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CLIMATE CHANGE, ENVIRONMENTAL JUSTICE, AND COVID-19:

THE PERFECT STORM

Pamela J. Lein, PhD
Allison K. Ehrlich, PhD
Lisa A. Miller, PhD
Laura S. Van Winkle, PhD

University of California, Davis

"People with chronic health conditions, lower-income, and communities of color are disproportionately impacted by both COVID-19 and climate change, and environmental pollution is at the heart of both problems. We owe it to everyone to improve health, and we do that by reducing the sources of pollution that drive a large burden of disease in the United States and around the world."

Dr. Aaron Bernstein, Harvard T.H. Chan School of Public Health

Introduction

The COVID-19 pandemic is an unprecedented global public health challenge that has resulted in staggering human loss around the world. As of November 21, 2020, there are more than 57 million confirmed cases in 191 countries with over 1.37 million deaths from the disease caused by the SARS-CoV-2 virus. Even more sobering, these numbers continue to increase exponentially and are not expected to decrease any time soon. Health outcomes of COVID-19 vary widely from asymptomatic to mild respiratory symptoms to life-threatening illness. Older people and those with underlying health conditions, including heart and lung disease, diabetes, obesity, and cancer, are at much greater risk for developing more severe illness.

Although COVID-19 is widespread, its impacts vary dramatically among subgroups of people. In many parts of the world, communities of colour and disadvantaged communities are disproportionately affected. For example, Black, Asian, and other minority ethnic groups in the United Kingdom are at significantly higher risk of both contracting and dying from the virus. Similarly, in the United States, Black, Indigenous, and LatinX Americans are three times more likely to become infected and nearly twice as likely to die from COVID-19, as are whites. This increased vulnerability to infection with SARS-CoV-2 and severe outcomes from COVID-19 is widely attributed to higher rates of pre-existing health issues, such as asthma, hypertension, diabetes, and obesity, within these communities. While genetic and lifestyle factors play a role in determining individual susceptibility to these health conditions, they do not fully explain the racial disparities in COVID-19 health-related outcomes. An additional contributing factor is likely increased exposure to the virus because individuals in disadvantaged communities often do not have the resources to work from home or are employed in service or production jobs that cannot be done remotely. Further, disadvantaged communities tend to rely more on public transportation, live in cramped apartments with a single bathroom or multigenerational homes, and often need to keep working even in risky settings. Exposure to environmental pollution represents another important piece of the puzzle underlying the increased incidence and severity of COVID-19 in communities of colour and disadvantaged communities. In many countries, there are significant racial disparities in environmental exposures. Hazardous waste landfills, industrial activities that generate hazardous wastes, such as mining and chemical plants, and heavily trafficked roadways with high levels of vehicular emissions tend to be situated in areas where lower-income communities and people of colour live, work, and play. The well-documented relationship between race, poverty, and increased environmental risk was the springboard for the environmental justice movement. In light of emerging data indicating that exposure to environmental pollution critically influences COVID-19 health-related outcomes, environmental justice is likely a key factor in explaining the racial inequities of the COVID-19 pandemic.

Further exacerbating the interaction between environmental justice and COVID-19 is climate change. Climate change is increasing environmental pollution in numerous ways, for example, via the release of persistent organic pollutants sequestered in now melting polar ice caps, generation of air pollutants from more frequent and larger wildfires, acidification of the oceans, which is increasing the bioavailability of toxic metals, and the expansion of the range of insects, which is driving increased use of pesticides. These impacts of climate change are disproportionately affecting disadvantaged communities. Further, climate change is widening the economic and social security gaps between disadvantaged and higher socioeconomic groups. Both factors contribute to the inequities in COVID-19 health-related outcomes. In an ironic twist, climate change is likely also a significant factor in the transmission of the SARS-CoV-2 virus from wildlife to humans. Thus, addressing climate change is essential for not only mitigating severe COVID-related illness and other environmental justice issues but also for limiting future viral pandemics.

Below, we describe the science supporting the critical influence of environmental pollution on COVID-19 health-related outcomes and the response to vaccines against viruses. We also discuss the perfect storm set up by the confluence of climate change, environmental justice, and COVID-19 ***(Figure 1)***.

