2019 Jul 9;16(13):2441. doi: 10.3390/ijerph16132441. PMID: 31323996; PMCID: PMC6650813.
30 Reddam A, Tait G, Herkert N, Hammel SC, Stapleton HM, Volz DC. Longer commutes are associated with increased human exposure to tris(1,3-dichloro-2-propyl) phosphate. Environ Int. 2020 Jan 27;136:105499. doi: 10.1016/j.envint.2020.105499. [Epub ahead of print]
31 Wang Y, Chang W, Wang L, Zhang Y, Zhang Y, Wang M, Wang Y, Li P. A review of sources, multimedia distribution and health risks of novel fluorinated alternatives. Ecotoxicol Environ Saf. 2019 Oct 30;182:109402. doi: 10.1016/j.ecoenv.2019.109402. Epub 2019 Jul 4.
32 Bo Guo, Jicai Zeng, Mark L. Brusseau. A Mathematical Model for the Release, Transport, and Retention of Per- and Polyfluoroalkyl Substances (PFAS) in the Vadose Zone. Water Resources Research, 2020; 56 (2) DOI: 10.1029/2019WR026667
33 Ghisi R, Vamerali T, Manzetti S. Accumulation of perfluorinated alkyl substances (PFAS) in agricultural plants: A review. Environ Res. 2019 Feb;169:326-341.
34 Davis AN, Carlo G, Gulseven Z, Palermo F, Lin CH, Nagel SC, Vu DC, Vo PH, Ho TL, McElroy JA. Exposure to environmental toxicants and young children's cognitive and social development. Rev Environ Health. 2019 Mar 26;34(1):35-56.
35 Egendorf SP, Gailey AD, Schachter AE, Mielke HW. Soil toxicants that potentially affect children's health. Curr Probl Pediatr Adolesc Health Care. 2020 Jan 25:100741. doi: 10.1016/j.cppeds.2019.100741. [Epub ahead of print]
36 Schachter AE, Gailey A, Egendorf SP, Mielke HW. Mechanisms of children's soil exposure. Curr Probl Pediatr Adolesc Health Care. 2020 Jan 26:100742. doi: 10.1016/j.cppeds.2019.100742. [Epub ahead of print]
37 Davis AN, Carlo G, Gulseven Z, Palermo F, Lin CH, Nagel SC, Vu DC, Vo PH, Ho TL, McElroy JA. Exposure to environmental toxicants and young children's cognitive and social development. Rev Environ Health. 2019 Mar 26;34(1):35-56.
[38] Anderson DM, Cembella AD, Hallegraeff GM. Progress in understanding harmful algal blooms: paradigm shifts and new technologies for research, monitoring, and management. Ann Rev Mar Sci. 2012;4:143-176. doi:10.1146/annurev-marine-120308-081121.
[39] Kim H, Lee K, Lim DI, Nam SI, Kim TW, Yang JT, Ko YH, Shin KH, Lee E. Widespread Anthropogenic Nitrogen in Northwestern Pacific Ocean Sediment. Environ Sci Technol. 2017 Jun 6;51(11):6044-6052. doi: 10.1021/acs.est.6b05316. Epub 2017 May 11. PMID: 28462990.
[40] Monney I, Donkor EA, Buamah R. Clean vehicles, polluted waters: empirical estimates of water consumption and pollution loads of the carwash industry. Heliyon. 2020 May 13;6(5):e03952. doi: 10.1016/j.heliyon.2020.e03952. PMID: 32426547; PMCID: PMC7226662.
[41] Sarmadi M, Foroughi M, Najafi Saleh H, Sanaei D, Zarei AA, Ghahrchi M, Bazrafshan E. Efficient technologies for carwash wastewater treatment: a systematic review. Environ Sci Pollut Res Int. 2020 Oct;27(28):34823-34839. doi: 10.1007/s11356-020-09741-w. Epub 2020 Jul 7. PMID: 32632696.
42 Chen K, Schneider A, Cyrys J, Wolf K, Meisinger C, Heier M, von Scheidt W, Kuch B, Pitz M, Peters A, Breitner S; KORA Study Group. Hourly Exposure to Ultrafine Particle Metrics and the Onset of Myocardial Infarction in Augsburg, Germany. Environ Health Perspect. 2020 Jan;128(1):17003. doi: 10.1289/EHP5478. Epub 2020 Jan 15.
43 Bailey MJ, Naik NN, Wild LE, Patterson WB, Alderete TL. Exposure to air pollutants and the gut microbiota: a potential link between exposure, obesity, and type 2 diabetes. Gut Microbes. 2020 Sep 2;11(5):1188-1202.
44 Moelling K, Broecker F. Air Microbiome and Pollution: Composition and Potential Effects on Human Health, Including SARS Coronavirus Infection. J Environ Public Health. 2020 May 28;2020:1646943. doi: 10.1155/2020/1646943. eCollection 2020. PMID: 32565838.
45 da Costa E Oliveira JR, Base LH, de Abreu LC, Filho CF, Ferreira C, Morawska L. Ultrafine particles and children's health: Literature review. Paediatr Respir Rev. 2019 Nov;32:73-81.
46 Cory-Slechta DA, Sobolewski M, Marvin E, Conrad K, Merrill A, Anderson T, Jackson BP, Oberdorster G. The Impact of Inhaled Ambient Ultrafine Particulate Matter on Developing Brain: Potential Importance of Elemental Contaminants. Toxicol Pathol. 2019 Dec;47(8):976-992.
47 Costa LG, Cole TB, Dao K, Chang YC, Garrick JM. Developmental impact of air pollution on brain function. Neurochem Int. 2019 Dec;131:104580. doi: 10.1016/j.neuint.2019.104580. Epub 2019 Oct 15.