Direct impacts of the environment on COVID-19 risk and severity

Air pollution is a widespread source of human exposure to environmental chemicals. Air pollutants such as wildfire smoke, cigarette smoke, dust, vehicle exhaust, near roadway traffic emissions and cooking fumes are complex mixtures that include both vapours and particles. Chemical pollutants exist in both

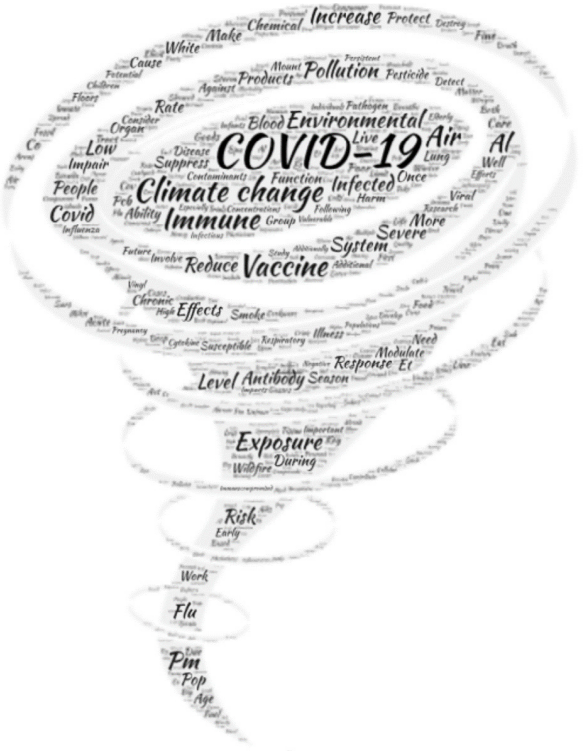

Figure 1: *Climate change, environmental justice, and COVID-19: The perfect storm.*

the vapour (gas) and the particulate fraction, the latter referred to as particulate matter (PM). PM includes chemicals adsorbed to the surface of particles as well as chemicals embedded within the particles. Air pollution is a likely confounding factor for COVID-19 health-related outcomes because both air pollutants and SARS-CoV-2 enter the body via the respiratory system. While SARS-CoV-2 was first described as a respiratory virus that results in difficulty breathing, our understanding of SARS-CoV-2 has advanced since the pandemic began, and we now know that individuals with symptomatic COVID-19 can exhibit neurologic, immune and cardiovascular symptoms in addition to respiratory symptoms. These symptoms overlap known health impacts of chronic exposure to air pollutants. Epidemiologic studies indicating a strong association between exposure to indoor or outdoor air

Environmental Pollutant	*Mechanism(s) Mediating Effect on COVID-19 Related Health Outcomes*
Particulate Matter 2.5 µm (PM2.5)	- Increases ACE2 receptor expression - Inhibits mucociliary clearance - Increases epithelial permeability, promoting viral spread - Alters immune response, specifically decreasing macrophage functions - Delays or complicates recovery of patients of COVID-19
Wildfires	- Increase susceptibility to infections
Tobacco Smoke	- Alters the innate and adaptive immune response
E-Cigarettes	- Alters nasal mucosal immune responses, essential for host defense
Oxidant Gases	- Increases ACE2 receptor expression - Damage alveolar cells - Respiratory distress syndrome - Decrease alveolar macrophage inactivity
Indoor Air Pollution	- Increase susceptibility to infections - Alters olfactory functions

Table 1: *Inhaled environmental pollutants that directly influence COVID-19 risk and severity*

pollutants and increased risk of COVID-19 morbidity and mortality further support the hypothesis that co-exposure to air pollution exacerbates the adverse health effects of SARS-CoV-2 infection ***(Table 1)***.

Emerging data suggests that virus particles can attach to PM, which may directly increase COVID-19 risk and severity by increasing dispersal of the virus in the atmosphere. Humans emit airborne droplets when coughing, sneezing, speaking or singing, and even just breathing. Risk of viral transmission increases in enclosed indoor spaces because the concentration of airborne droplets from infected individuals, which contain live virus, is significantly higher. There are concerns that these droplets may merge with PM thereby increasing viral infectivity and/or deposition, or perhaps increase the persistence of the virus in the environment or the distance the virus is transported. However, these possibilities have yet to be proven.

Similar to other viral respiratory diseases, such as influenza, air pollution has greater effects on COVID-19 health-related outcomes when exposures are chronic and occur before infection. Specific mechanisms by which chronic exposure to air pollution increases COVID-19 infection, the severity of symptoms and mortality are beginning to emerge. Chronic air pollution exposure inhibits the physiological mechanisms that would normally remove viral particles from the lung, resulting in a higher viral load, which can exacerbate