48 Sobolewski M, Anderson T, Conrad K, Marvin E, Klocke C, Morris-Schaffer K, Allen JL, Cory-Slechta DA. 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. Neurotoxicology. 2018 Sep;68:203-211.
49 Hornos Carneiro MF, Shin N, Karthikraj R, Barbosa F Jr, Kannan K, Colaiácovo MP. Antioxidant CoQ10 Restores Fertility by Rescuing Bisphenol A-Induced Oxidative DNA Damage in the Caenorhabditis elegans Germline. Genetics. 2020 Feb;214(2):381-395.
50 Bazyar J, Pourvakhshoori N, Khankeh H, Farrokhi M, Delshad V, Rajabi E. 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. Environ Sci Pollut Res Int. 2019 May;26(13):12648-12661. doi: 10.1007/s11356-019-04874-z. Epub 2019 Mar 22. PMID: 30903465.
51 Lanphear BP, Rauch S, Auinger P, Allen RW, Hornung RW. 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. Epub 2018 Mar 12. PMID: 29544878.
52 Paithankar JG, Saini S, Dwivedi S, Sharma A, Chowdhuri DK. Heavy metal associated health hazards: An interplay of oxidative stress and signal transduction. Chemosphere. 2021 Jan;262:128350. doi: 10.1016/j.chemosphere.2020.128350. Epub 2020 Sep 16. PMID: 33182141.
53 Skalny AV, Lima TRR, Ke T, Zhou JC, Bornhorst J, Alekseenko SI, Aaseth J, Anesti O, Sarigiannis DA, Tsatsakis A, Aschner M, Tinkov AA. Toxic metal exposure as a possible risk factor for COVID-19 and other respiratory infectious diseases. Food Chem Toxicol. 2020 Dec;146:111809. doi: 10.1016/j.fct.2020.111809. Epub 2020 Oct 16. PMID: 33069759.
54 Park SK, Sack C, Sirén MJ, Hu H. Environmental Cadmium and Mortality from Influenza and Pneumonia in U.S. Adults. Environ Health Perspect. 2020 Dec;128(12):127004. doi: 10.1289/EHP7598. Epub 2020 Dec 16. PMID: 33325772; PMCID: PMC7739956.
55 Bao W, Liu B, Rong S, Dai SY, Trasande L, Lehmler HJ. Association Between Bisphenol A Exposure and Risk of All-Cause and Cause-Specific Mortality in US Adults. JAMA Netw Open. 2020 Aug 3;3(8):e2011620. doi: 10.1001/jamanetworkopen.2020.11620. PMID: 32804211.
56 Shams M, Alam I, Chowdhury I. Aggregation and stability of nanoscale plastics in aquatic environment. Water Res. 2020 Mar 15;171:115401. doi: 10.1016/j.watres.
57 Littman RA, Fiorenza EA, Wenger AS, Berry KLE, van de Water JAJM, Nguyen L, Aung ST, Parker DM, Rader DN, Harvell CD, Lamb JB. Coastal urbanization influences human pathogens and microdebris contamination in seafood. Sci Total Environ. 2020 Sep 20;736:139081. doi: 10.1016/j.scitotenv.2020.139081. Epub 2020 May 5. PMID: 32504866. Full text available at: **https://reader.elsevier.com/reader/sd/pii/S004896972032598 5?token=23B3D2A663EDB201D6A35C0CBD05743E27A8CC3764271EBDE52F4C120BCF8D98FDEA37ACBFC5DB15552D5F85163991C6**.
58 Ramsperger AFRM, Narayana VKB, Gross W, Mohanraj J, Thelakkat M, Greiner A, Schmalz H, Kress H, Laforsch C. Environmental exposure enhances the internalization of microplastic particles into cells. Sci Adv. 2020 Dec 9;6(50):eabd1211. doi: 10.1126/sciadv.abd1211. PMID: 33298447; PMCID: PMC7725476. **Full text in PDF format available at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7725476/pdf/abd1211.pdf.**
59 Hohn S, Acevedo-Trejos E, Abrams JF, Fulgencio de Moura J, Spranz R, Merico A. The long-term legacy of plastic mass production. Sci Total Environ. 2020 Jul 22;746:141115. doi: 10.1016/j.scitotenv.2020.141115. Online ahead of print. PMID: 32745856. **Full text in PDF format available at https://reader.elsevier.com/reader/sd/pii/S004896972034644 1?token=62B6F6998C28EE22EBDC5001A8E42C3B447D7FF50EBD924AC1902662421409122B8D7AA3B7F40715C2F7CBFC1524EC95.**
60 Egger M, Sulu-Gambari F, Lebreton L. First evidence of plastic fallout from the North Pacific Garbage Patch. Sci Rep. 2020 May 6;10(1):7495. doi: 10.1038/s41598-020-64465-8. PMID: 32376835. Full text in PDF format available at: **https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7203237/pdf/41598_2020_Article_64465.pdf**.
[61] https://www.who.int/teams/environment-climate-change-and-health/air-quality-and-health/ambient-air-pollution.

[62] Bakolis I, Hammoud R, Stewart R, Beevers S, Dajnak D, MacCrimmon S, Broadbent M, Pritchard M, Shiode N, Fecht D, Gulliver J, Hotopf M, Hatch SL, Mudway IS. Mental health consequences of urban air pollution: prospective population-based longitudinal survey. Soc Psychiatry Psychiatr Epidemiol. 2020 Oct 24:1–13. doi: 10.1007/s00127-020-01966-x. Epub ahead of print. PMID: 33097984; PMCID: PMC7584487. Full text in PDF format is available at: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7584487/pdf/127_2020_Article_1966.pdf.

63 Chung Y, Dominici F, Wang Y, Coull BA, Bell ML. Associations between long-term exposure to chemical constituents of fine particulate matter (PM2.5) and mortality in Medicare enrollees in the eastern United States. Environ Health Perspect. 2015 May;123(5):467-74.

64 Shi L, Zanobetti A, Kloog I, Coull BA, Koutrakis P, Melly SJ, Schwartz JD. Low-Concentration PM2.5 and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study. Environ Health Perspect. 2016 Jan;124(1):46-52.

65 Goldberg MS, Burnett RT. A new longitudinal design for identifying subgroups of the population who are susceptible to the short-term effects of ambient air pollution. J Toxicol Environ Health A. 2005 Jul 9-23;68(13-14):1111-25.

66 Zigler CM, Kim C, Choirat C, Hansen JB, Wang Y, Hund L, Samet J, King G, Dominici F; HEI Health Review Committee. Causal Inference Methods for Estimating Long-Term Health Effects of Air Quality Regulations. Res Rep Health Eff Inst. 2016 May;(187):5-49.

67 Stockfelt L, Andersson EM, Molnár P, Gidhagen L, Segersson D, Rosengren A, Barregard L, Sallsten G. Long-term effects of total and source-specific particulate air pollution on incident cardiovascular disease in Gothenburg, Sweden. Environ Res. 2017 Oct;158:61-71.

68 Zilong Zhang, Cui Guo, Alexis K.H. Lau, Ta-Chien Chan, Yuan Chieh Chuang, Changqing Lin, Wun Kai Jiang, Eng-kiong Yeoh, Tony Tam, Kam S. Woo, Bryan P. Yan, Ly-yun Chang, Martin C.S. Wong, Xiang Qian Lao Long-Term Exposure to Fine Particulate Matter, Blood Pressure, and Incident Hypertension in Taiwanese Adults Environ Health Perspect. 2018 Jan; 126(1): 017008.

69 Shen S, Li X, Yuan C, Huang Q, Liu D, Ma S, Hui J, Liu R, Wu T, Chen Q. Association of short-term exposure to sulfur dioxide and hospitalization for ischemic and hemorrhagic stroke in Guangzhou, China. BMC Public Health. 2020 Feb 21;20(1):263. doi: 10.1186/s12889-020-8354-0.

70 Byrne CP, Bennett KE, Hickey A, Kavanagh P, Broderick B, O'Mahony M, Williams DJ. Short-Term Air Pollution as a Risk for Stroke Admission: A Time-Series Analysis. Cerebrovasc Dis. 2020 Aug 10:1-8. doi: 10.1159/000510080. Online ahead of print. PMID: 32777785

71 Di Q, Wang Y, Zanobetti A, Wang Y, Koutrakis P, Choirat C, Dominici F, Schwartz JD. Air Pollution and Mortality in the Medicare Population. N Engl J Med. 2017 Jun 29;376(26):2513-2522.