illness following viral infection. This can occur through changes in immune responses and direct damage to airway epithelium (the cells that line the lumen of the respiratory tract) from both the virus and the air pollution. Detailed studies of the expression of the ACE2 receptor that binds SARS-CoV-2 and facilitates entry of the virus into cells, indicates increased ACE2 receptor expression in both the lung and heart in individuals chronically exposed to air pollution compared to control subjects living in cleaner air. Within the respiratory tract, ACE2 receptor expression is highest in the nasal epithelium, which is a likely initial target for impact and infection by droplets carrying SARS-CoV-2. In the nasal epithelium, ACE2 is expressed at high levels on non-neural cell types that support olfactory neurons (neurons that mediate the sense of smell). The decreased viability of the supporting cells because of viral infection may explain the loss of sense of smell associated with COVID-19. The ACE2 receptor is also found on ciliated cells in the airways and on alveolar type 2 cells in the gas exchange region of the lung, infection of which is linked to impaired mucociliary clearance in airways and reduced oxygen exchange in alveoli, respectively. Loss of alveolar epithelial cells due to viral damage promotes conditions associated with

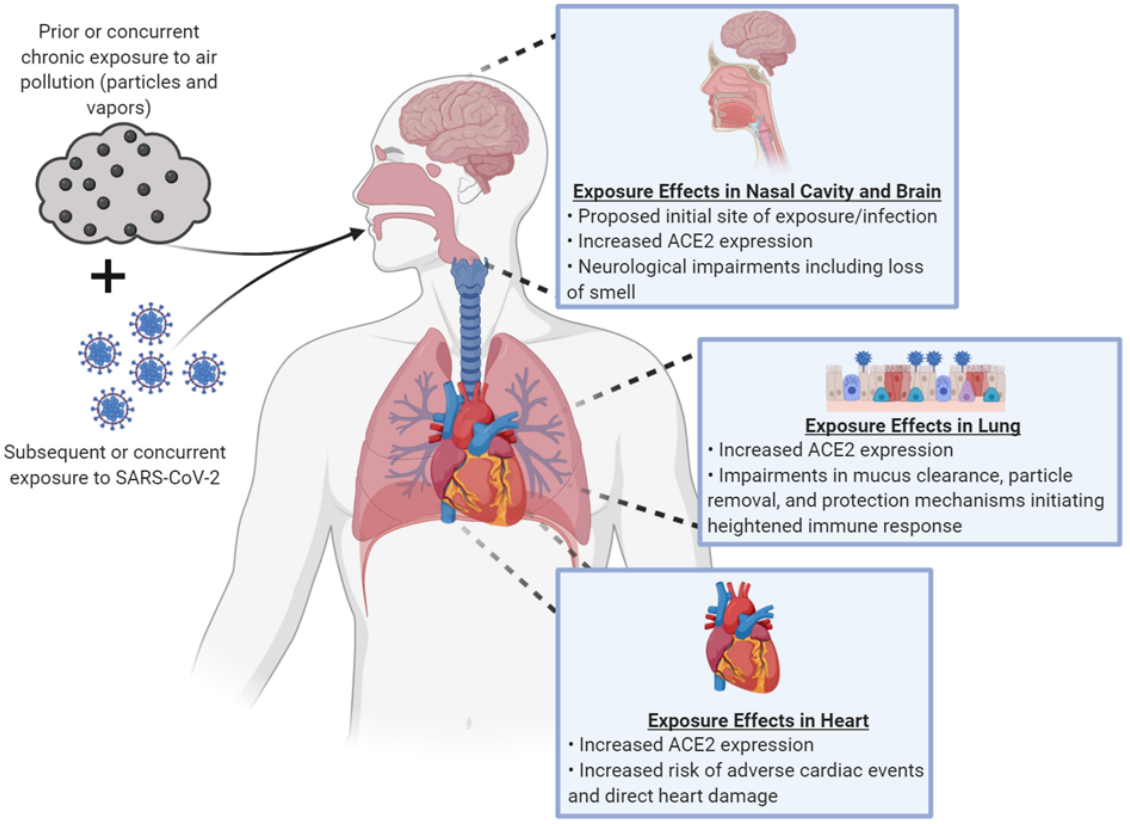

Figure 2: *Mechanisms by which air pollutants directly enhance risk and severity of adverse outcomes of SARS-CoV-2 infection. Figure created with BioRender.com.*

pneumonia and respiratory distress caused by increased fluid accumulation in the lung. The heart and blood vessels also express ACE2 receptors, which may partly explain the abnormal blood clotting and heart problems observed in COVID-19 patients. PM exposure is known to adversely affect these systems, raising concern about a “two-hit effect” in which chronic PM exposure predisposes to more severe infections and worse outcomes in patients with SARS-CoV-2 ***(Figure 2)***. Still unknown is whether the impacts of PM in the vasculature and heart are due to the very small inhaled air pollution particles migrating out of the lung and into the blood or to immune responses triggered by particle exposure in the lung or by the release of soluble chemicals from the particles. Regardless, epidemiologic studies are clear that chronic exposure to air pollution exacerbates SARS-CoV-2 and worsen outcomes in the lung and possibly throughout the body.

Environmental Pollutant	***Source***	***Route of Exposure***	***Targeted Biological System***		
			Lung	Cardiovascular	Metabolic
Arsenic	Contaminated groundwater and food, cigarette smoke	Ingestion	x	x	x
Biomass burning	Fuel for household cooking	Inhalation	x	x	x
Bisphenol A	Soft plastics, epoxy resin lining, and thermal paper	Ingestion, Dermal	x	x	x
Dust Mites	Bedding, mattresses, upholstered furniture, carpets and curtains	Inhalation		x	
Lead	Contaminated soils, leaded paints, leaded gas	Ingestion, Inhalation		x	x
Organotins	Anti-fouling paint for marine vessels; agricultural fungicide and miticide	Ingestion	x	x	x
Ozone	Combustion products reacting with sunlight; smog	Inhalation	x	x	x
Particle Matter 2.5 µm (PM2.5)	Combustion of fossil fuels, dust, wildfire smoke	Inhalation	x	x	x
Perfluoroctanoic acid (PFOA)	Non-stick coating on cookware, waterproof clothing	Ingestion, Dermal	x	x	x
Phthalates	Plasticizer and cosmetic ingredient	Ingestion, Dermal, Inhalation	x	x	x
Tobacco smoke	Cigarettes and other tobacco products	Inhalation	x	x	x

Table 2: *Environmental pollutants that indirectly influence COVID-19 risk and severity*

Environmental exposures indirectly influence COVID-19 risk and severity

While there is still much to learn about COVID-19 disease development, it has become abundantly clear that the progression and outcome of the disease vary widely amongst individuals infected with SARS-CoV-2. In addition to direct impacts of environmental exposures on disease progression, epidemiological studies of hospitalised populations around the world have revealed an emerging pattern: individuals with preexisting conditions are statistically more likely to be hospitalised, enter the intensive care unit, and die from COVID-19. Of these preexisting conditions, metabolic, lung, and cardiovascular disease are over-represented in individuals hospitalised for COVID-19. Of note, exposures to environmental pollutants are linked to increased incidence of all these diseases associated with worse health outcomes following infection with SARS-CoV-2. Thus, environmental chemical exposures may indirectly influence the severity of COVID-19-related disease by increasing the individual risk for these pre-existing health conditions ***(Table 2)***.

While the mechanisms of how these chronic diseases influence SARS-CoV-2 infection are not fully understood, some general commonalities include an increase in expression of the ACE2 receptor, abnormal baseline inflammation, and physiologic vascular changes that synergize with COVID-19 disease manifestations ***(Figure 3)***. The impaired immune function associated with these comorbidities interferes with an effective antiviral response. Individuals with metabolic syndrome, chronic lung disease, and cardiovascular disease also have higher