72 Wang B, Eum KD, Kazemiparkouhi F, Li C, Manjourides J, Pavlu V, Suh H. The impact of long-term PM(2.5) exposure on specific causes of death: exposure-response curves and effect modification among 53 million U.S. Medicare beneficiaries. Environ Health. 2020 Feb 17;19(1):20. doi: 10.1186/s12940-020-00575-0. PMID: 32066433.

73 Hahad O, Lelieveld J, Birklein F, Lieb K, Daiber A, Münzel T. Ambient Air Pollution Increases the Risk of Cerebrovascular and Neuropsychiatric Disorders through Induction of Inflammation and Oxidative Stress. Int J Mol Sci. 2020 Jun 17;21(12):4306. doi: 10.3390/ijms21124306. PMID: 32560306

74 Guxens M, Lubczynska MJ, Muetzel RL, Dalmau-Bueno A, Jaddoe VWV, Hoek G, van der Lugt A, Verhulst FC, White T, Brunekreef B, Tiemeier H, El Marroun H. Air Pollution Exposure During Fetal Life, Brain Morphology, and Cognitive Function in School-Age Children. Biol Psychiatry. 2018 Aug 15;84(4):295-303.

75 Guxens M, Lubczynska MJ, Muetzel RL, Dalmau-Bueno A, Jaddoe VWV, Hoek G, van der Lugt A, Verhulst FC, White T, Brunekreef B, Tiemeier H, El Marroun H. Air Pollution Exposure During Fetal Life, Brain Morphology, and Cognitive Function in School-Age Children. Biol Psychiatry. 2018 Aug 15;84(4):295-303.

76 Sunyer J, Dadvand P. Prenatal brain development as a target for urban air pollution. Basic Clin Pharmacol Toxicol. 2019 Mar 18.

77 Guxens M, Lubczynska MJ, Muetzel RL, Dalmau-Bueno A, Jaddoe VWV, Hoek G, van der Lugt A, Verhulst FC, White T, Brunekreef B, Tiemeier H, El Marroun H. Air Pollution Exposure During Fetal Life, Brain Morphology, and Cognitive Function in School-Age Children. Biol Psychiatry. 2018 Aug 15;84(4):295-303.
78 Usemann J, Decrue F, Korten I, Proietti E, Gorlanova O, Vienneau D, Fuchs O, Latzin P, Röösli M, Frey U; BILD study group. Exposure to moderate air pollution and associations with lung function at school-age: A birth cohort study. Environ Int. 2019 May;126:682-689.
79 Schultz ES, Hallberg J, Bellander T, Bergström A, Bottai M, Chiesa F, Gustafsson PM, Gruzieva O, Thunqvist P, Pershagen G, Melén E. Early-Life Exposure to Traffic-related Air Pollution and Lung Function in Adolescence. Am J Respir Crit Care Med. 2016 Jan 15;193(2):171-7.
80 Breton CV, Wang X, Mack WJ, Berhane K, Lopez M, Islam TS, Feng M, Lurmann F, McConnell R, Hodis HN, Künzli N, Avol E. Childhood air pollutant exposure and carotid artery intima-media thickness in young adults. Circulation. 2012 Sep 25;126(13):1614-20.
81 Fu L, Yang X, Liu X, Yu G, Wang Z. Prenatal O(3) exposure increases the severity of OVA-induced asthma in offspring. Ecotoxicol Environ Saf. 2020 Jan 30;188:109867. doi: 10.1016/j.ecoenv.2019.109867. Epub 2019 Nov 2. PMID: 31689658
82 Martens DS, Cox B, Janssen BG, Clemente DBP, Gasparrini A, Vanpoucke C, Lefebvre W, Roels HA, Plusquin M, Nawrot TS. Prenatal Air Pollution and Newborns' Predisposition to Accelerated Biological Aging. JAMA Pediatr. 2017 Dec 1;171(12):1160-1167.
83 Gregory DJ, Kobzik L, Yang Z, McGuire CC, Fedulov AV. Transgenerational transmission of asthma risk after exposure to environmental particles during pregnancy. Am J Physiol Lung Cell Mol Physiol. 2017 Aug 1;313(2):L395-L405.
84 Moody EC, Cantoral A, Tamayo-Ortiz M, et al. Association of Prenatal and Perinatal Exposures to Particulate Matter With Changes in Hemoglobin A1c Levels in Children Aged 4 to 6 Years. JAMA Netw Open. 2019;2(12):e1917643. doi:10.1001/jamanetworkopen.2019.17643.
85 Zhou Y, Zhang M, Liu W, Li Y, Qin Y, Xu Y. Transgenerational transmission of neurodevelopmental disorders induced by maternal exposure to PM2.5. Chemosphere. 2020 Sep;255:126920. doi: 10.1016/j.chemosphere.2020.126920. Epub 2020 Apr 29. PMID: 32387734
86 Elten M, Benchimol EI, Fell DB, Kuenzig ME, Smith G, Chen H, Kaplan GG, Lavigne E. Ambient air pollution and the risk of pediatric-onset inflammatory bowel disease: A population-based cohort study. Environ Int. 2020 May;138:105676. doi: 10.1016/j.envint.2020.105676. Epub 2020 Mar 24. PMID: 32217428.
87 Ha S, Yeung E, Bell E, Insaf T, Ghassabian A, Bell G, Muscatiello N, Mendola P. Prenatal and early life exposures to ambient air pollution and development. Environ Res. 2019 Jul;174:170-175.
88 Mortamais M, Pujol J, Martínez-Vilavella G, Fenoll R, Reynes C, Sabatier R, Rivas I, Forns J, Vilor-Tejedor N, Alemany S, Cirach M, Alvarez-Pedrerol M, Nieuwenhuijsen M, Sunyer J. Effects of prenatal exposure to particulate matter air pollution on corpus callosum and behavioral problems in children. Environ Res. 2019 Nov;178:108734. doi: 10.1016/j.envres.2019.108734. Epub 2019 Sep 7. PMID: 31539824.
89 Gregory DJ, Kobzik L, Yang Z, McGuire CC, Fedulov AV. Transgenerational transmission of asthma risk after exposure to environmental particles during pregnancy. Am J Physiol Lung Cell Mol Physiol. 2017 Aug 1;313(2):L395-L405.
90 Loftus CT, Ni Y, Szpiro AA, Hazlehurst MF, Tylavsky FA, Bush NR, Sathyanarayana S, Carroll KN, Young M, Karr CJ, LeWinn KZ. Exposure to ambient air pollution and early childhood behavior: A longitudinal cohort study. Environ Res. 2020 Apr;183:109075. doi: 10.1016/j.envres.2019.109075. Epub 2019 Dec 23. PMID: 31999995.
91 Herting MM, Younan D, Campbell CE, Chen JC. Outdoor Air Pollution and Brain Structure and Function From Across Childhood to Young Adulthood: A Methodological Review of Brain MRI Studies. Front Public Health. 2019 Dec 6;7:332. doi: 10.3389/fpubh.2019.00332. eCollection 2019. PMID: 31867298.
92 Morgan HL, Watkins AJ. Transgenerational Impact of Environmental Change. Adv Exp Med Biol. 2019;1200:71-89. doi: 10.1007/978-3-030-23633-5_4. PMID: 31471795.

93 Zhou Y, Zhang M, Liu W, Li Y, Qin Y, Xu Y. Transgenerational transmission of neurodevelopmental disorders induced by maternal exposure to PM2.5. Chemosphere. 2020 Sep;255:126920. doi: 10.1016/j.chemosphere.2020.126920. Epub 2020 Apr 29. PMID: 32387734.
94 Ouidir M, Mendola P, Buck Louis GM, Kannan K, Zhang C, Tekola-Ayele F. Concentrations of persistent organic pollutants in maternal plasma and epigenome-wide placental DNA methylation. Clin Epigenetics. 2020 Jul 11;12(1):103. doi: 10.1186/s13148-020-00894-6. PMID: 32653021.
95 Saenen ND, Martens DS, Neven KY, Alfano R, Bové H, Janssen BG, Roels HA, Plusquin M, Vrijens K, Nawrot TS. Air pollution-induced placental alterations: an interplay of oxidative stress, epigenetics, and the aging phenotype? Clin Epigenetics. 2019 Sep 17;11(1):124. doi: 10.1186/s13148-019-0688-z. PMID: 31530287.
96 He QL, Lyu TQ, Zhang YT, Wang HQ, Zhou Q, Zhang JM, Liu YY, Li JS, Jiang LG, Cheng D, Ge ZJ, Liu SZ. Effects of intrauterine exposure to 2,3',4,4',5-pentachlorobiphenyl on the reproductive system and sperm epigenetic imprinting of male offspring. J Appl Toxicol. 2020 May 17. doi: 10.1002/jat.3992. Online ahead of print. PMID: 32418265.
[97] Russ TC, Cherrie MPC, Dibben C, Tomlinson S, Reis S, Dragosits U, Vieno M, Beck R, Carnell E, Shortt NK, Muniz-Terrera G, Redmond P, Taylor AM, Clemens T, van Tongeren M, Agius RM, Starr JM, Deary IJ, Pearce JR. Life Course Air Pollution Exposure and Cognitive Decline: Modelled Historical Air Pollution Data and the Lothian Birth Cohort 1936. J Alzheimers Dis. 2021 Jan 8. doi: 10.3233/JAD-200910. Epub ahead of print. PMID: 33427734.
[98] Volk HE, Perera F, Braun JM, Kingsley SL, Gray K, Buckley J, Clougherty JE, Croen LA, Eskenazi B, Herting M, Just AC, Kloog I, Margolis A, McClure LA, Miller R, Levine S, Wright R; Environmental influences on Child Health Outcomes. Prenatal air pollution exposure and neurodevelopment: A review and blueprint for a harmonized approach within ECHO. Environ Res. 2020 Oct 22:110320. doi: 10.1016/j.envres.2020.110320. Epub ahead of print. PMID: 33098817.
99 Van Deusen A, Hyland A, Travers MJ, Wang C, Higbee C, King BA, Alford T, Cummings KM. Secondhand smoke and particulate matter exposure in the home. Nicotine Tob Res. 2009 Jun;11(6):635-41.
100 MacNeill M, Kearney J, Wallace L, Gibson M, Héroux ME, Kuchta J, Guernsey JR, Wheeler AJ. Quantifying the contribution of ambient and indoor-generated fine particles to indoor air in residential environments. Indoor Air. 2014 Aug;24(4):362-75.
101 Morin PC, Rosenbaum PF, Abraham JL, Weinstock RS. Poor air quality in homes of Medicare recipients with diabetes. Home Healthc Nurse. 2014 Jun;32(6):354-61.
102 Delgado-Rendon A, Cruz TB, Soto D, Baezconde-Garbanati L, Unger JB. Second and Thirdhand Smoke Exposure, Attitudes and Protective Practices: Results from a Survey of Hispanic Residents in Multi-unit Housing. J Immigr Minor Health. 2017 Jan 10.
103 Jacob P 3rd, Benowitz NL, Destaillats H, Gundel L, Hang B, Martins-Green M, Matt GE, Quintana PJ, Samet JM, Schick SF, Talbot P, Aquilina NJ, Hovell MF, Mao JH, Whitehead TP. Thirdhand Smoke: New Evidence, Challenges, and Future Directions. Chem Res Toxicol. 2017 Jan 17;30(1):270-294.
104 Repace JL, Jiang RT, Acevedo-Bolton V, Cheng KC, Klepeis NE, Ott WR, Hildemann LM. Fine particle air pollution and secondhand smoke exposures and risks inside 66 US casinos. Environ Res. 2011 May;111(4):473-84.
105 Zhou Z, Bohac D, Boyle RG. Continuous weeklong measurements of indoor particle levels in a Minnesota Tribal Casino Resort. BMC Public Health. 2016 Aug 24;16(1):870.
106 Tsai WT. An overview of health hazards of volatile organic compounds regulated as indoor air pollutants. Rev Environ Health. 2019 Mar 26;34(1):81-89.
107 Siponen T, Yli-Tuomi T, Tiittanen P, Taimisto P, Pekkanen J, Salonen RO, Lanki T. Wood stove use and other determinants of personal and indoor exposures to particulate air pollution and ozone among elderly persons in a Northern Suburb. Indoor Air. 2019 Feb 20.
108 Maddela NR, Venkateswarlu K, Kakarla D, Megharaj M. Inevitable human exposure to emissions of polybrominated diphenyl ethers: A perspective on potential health risks. Environ Pollut. 2020 Jul 14;266(Pt 1):115240. doi: 10.1016/j.envpol.2020.115240. Online ahead of print. PMID: 32698055
109 Mohd Isa KN, Hashim Z, Jalaludin J, Lung Than LT, Hashim JH. The Effects of Indoor Pollutants Exposure on Allergy and Lung Inflammation: An Activation State of Neutrophils and Eosinophils in Sputum. Int J Environ Res Public Health. 2020 Jul 28;17(15):E5413. doi: 10.3390/ijerph17155413. PMID: 32731346.