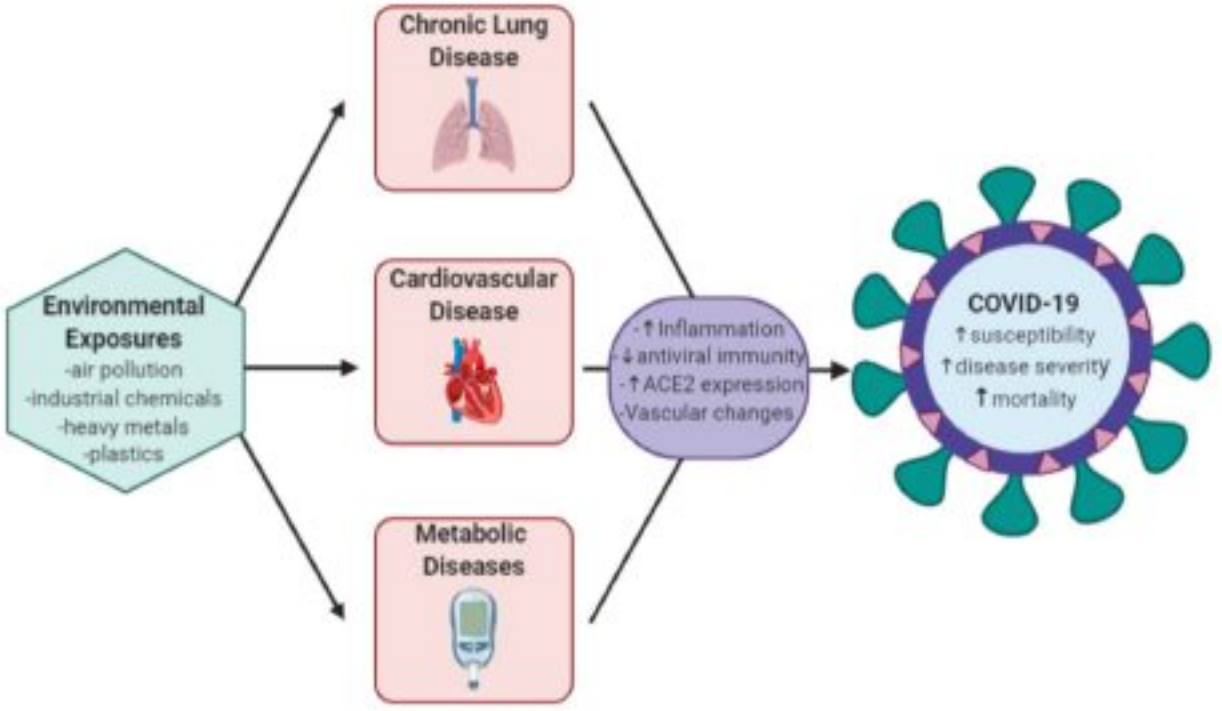

Figure 3: *Environmental contaminants increase risk for co-morbidities associated with increased COVID-19 risk and/or severity of COVID 19-related health outcomes.*

baseline levels of inflammation. Since a productive antiviral immune response involves the tight coordination of several arms of the immune system, the inability to mount a protective immune response in combination with a non-useful inflammatory response amounts to a worst-case scenario for COVID-19 immunity. The increased inflammation can also damage blood vessels and cause cardiovascular complications as observed in many severe cases of COVID-19. Further, in the absence of the ability to destroy the virus, the excessive inflammatory response can result in a cytokine storm (an uncontrolled release of chemical mediators from immune cells that destroy healthy tissue), a well-documented precursor of COVID-19 mortality.

Metabolic syndrome is a category of disease that includes obesity and type 2 diabetes. Among people with COVID-19, those who have metabolic syndrome are more likely to require hospitalisation and to develop a severe course of viral infection. Obesogens, defined as chemicals that interfere with the regulation of metabolism and body weight, can contribute to the progression of metabolic syndrome by increasing the number of adipocytes (fat-storing cells), shifting energy balance to favour caloric storage, and altering the hormonal regulation of appetite and satiety. Obesogens can be found in various consumer products (e.g. food containers, cookware, and cosmetics), and thus, there is widespread human exposure to these environmental contaminants. For example, bisphenol-A (BPA), a synthetic compound used in food packaging, is consistently detected in tissue samples from more than 90% of subjects in numerous studies. Human exposure to BPA is positively associated with obesity and insulin resistance across all age ranges. Therefore, in addition to genetic predisposition and excess caloric intake, environmental obesogens should be acknowledged as crucial drivers to metabolic syndrome, which can substantially impact the response of individuals to infectious diseases, such as COVID-19.

Like diabetes, chronic lung diseases, such as asthma and chronic obstructive pulmonary disease (COPD), influence recovery from, and survival after, COVID-19. Asthma is a chronic condition that impairs lung function. Diverse environmental factors such as pollen, tobacco smoke, dust, diesel fumes, pesticides, and dust mites can trigger and exacerbate asthma. COPD, an inflammatory lung disease that affects about 250 million people worldwide, obstructs the airways. Tobacco smoke is the primary cause of COPD; however, there is evidence that urban air pollution can also contribute to the risk and severity of COPD. The resulting inflammation of the respiratory system caused by exposure to these environmental factors can contribute to increased severity of symptoms and slower recovery from COVID-19.

Cardiovascular disease is the number one cause of death worldwide and unfortunately synergizes with the cardiac manifestations of COVID-19, which include arrhythmias, myocardial injury, blood clots and stroke. Cardiovascular disease arises from an interaction between multiple types of risk factors, including genetic susceptibility, lifestyle habits, and notably, exposure to environmental contaminants, including industrial emissions and waste (e.g., carbon dioxide, metals), emissions from the burning of fossil fuels for heating, electricity, and transportation, and chemicals found in consumer products (e.g., plastics, nonstick coatings). These environmental exposures alter cardiovascular function via three common mechanisms: inflammation (as described above), induction of oxidative stress, and endothelial cell dysfunction. Oxidative stress results when the production of reactive oxygen

and nitrogen species overwhelms the antioxidant capacity of the cells, resulting in damage to cellular macromolecules, including lipids, proteins, and DNA. Endothelial cell dysfunction is often a precursor to hypertension and atherosclerosis. In blood vessels with endothelial cell dysfunction, oxidative stress may also directly alter the ACE2 receptor such that SARS-CoV-2 spike protein can more readily bind to the receptor and gain entry to cells.