110 Branco PTBS, Alvim-Ferraz MCM, Martins FG, Ferraz C, Vaz LG, Sousa SIV. Impact of indoor air pollution in nursery and primary schools on childhood asthma. Sci Total Environ. 2020 Jul 18;745:140982. doi: 10.1016/j.scitotenv.2020.140982. Online ahead of print. PMID: 32736106
111 Ferri GM, Intranuovo G, Cavone D, Corrado V, Birtolo F, Tricase P, Fuso R, Vilardi V, Sumerano M, L'abbate N, Vimercati L. Estimates of the Lung Cancer Cases Attributable to Radon in Municipalities of Two Apulia Provinces (Italy) and Assessment of Main Exposure Determinants. Int J Environ Res Public Health. 2018 Jun 20;15(6):1294. doi: 10.3390/ijerph15061294. PMID: 29925825; PMCID: PMC6025095.
112 Markwald RR, Melanson EL, Smith MR, Higgins J, Perreault L, Eckel RH, Wright KP Jr. Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain. Proc Natl Acad Sci U S A. 2013 Apr 2;110(14):5695-700.
113 Depner CM, Stothard ER, Wright KP Jr. Metabolic consequences of sleep and circadian disorders. Curr Diab Rep. 2014 Jul;14(7):507.
114 Broussard JL, Van Cauter E. Disturbances of sleep and circadian rhythms: novel risk factors for obesity. Curr Opin Endocrinol Diabetes Obes. 2016 Oct;23(5):353-9.
115 McHill AW, Wright KP Jr. Role of sleep and circadian disruption on energy expenditure and in metabolic predisposition to human obesity and metabolic disease. Obes Rev. 2017 Feb;18 Suppl 1:15-24.
116 Simpson NS, Gibbs EL, Matheson GO. Optimizing sleep to maximize performance: implications and recommendations for elite athletes. Scand J Med Sci Sports. 2017 Mar;27(3):266-274.
117 Antunes BM, Campos EZ, Parmezzani SS, Santos RV, Franchini E, Lira FS. Sleep quality and duration are associated with performance in maximal incremental test. Physiol Behav. 2017 May 11;177:252-256.
118 Chase JD, Roberson PA, Saunders MJ, Hargens TA, Womack CJ, Luden ND. One Night of Sleep Restriction Following Heavy Exercise Impairs 3-km Cycling Time Trial Performance in the Morning. Appl Physiol Nutr Metab. 2017 Sep;42(9):909-915. doi: 10.1139/apnm-2016-0698. Epub 2017 May 3. PMID: 28467857.
119 Yannakoulia M, Anastasiou CA, Karfopoulou E, Pehlivanidis A, Panagiotakos DB, Vgontzas A. Sleep quality is associated with weight loss maintenance status: the MedWeight study. Sleep Med. 2017 Jun;34:242-245.
120 Liu J, Ghastine L, Um P, Rovit E, Wu T. Environmental exposures and sleep outcomes: A review of evidence, potential mechanisms, and implications. Environ Res. 2020 Oct 29:110406. doi: 10.1016/j.envres.2020.110406. Epub ahead of print. PMID: 33130170.
121 Khan A, Plana-Ripoll O, Antonsen S, Brandt J, Geels C, Landecker H, Sullivan PF, Pedersen CB, Rzhetsky A. Environmental pollution is associated with increased risk of psychiatric disorders in the US and Denmark. PLoS Biol. 2019 Aug 20;17(8):e3000353. doi: 10.1371/journal.pbio.3000353. eCollection 2019 Aug.
122 Barouki R, Melén E, Herceg Z, Beckers J, Chen J, Karagas M, Puga A, Xia Y, Chadwick L, Yan W, Audouze K, Slama R, Heindel J, Grandjean P, Kawamoto T, Nohara K. Epigenetics as a mechanism linking developmental exposures to long-term toxicity. Environ Int. 2018 Feb 27;114:77-86.
123 Barouki R, Melén E, Herceg Z, Beckers J, Chen J, Karagas M, Puga A, Xia Y, Chadwick L, Yan W, Audouze K, Slama R, Heindel J, Grandjean P, Kawamoto T, Nohara K. Epigenetics as a mechanism linking developmental exposures to long-term toxicity. Environ Int. 2018 Feb 27;114:77-86.
124 Martos SN, Tang WY, Wang Z. Elusive inheritance: Transgenerational effects and epigenetic inheritance in human environmental disease. Prog Biophys Mol Biol. 2015 Jul;118(1-2):44-54.
125 Shukla A, Bunkar N, Kumar R, Bhargava A, Tiwari R, Chaudhury K, Goryacheva IY, Mishra PK. Air pollution associated epigenetic modifications: Transgenerational inheritance and underlying molecular mechanisms. Sci Total Environ. 2019 Mar 15;656:760-777.
126 Saenen ND, Martens DS, Neven KY, Alfano R, Bové H, Janssen BG, Roels HA, Plusquin M, Vrijens K, Nawrot TS. Air pollution-induced placental alterations: an interplay of oxidative stress, epigenetics, and the aging phenotype? Clin Epigenetics. 2019 Sep 17;11(1):124. doi: 10.1186/s13148-019-0688-z. PMID: 31530287.