Disadvantaged communities have higher burdens of cardiovascular, respiratory, and metabolic disease, coinciding with disproportionately higher exposures to environmental pollution. According to the World Health Organization, socioeconomic factors including ethnicity, education level, employment status, and income level influence susceptibility to chronic diseases in individuals living across low- middle-, and high-income countries. Using education as an indicator of socioeconomic status, a study of adults across 20 European countries found a social-health gradient, with cardiovascular disease, hypertension, respiratory problems, diabetes, and obesity negatively correlated with education level. In the United States, these health disparities are well documented with

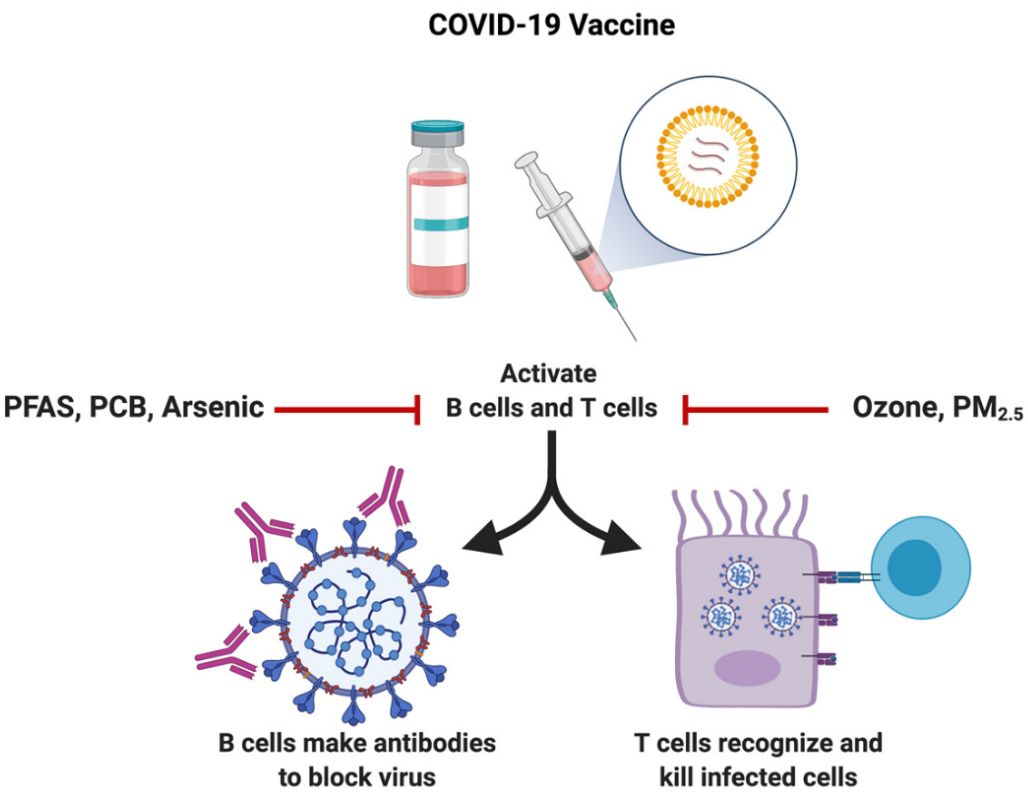

Figure 4: *COVID-19 vaccines activate B cells to produce antibodies that prevent the SARS-CoV-2 virus from binding to and infecting cells. COVID-19 vaccines also activate a specialized T cell that can recognize and kill cells infected with virus. The process of activating B and T cells can be altered by environmental toxicants that are influenced by climate change, including PFAS, PCBs, arsenic, ozone and PM2.5. Created with BioRender.com.*

Hispanic and non-Hispanic Black adults having a higher prevalence of obesity, diabetes, asthma, and heart disease than non-Hispanic white adults. When disease outcomes are considered, the picture becomes even grimmer, with mortality associated with these diseases significantly higher in non-white individuals. These data underscore the need for a proactive focus on health disparities and investment in healthy, sustainable environments if we are to resolve the COVID-19 crisis.

Potential for environmental exposures to influence vaccine response

Vaccines against SARS-CoV-2 are a crucial public health tool for preventing COVID-19. Vaccines help prevent severe health outcomes from viral and bacterial infections by training the immune system to develop a rapid response to protect against these pathogens. The process of vaccination closely mimics a natural encounter with a pathogen by first initiating an immediate (innate) immune response, followed by a more powerful but delayed (adaptive) immune response. The immediate response has limited efficacy due to its broad mechanism, whereas the delayed response is specific to each pathogen. Central to the protective role of vaccines is the generation of potent antibodies and “memory” immune cells that can be rapidly deployed and amplified if the target pathogen is

encountered again. Vaccines activate B cells to produce antibodies that circulate in the bloodstream; these antibodies can neutralise a pathogen by preventing it from infecting cells in the body. In addition to B cells, vaccines can also activate specialised cytotoxic T cells that target and kill other cells infected by a pathogen. The combination of blood antibodies (humoral immunity) and cytotoxic T cells (cellular immunity) is essential for a robust protective response against a pathogen ***(Figure 4)***.

Many different vaccination strategies have been developed that incorporate either a weakened version or a synthetic segment of the pathogen, to effectively mimic the immediate and delayed immune responses necessary to eliminate the pathogen. In the case of SARS-CoV-2 virus, vaccine candidates under development have primarily targeted the spike protein thought to be important for viral particles to adhere to cells of the respiratory tract. Typically, vaccine development can take months to years; however, the timeline for a SARS-CoV-2 vaccine has been accelerated due to the urgency of the COVID-19 pandemic. As of November 2020, there are over 50 COVID-19 vaccines with a range of immunization platforms in the clinical trial. Leading vaccine candidates from Pfizer and Moderna utilise synthetic nucleic acids as the mechanism for triggering a protective immune response against SARS-CoV-2. COVID-19 vaccines have yet to be broadly tested; however, Phase III trials with an enrollment of thousands of volunteers support the efficacy of over 90% for both Pfizer and Moderna vaccines.

While there are many pathways by which environmental pollution can influence immunity and susceptibility to SARS-CoV-2 infection, there are limited studies on the impact of environmental exposures on vaccine efficacy. Vaccines that minimise the severity of virus infection elicit targeted immune responses that generate antibodies and cellular memory. Both arms of the immune system are required to provide a comprehensive strategy to limit replication of the virus in host cells while simultaneously eliminating cells infected by the virus that avoided detection by antibodies. Given what is currently known about how the immune system can be influenced by environmental pollutants, the efficacy of the COVID-19 vaccine may be limited. Environmental pollutants often elicit inflammation, but under conditions of chronic exposure, environmental pollutants can suppress the ability of the immune system to generate a powerful antibody and/or cellular immune response by inhibiting the production of memory B and T cells.

Studies conducted in animal and cell culture models have shown that experimental exposure to air pollutants such as ozone or PM2.5 can suppress the inflammatory response to pathogens. There are also a growing number of studies supporting a link between human exposures to air pollutants and increased infection with influenza virus. Recent wildfire events, such as those in the United States and Australia, have generated great interest in understanding the long-term health effects in nearby populations exposed to high levels of smoke from burning biomass for extended periods. Seasonal wildfires in Montana have been linked to increased rates of influenza in nearby regions, suggesting exposure is associated with suppressed immunity against viruses. Experimental exposure of human volunteers to low concentrations of wood smoke has also been reported to reduce the T cell response to an

intranasal influenza vaccine, raising concerns that environmental air pollutants may have similar effects in the general population.

Environmental levels of man-made pollutants, such as perfluoroalkyl substances (PFAS), are inversely associated with concentrations of circulating antibodies for childhood and adolescent vaccines, including Haemophilus influenza type b, tetanus, and diphtheria. In addition, exposures to other environmental pollutants, such as arsenic and polychlorinated biphenyls (PCBs), are linked to reduced antibody titers against vaccines for measles and mumps. While studies supporting an association of environmental pollutants with reduced vaccine efficacy have been primarily focused on early childhood, there is evidence environmental exposures may also immunocompromise adults. For example, clinical studies have reported increased incidence of varicella-zoster virus activation from adult patients recovering from arsenic poisoning: urinary arsenic levels were found to be inversely linked to reduced antibodies against varicella in these subjects. Collectively, these findings support the notion that exposure to environmental pollutants whose levels are increasing because of climate change can reduce the efficacy of vaccinations to prevent severe disease resulting from infection by pathogens, including SARS-CoV-2.

These observations suggest that vaccines for SARS-CoV-2 may have reduced efficacy in populations with higher exposures to environmental pollution, which, unfortunately, are the populations at increased risk for more severe COVID-19 health-related outcomes. Breaking this vicious cycle will require focused efforts on reducing pollution burdens worldwide.