127 Hermanova B, Riedlova P, Dalecka A, Jirik V, Janout V, Sram RJ. Air pollution and molecular changes in age-related diseases. Int J Environ Health Res. 2020 Jul 29:1-19. doi: 10.1080/09603123.2020.1797643. Online ahead of print. PMID: 32723182.
128 Zhu Y, Costa M. Metals and Molecular Carcinogenesis. Carcinogenesis. 2020 Jul 17:bgaa076. doi: 10.1093/carcin/bgaa076. Online ahead of print. PMID: 32674145.
129 Hermanova B, Riedlova P, Dalecka A, Jirik V, Janout V, Sram RJ. Air pollution and molecular changes in age-related diseases. Int J Environ Health Res. 2020 Jul 29:1-19.
130 Finicelli M, Squillaro T, Galderisi U, Peluso G. Micro-RNAs: Crossroads between the Exposure to Environmental Particulate Pollution and the Obstructive Pulmonary Disease. Int J Mol Sci. 2020 Sep 30;21(19):7221. doi: 10.3390/ijms21197221. PMID: 33007849.
131 Shin CH, Byun J, Lee K, Kim B, Noh YK, Tran NL, Park K, Kim SH, Kim TH, Oh SJ. Exosomal miRNA-19a and miRNA-614 Induced by Air Pollutants Promote Proinflammatory M1 Macrophage Polarization via Regulation of RORalpha Expression in Human Respiratory Mucosal Microenvironment. J Immunol. 2020 Dec 1;205(11):3179-3190.
132 Hernández ÁR, Boada LD, Mendoza Z, Ruiz-Suárez N, Valerón PF, Camacho M, Zumbado M, Almeida-González M, Henríquez-Hernández LA, Luzardo OP. 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.
133 Dervilly-Pinel G, Guérin T, Minvielle B, Travel A, Normand J, Bourin M, Royer E, Dubreil E, Mompelat S, Hommet F, Nicolas M, Hort V, Inthavong C, Saint-Hilaire M, Chafey C, Parinet J, Cariou R, Marchand P, Le Bizec B, Verdon E, Engel E. Micropollutants and chemical residues in organic and conventional meat. Food Chem. 2017 Oct 1;232:218-228.
134 Grigoratos T, Martini G. Brake wear particle emissions: a review. Environ Sci Pollut Res Int. 2015 Feb;22(4):2491-504. doi: 10.1007/s11356-014-3696-8. Epub 2014 Oct 17. PMID: 25318420; PMCID: PMC4315878.
135 Askariyeh MH, Venugopal M, Khreis H, Birt A, Zietsman J. Near-Road Traffic-Related Air Pollution: Resuspended PM2.5 from Highways and Arterials. Int J Environ Res Public Health. 2020 Apr 21;17(8):2851. doi: 10.3390/ijerph17082851. PMID: 32326193; PMCID: PMC7215985.
136 Adamiec E, Jarosz-Krzeminska E, Wieszala R. Heavy metals from non-exhaust vehicle emissions in urban and motorway road dusts. Environ Monit Assess. 2016 Jun;188(6):369. doi: 10.1007/s10661-016-5377-1. Epub 2016 May 26. PMID: 27226173; PMCID: PMC4880625.
137 Sheu R, Stönner C, et al. Human transport of thirdhand tobacco smoke: A prominent source of hazardous air pollutants into indoor nonsmoking environments. Science Advances, 2020; 6 (10): eaay4109 DOI: 10.1126/sciadv.aay4109.
138 Eric Praske, Rasmus V. Otkjær, John D. Crounse, J. Caleb Hethcox, Brian M. Stoltz, Henrik G. Kjaergaard, Paul O. Wennberg. Atmospheric autoxidation is increasingly important in urban and suburban North America. Proceedings of the National Academy of Sciences, 2017; 201715540 DOI: 10.1073/pnas.1715540115.
139 Zulauf N, Dröge J, Klingelhöfer D, Braun M, Oremek GM, Groneberg DA. Indoor Air Pollution in Cars: An Update on Novel Insights. Int J Environ Res Public Health. 2019 Jul 9;16(13):2441. doi: 10.3390/ijerph16132441. PMID: 31323996; PMCID: PMC6650813.
140 Reddam A, Tait G, Herkert N, Hammel SC, Stapleton HM, Volz DC. Longer commutes are associated with increased human exposure to tris(1,3-dichloro-2-propyl) phosphate. Environ Int. 2020 Jan 27;136:105499. doi: 10.1016/j.envint.2020.105499. [Epub ahead of print]
141 Matta MK, Zusterzeel R, Pilli NR, Patel V, Volpe DA, Florian J, Oh L, Bashaw E, Zineh I, Sanabria C, Kemp S, Godfrey A, Adah S, Coelho S, Wang J, Furlong LA, Ganley C, Michele T, Strauss DG. Effect of Sunscreen Application Under Maximal Use Conditions on Plasma Concentration of Sunscreen Active Ingredients: A Randomized Clinical Trial. JAMA. 2019 May 6. doi: 10.1001/jama.2019.5586.
142 Aylward L, Vilone G, Cowan-Ellsberry C, Arnot JA, Westgate JN, O'Mahony C, Hays SM. Exposure to selected preservatives in personal care products: case study comparison of exposure models and observational biomonitoring data. J Expo Sci Environ Epidemiol. 2020 Jan;30(1):28-41. **Full text available at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6914665/**.

143 Leppert B, Strunz S, Seiwert B, Schlittenbauer L, Schlichting R, Pfeiffer C, Röder S, Bauer M, Borte M, Stangl GI, Schöneberg T, Schulz A, Karkossa I, Rolle-Kampczyk UE, Thürmann L, von Bergen M, Escher BI, Junge KM, Reemtsma T, Lehmann I, Polte T. Maternal paraben exposure triggers childhood overweight development. Nat Commun. 2020 Feb 11;11(1):561. doi: 10.1038/s41467-019-14202-1.
144 Khlystov A, Samburova V. Flavoring Compounds Dominate Toxic Aldehyde Production during E-Cigarette Vaping. Environ Sci Technol. 2016 Dec 6;50(23):13080-13085.
145 Clapp PW, Jaspers I. Electronic Cigarettes: Their Constituents and Potential Links to Asthma. Curr Allergy Asthma Rep. 2017 Oct 5;17(11):79.
146 Glynos C, Bibli SI, Katsaounou P, Pavlidou A, Magkou C, Karavana V, Topouzis S, Kalomenidis I, Zakynthinos S, Papapetropoulos A. Comparison of the effects of e-cigarette vapor with cigarette smoke on lung function and inflammation in mice. Am J Physiol Lung Cell Mol Physiol. 2018 Nov 1;315(5):L662-L672.
147 Caporale A, Langham MC, Guo W, Johncola A, Chatterjee S, Wehrli FW. Acute Effects of Electronic Cigarette Aerosol Inhalation on Vascular Function Detected at Quantitative MRI. Radiology. 2019 Aug 20:190562. doi: 10.1148/radiol.2019190562. [Epub ahead of print]
148 Rankin GD, Wingfors H, Uski O, Hedman L, Ekstrand-Hammarström B, Bosson J, Lundbäck M. The toxic potential of a fourth-generation E-cigarette on human lung cell lines and tissue explants. J Appl Toxicol. 2019 Apr 8. doi: 10.1002/jat.3799. [Epub ahead of print]
149 Son Y, Mishin V, Laskin JD, Mainelis G, Wackowski OA, Delnevo C, Schwander S, Khlystov A, Samburova V, Meng Q. Hydroxyl Radicals in E-Cigarette Vapor and E-Vapor Oxidative Potentials under Different Vaping Patterns. Chem Res Toxicol. 2019 Apr 23. doi: 10.1021/acs.chemrestox.8b00400. [Epub ahead of print]
150 Schober W, Szendrei K, Matzen W, Osiander-Fuchs H, Heitmann D, Schettgen T, Jörres RA, Fromme H. Use of electronic cigarettes (e-cigarettes) impairs indoor air quality and increases FeNO levels of e-cigarette consumers. Int J Hyg Environ Health. 2014 Jul;217(6):628-37.
151 Geiss O, Bianchi I, Barahona F, Barrero-Moreno J. Characterisation of mainstream and passive vapours emitted by selected electronic cigarettes. Int J Hyg Environ Health. 2015 Jan;218(1):169-80.
152 Wang P, Chen W, Liao J, Matsuo T, Ito K, Fowles J, Shusterman D, Mendell M, Kumagai K. A Device-Independent Evaluation of Carbonyl Emissions from Heated Electronic Cigarette Solvents. PLoS One. 2017 Jan 11;12(1):e0169811. doi: 10.1371/journal.pone.0169811. eCollection 2017.
153 Visser WF, Klerx WN, Cremers HWJM, Ramlal R, Schwillens PL, Talhout R. The Health Risks of Electronic Cigarette Use to Bystanders. Int J Environ Res Public Health. 2019 Apr 30;16(9). pii: E1525. doi: 10.3390/ijerph16091525.
154 Papaefstathiou E, Stylianou M, Agapiou A. Main and side stream effects of electronic cigarettes. J Environ Manage. 2019 May 15;238:10-17.
155 Yen YC, Yang CY, Mena KD, Cheng YT, Yuan CS, Chen PS. Jumping on the bed and associated increases of PM10, PM2.5, PM1, airborne endotoxin, bacteria, and fungi concentrations. Environ Pollut. 2019 Feb;245:799-809. d
156 Guo NL, Poh TY, Pirela S, Farcas MT, Chotirmall SH, Tham WK, Adav SS, Ye Q, Wei Y, Shen S, Christiani DC, Ng KW, Thomas T, Qian Y, Demokritou P. Integrated Transcriptomics, Metabolomics, and Lipidomics Profiling in Rat Lung, Blood, and Serum for Assessment of Laser Printer-Emitted Nanoparticle Inhalation Exposure-Induced Disease Risks. Int J Mol Sci. 2019 Dec 16;20(24). pii: E6348. doi: 10.3390/ijms20246348. PMID: 31888290
157 Steinvil A, Shmueli H, Ben-Assa E, Leshem-Rubinow E, Shapira I, Berliner S, Kordova-Biezuner L, Rogowski O. Environmental exposure to combustion-derived air pollution is associated with reduced functional capacity in apparently healthy individuals. Clin Res Cardiol. 2013 Aug;102(8):583-91.
158 Wang Y, Xiong L, Tang M. Toxicity of inhaled particulate matter on the central nervous system: neuroinflammation, neuropsychological effects and neurodegenerative disease. J Appl Toxicol. 2017 Jun;37(6):644-667.
159 Kargarfard M, Poursafa P, Rezanejad S, Mousavinasab F. Effects of exercise in polluted air on the aerobic power, serum lactate level and cell blood count of active individuals. Int J Prev Med. 2011 Jul;2(3):145-50