Climate change as a driver of COVID-19 risk

Climate change is one of the greatest threats to public health in the 21st century, worsening pollution of the air, water and soil, and jeopardizing water and food security. As discussed earlier, these impacts of climate change contribute to the increased vulnerability of disadvantaged communities to COVID-19. Exposure to environmental pollutants is strongly associated with increased risk and severity of COVID-19, as well as to pre-existing health conditions that predispose to worse health outcomes in individuals infected with SARS-CoV-2. It is well documented that environmental exposures are, on average, significantly higher in disadvantaged communities.

In addition, climate change, and the associated extreme weather events such as heatwaves, drought, flooding, and wildfires, increases the likelihood of transmission of emerging infectious diseases (EID), such as COVID-19, from wildlife to humans. Approximately 70% of EID are zoonotic in nature and one of the main drivers of increased zoonotic disease transmission is a loss of biodiversity. Climate change has resulted in an extreme loss of biodiversity over the past few decades: since the 1970s, it is estimated that populations of birds, mammals, amphibians, reptiles, and fish have declined by 30%. This increases zoonotic disease transmission because of changing predator-prey balances, reduced competition, and die-off of species that are suboptimal disease hosts resulting in an increased density of reservoir species.

Simultaneously, habitat loss due to climate change, changing land usage, and spread of

human populations into more rural and remote areas, increases the likelihood of human-wildlife interactions, resulting in greater exposure of people to emerging pathogens. This increased exposure of humans to novel zoonotic pathogens creates the opportunity for pathogens to move from wildlife populations to humans in a process called a “spillover” event. The number of these spillover events and zoonotic disease outbreaks are increasing, and they are responsible for the majority of major human disease outbreaks worldwide including the current SARS-CoV-2 pandemic and previous outbreaks of SARS-CoV-1, MERS, Nipah virus, HIV/AIDS, and Ebola virus.

Monitoring of emerging infectious disease “hotspots” where changes in land-use patterns and biodiversity make human-wildlife interactions more common, as well as surveillance of endemic wildlife pathogens, is now a critical step in preventing future viral disease outbreaks.

Environmental pollution in the eye of the storm

The year 2020 has brought into sharp focus the inextricable connections between climate change, environmental justice and COVID-19. At the centre of this confluence is environmental pollution. More and more scientists are making the argument that this “perfect storm” demands early aggressive action to *mitigate dramatic human loss*. As the eminent scientist Jane Goodall remarked in a discussion on conservation, climate change and COVID-19, “If we carry on with business as usual, we’re going to destroy ourselves”.

So what can be done? Evidence that people in polluted areas are far more likely to die from COVID-19 than those living in cleaner areas provides compelling rationale to not only enforce, but also strengthen, environmental pollution regulations, and for humans globally to embrace lifestyle and policy changes that mitigate climate change to the greatest extent possible. To achieve this vision, it will be essential to develop an inclusive framework that advocates for all people and the planet, and that highlights how injustices happening to marginalised communities are interconnected with the degradation and poisoning of places where they live, work, and play. We cannot ignore social inequality and expect to see progress in environmental health.

As we struggle to overcome the complex issues associated with environmental justice, climate change, and COVID-19, we must keep in mind that the solution to the next viral pandemic threatening the planet may be locked in the brain of a child living in a disadvantaged community experiencing higher levels of environmental pollution that limits not only their potential but also society’s benefit from their contribution.

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NIEHS T32 Trainees/PhD graduate students

Veneese J. Brown
Cindy Khuu
Lowei Lin
Nicole A. McNabb
Claire O'Brien
Osman Sharifi
Nathanial C. Stevens

Training Faculty

Allison K. Ehrlich, PhD
Assistant Professor, Department of Environmental Toxicology
UC Davis College of Agriculture and Environmental Sciences

Pamela J. Lein, PhD
Professor and Chair, Department of Molecular Biosciences
UC Davis School of Veterinary Medicine
Director and PI, UC Davis CounterACT Center of Excellence

Lisa A. Miller, PhD
Professor/Department of Anatomy, Physiology & Cell Biology
UC Davis School of Veterinary Medicine
Respiratory Disease Unit Leader and Inhalation Core Lead
California National Primate Research Center

Laura S. Van Winkle, PhD
Professor/Department of Anatomy, Physiology & Cell Biology
UC Davis School of Veterinary Medicine
Chair, Pharmacology and Toxicology Graduate Group
Director and PI, NIEHS T32 ES007059