160 Bos I, De Boever P, Vanparijs J, Pattyn N, Panis LI, Meeusen R. Subclinical effects of aerobic training in urban environment. Med Sci Sports Exerc. 2013 Mar;45(3):439-47.
161 Kargarfard M, Shariat A, Shaw BS, Shaw I, Lam ET, Kheiri A, Eatemadyboroujeni A, Tamrin SB. Effects of polluted air on cardiovascular and hematological parameters after progressive maximal aerobic exercise. Lung. 2015 Apr;193(2):275-81.
162 Boussetta N, Abedelmalek S, Aloui K, Souissi N. The effect of air pollution on diurnal variation of performance in anaerobic tests, cardiovascular and hematological parameters, and blood gases on soccer players following the Yo-Yo Intermittent Recovery Test Level-1. Chronobiol Int. 2017;34(7):903-920.
163 An R, Zhang S, Ji M, Guan C. Impact of ambient air pollution on physical activity among adults: a systematic review and meta-analysis. Perspect Public Health. 2018 Mar;138(2):111-121.
164 Zhong J, Trevisi L, Urch B, Lin X, Speck M, Coull BA, Liss G, Thompson A, Wu S, Wilson A, Koutrakis P, Silverman F, Gold DR, Baccarelli AA. B-vitamin Supplementation Mitigates Effects of Fine Particles on Cardiac Autonomic Dysfunction and Inflammation: A Pilot Human Intervention Trial. Sci Rep. 2017 Apr 3;7:45322.
165 Zhong J, Karlsson O, Wang G, Li J, Guo Y, Lin X, Zemplenyi M, Sanchez-Guerra M, Trevisi L, Urch B, Speck M, Liang L, Coull BA, Koutrakis P, Silverman F, Gold DR, Wu T, Baccarelli AA. B vitamins attenuate the epigenetic effects of ambient fine particles in a pilot human intervention trial. Proc Natl Acad Sci U S A. 2017 Mar 28;114(13):3503-3508.
166 Xuan L L, Shi J, Yao C S, Bai J Y, Qu F, Zhang J L, et al. Vam3, a resveratrol dimer, inhibits cigarette smoke-induced cell apoptosis in lungs by improving mitochondrial function[J]. Acta Pharmacol Sin, 2014, 35(6):779±791.
167 Blaylock RL, Maroon J. Natural plant products and extracts that reduce immunoexcitotoxicity-associated neurodegeneration and promote repair within the central nervous system. Surg Neurol Int. 2012;3:19.
168 Rangarajan P, Karthikeyan A, Dheen ST. Role of dietary phenols in mitigating microglia-mediated neuroinflammation. Neuromolecular Med. 2016 Sep;18(3):453-64.
169 Polley KR, Jenkins N, O'Connor P, McCully K. Influence of exercise training with resveratrol supplementation on skeletal muscle mitochondrial capacity. Appl Physiol Nutr Metab. 2016 Jan;41(1):26-32.
170 Cheserek MJ, Wu G, Li L, Li L, Karangwa E, Shi Y, Le G. Cardioprotective effects of lipoic acid, quercetin and resveratrol on oxidative stress related to thyroid hormone alterations in long-term obesity. J Nutr Biochem. 2016 Jul;33:36-44.
171 Fan L, Feng Y, Chen GC, Qin LQ, Fu CL, Chen LH. Effects of coenzyme Q10 supplementation on inflammatory markers: A systematic review and meta-analysis of randomized controlled trials. Pharmacol Res. 2017 May;119:128-136.
172 Sung D, Kim S, Kim J, An H, So W. Role of l-carnitine in sports performance: Focus on ergogenic aid and antioxidant Science & Sports (2016) 31, 177—188
173 Islam MA, Alam F, Solayman M, Khalil MI, Kamal MA, Gan SH. Dietary Phytochemicals: Natural Swords Combating Inflammation and Oxidation-Mediated Degenerative Diseases. Oxid Med Cell Longev. 2016;2016:5137431.
174 Gormaz JG, Valls N, Sotomayor C, Turner T, Rodrigo R. Potential Role of Polyphenols in the Prevention of Cardiovascular Diseases: Molecular Bases. Curr Med Chem. 2016;23(2):115-28.
175 Upadhyay S, Dixit M. Role of Polyphenols and Other Phytochemicals on Molecular Signaling. Oxid Med Cell Longev. 2015;2015:504253.
176 Montesano A, Senesi P, Luzi L, Benedini S, Terruzzi I. Potential therapeutic role of L-carnitine in skeletal muscle oxidative stress and atrophy conditions. Oxid Med Cell Longev. 2015;2015:646171.
177 Whyand T, Hurst JR, Beckles M, Caplin ME. Pollution and respiratory disease: can diet or supplements help? A review. Respir Res. 2018 May 2;19(1):79.
178 Manson J. Vitamin D and Omega-3 Supplements for Preventing Cancer and Other Chronic Diseases. Oncology (Williston Park). 2019 Jan 17;33(1):36-8.
179 Tong H, Rappold AG, Diaz-Sanchez D, Steck SE, Berntsen J, Cascio WE, Devlin RB, Samet JM. Omega-3 fatty acid supplementation appears to attenuate particulate air pollution-induced cardiac

effects and lipid changes in healthy middle-aged adults. Environ Health Perspect. 2012 Jul;120(7):952-7.
180 Li XY, Hao L, Liu YH, Chen CY, Pai VJ, Kang JX. Protection against fine particle-induced pulmonary and systemic inflammation by omega-3 polyunsaturated fatty acids. Biochim Biophys Acta. 2017 Mar;1861(3):577-584.
181 Siasos G, Tousoulis D, Oikonomou E, Zaromitidou M, Verveniotis A, Plastiras A, Kioufis S, Maniatis K, Miliou A, Siasou Z, Stefanadis C, Papavassiliou AG. Effects of O-3 fatty acids on endothelial function, arterial wall properties, inflammatory and fibrinolytic status in smokers: a cross over study. Int J Cardiol. 2013 Jun 20;166(2):340-6.
182 Wiest EF, Walsh-Wilcox MT, Walker MK. Omega-3 Polyunsaturated Fatty Acids Protect Against Cigarette Smoke-Induced Oxidative Stress and Vascular Dysfunction. Toxicol Sci. 2017 Mar 1;156(1):300-310.
183 Frei M, Kim C, Ames BN. Ubiquinol-10 is an effective lipid-soluble antioxidant at physiological concentrations. Proc Natl Acad Sci 1990; 87:4879–83.
184 Schmelzer C, Kubo H, Mori M, Sawashita J, Kitano M, Hosoe K, Boomgaarden I, Döring F, Higuchi K. Supplementation with the reduced form of Coenzyme Q10 decelerates phenotypic characteristics of senescence and induces a peroxisome proliferator-activated receptor-alpha gene expression signature in SAMP1 mice. Mol Nutr Food Res. 2010 Jun;54(6):805-15.
185 Tian G, Sawashita J, Kubo H, Nishio SY, Hashimoto S, Suzuki N, Yoshimura H, Tsuruoka M, Wang Y, Liu Y, Luo H, Xu Z, Mori M, Kitano M, Hosoe K, Takeda T, Usami S, Higuchi K. Ubiquinol-10 supplementation activates mitochondria functions to decelerate senescence in senescence-accelerated mice. Antioxid Redox Signal. 2014 Jun 1;20(16):2606-20.
186 Yan J, Fujii K, Yao J, Kishida H, Hosoe K, Sawashita J, Takeda T, Mori M, Higuchi K. Reduced coenzyme Q10 supplementation decelerates senescence in SAMP1 mice. Exp Gerontol. 2006 Feb;41(2):130-40.
187 Garrido-Maraver J, Cordero MD, Oropesa-Ávila M, Fernández Vega A, de la Mata M, Delgado Pavón A, de Miguel M, Pérez Calero C, Villanueva Paz M, Cotán D, Sánchez-Alcázar JA. Coenzyme q10 therapy. Mol Syndromol. 2014 Jul;5(3-4):187-97.
188 Safarinejad MR. Efficacy of coenzyme Q10 on semen parameters, sperm function and reproductive hormones in infertile men. J Urol. 2009 Jul;182(1):237-48.
189 Fouad AA, Al-Sultan AI, Yacoubi MT. Coenzyme Q10 counteracts testicular injury induced by sodium arsenite in rats. Eur J Pharmacol. 2011 Mar 25;655(1-3):91-8.
190 Walczak-Jedrzejowska R, Wolski JK, Slowikowska-Hilczer J. The role of oxidative stress and antioxidants in male fertility. Cent European J Urol. 2013;66(1):60-7.
191 Safarinejad MR. The effect of coenzyme Q10 supplementation on partner pregnancy rate in infertile men with idiopathic oligoasthenoteratozoospermia: an open-label prospective study. Int Urol Nephrol. 2012 Jun;44(3):689-700.
192 Banihani SA. Effect of Coenzyme Q10 Supplementation on Testosterone. Biomolecules. 2018 Dec 13;8(4). pii: E172. doi: 10.3390/biom8040172.
193 Banihani SA. Effect of Coenzyme Q10 Supplementation on Testosterone. Biomolecules. 2018 Dec 13;8(4). pii: E172. doi: 10.3390/biom8040172.
194 Hornos Carneiro MF, Shin N, Karthikraj R, Barbosa F Jr, Kannan K, Colaiácovo MP. Antioxidant CoQ10 Restores Fertility by Rescuing Bisphenol A-Induced Oxidative DNA Damage in the Caenorhabditis elegans Germline. Genetics. 2020 Feb;214(2):381-395.
195 Ghanbarzadeh S, Garjani A, Ziaee M, Khorrami A. Effects of L-carnitine and coenzyme q10 on impaired spermatogenesis caused by isoproterenol in male rats. Drug Res (Stuttg). 2014 Sep;64(9):449-53.
196 Ghanbarzadeh S, Garjani A, Ziaee M, Khorrami A. CoQ10 and L-carnitine attenuate the effect of high LDL and oxidized LDL on spermatogenesis in male rats. Drug Res (Stuttg). 2014 Oct;64(10):510-5.
197 Dokmeci D, Inan M, Basaran UN, Yalcin O, Aydogdu N, Turan FN, Uz YH. Protective effect of L-carnitine on testicular ischaemia-reperfusion injury in rats. Cell Biochem Funct. 2007 Nov-Dec;25(6):611-8.

198 Cavallini G, Caracciolo S, Vitali G, Modenini F, Biagiotti G. Carnitine versus androgen administration in the treatment of sexual dysfunction, depressed mood, and fatigue associated with male aging. Urology. 2004 Apr;63(4):641-6.
199 Zare Z, Eimani H, Mohammadi M, Mofid M, Dashtnavard H. The effect of orally administered l-carnitine on testis tissue, sperm parameters and daily sperm production in adult mice. Yakhteh Med J. 2010;11:382–389.
200 Sazegar G, Ebrahimi V, Boroujeni MJS, Mohammadi S, Salimnezhad R. Morphometric study of testis tissue and spermatogenesis following carnitine administration in diabetic rat induced with stereptozotocin. Iran J Diabetes Metab. 2014;14:9–14.
201 Kanter M, Topcu-Tarladacalisir Y, Parlar S. Antiapoptotic effect of l-carnitine on testicular irradiation in rats. J Mol Histol. 2010;41:121–128.
202 Rezaei N, Mardanshahi T, Shafaroudi MM, Abedian S, Mohammadi H, Zare Z. Effects of l-Carnitine on the Follicle-Stimulating Hormone, Luteinizing Hormone, Testosterone, and Testicular Tissue Oxidative Stress Levels in Streptozotocin-Induced Diabetic Rats. J Evid Based Integr Med. 2018 Jan-Dec;23:2515690X18796053. doi: 10.1177/2515690X18796053.
203 Elokil AA, Bhuiyan AA, Liu HZ, Hussein MN, Ahmed HI, Azmal SA, Yang L, Li S. The capability of L-carnitine-mediated antioxidant on cock during aging: evidence for the improved semen quality and enhanced testicular expressions of GnRH1, GnRHR, and melatonin receptors MT 1/2. Poult Sci. 2019 Sep 1;98(9):4172-4181. doi: 10.3382/ps/pez201. PMID: 31001634.