Statement of Dr. Jonathan Moses C. Jadloc, FPCP, FACP

I was invited by Ms. Desiree Llanos Dee, a petitioner in the human rights and climate change case, to act as a resource person for the petitioners to discuss the impact of the climate crisis in the carnal human body and to expound on the pathophysiology of heat stress, air pollution effects and zika virus in the third public hearing of the case on August 30, 2018 at the Commission on Human Rights in Quezon City, Philippines.

…." A healthy human body cannot thrive in an ailing planet. Its consequential demise is inevitable and not linear."

- dr jay c. jadloc
environment and health advocate, climate reality leader

"The Consequential Demise of the Human Body in the Climate Crisis"

abstract

As a practicing clinician, I strongly concur with the finding of Intergovernmental Panel on Climate Change (IPCC), which has stated that "human influence on the climate system is clear". The fossil fuels burning, deforestation, agriculture and food production and power plant emissions all release carbon dioxide and other greenhouse gases into the atmosphere, trapping heat causing global warming which changes the climate system subsequently.

Climate Change threatens human health and likely can stall global health gains from research and development. The magnitude of these looming crises led the The Lancet to declare in 2009 that *"Climate Change is the biggest global health threat of the 21st century", and will "put the lives and well being of billions of people at increased risk."*

The World Health Organization estimates that "climate change will cause additional 250,000 deaths per year from 2030-2050" likely the result of malnourishment, malaria, diarrhea, and heat exposure related to climate change effects. All populations will be affected but children, elderly persons and those living in economically poor and developing nations will be disproportionately impacted. In this abstract, I will outline the health effects and demise of human body in the climate crisis particularly on ***heat-related disorders, respiratory disorders and infectious diseases particularly Zika virus infection and consequent microcephaly.***

Heat Related Mortality

Miklós Székely,Luís Carletto,and András Garami Department of Pathophysiology and Gerontology; Medical School; University of Pécs; Pécs, Hungary

Climate Change and Health: A Position Paper of American College of PhysiciansRyan A. Crowley, BSJ

As the climate warms, more people could be susceptible to heat-related illness. Extreme heat exposure caused more than 7400 deaths in the United States. Excessive heat exposure caused more than 7400 deaths in the United States from 19190-2010. In France, 6 heat waves occuring from 1971 to 2003 were associated with significant excess mortality, contributing to 13,700 deaths in 2003 alone, Across Europe, about 70,000 premature deaths were attributed to the 2003 heat wave.

Heat exposure of the body can result in different physiological/pathological outcomes. Physiologically, successful activation of the effectors results in warmth tolerance. In case of repeated, chronic heat exposure the tolerance intensifies leading to heat acclimation .

Heat-related illnesses develop when the pathological effects of heat load are not prevented. Syndromes vary from less severe, such as heat syncope to severe forms as lethal heat stroke.

Activation of autonomic heat-defense effectors affects the regulation of homeostatic systems other than thermoregulation. Increased cutaneous vasodilation and decreased venous tone reduce ventricular filling, which, in an orthostatic position, may lead to low brain perfusion and heat syncope (with unconsciousness). Electrolyte imbalance can develop due to sweating, and promote the occurrence of heat cramps, i.e. short-lived, painful contractions of skeletal muscles during or after prolonged work in the heat.

In severe hypovolemia, heat exhaustion develops with domination of water- or salt-depletion. The former is mainly due to insufficient fluid replacement and consists of thirst, progressive hypovolemia, hyperosmolarity and hyperthermia. Salt-depletion dominates when sweating-induced water loss is replaced, but salt is not (e.g., by soft drinks), leading to hypoosmolarity and its consequences (e.g., cell swelling).

In patients with compensated heart failure, the need for a higher cardiac output during heat exposure may manifest heart failure: high end-diastolic volume/pressure with backward (venous congestion, edema) and forward (insufficient muscle/renal/intestinal perfusion) failure symptoms. Consequently, the patient collapses because of muscle weakness. Although consciousness is maintained, the developing heat decompensation is more severe than heat syncope.

The most severe form of the heat-related illnesses is heat stroke, when body temperature usually exceeds 41°C, neurological dysfunctions, and in some cases anhydrosis are present. Classic heat stroke affects subjects with compromised warmth-defense capabilities (e.g., infants, elderly), while exertional heat stroke occurs in healthy, young adults during high-performance activities in hot environment. In heat stroke, the signs and symptoms of systemic inflammation, distributive/hypovolemic circulatory shock, multiple organ dysfunction, disseminated intravascular coagulopathy, electrolyte, pH, and osmotic imbalance are manifested simultaneously

Air Pollution and Lung Disease

Physicians for Social Responsibility, Coals Assault on Human Health, 2009

When we breathe in dirty air, we bring air pollutants deep into our lungs, so it's no surprise that air pollution causes serious damage to the respiratory tract. Air pollution exposure can trigger new cases of asthma, exacerbate (worsen) a previously-existing respiratory illness, and provoke development or progression of chronic illnesses including lung cancer, chronic obstructive pulmonary disease, and emphysema. Air pollutants also negatively and significantly harm lung development, creating an additional risk factor for developing lung diseases later in life.

Asthma

Over 20 million people in the U.S., including six million children now gasp for breath due to asthma. Asthma, a chronic disease of the lungs characterized by inflammation and narrowing of the airways, causes a sensation of tightness in the chest, shortness of breath, wheezing, and coughing. If untreated, asthma episodes can be near- fatal or even fatal. Asthma is not currently curable, and damage that is done to lung tissue during asthma attacks may lead to permanent damage. Nearly 1.8 million emergency room visits were attributed to asthma in 2005. There are many triggers to asthma attacks, including dust, smoke, pollen, and volatile organic compounds. Common outdoor pollutant triggers include ozone, carbon monoxide, sulfur dioxide and nitrogen oxides.

The Asthma-Ozone Connection

Ozone, one of the most widespread air pollutants in the US, is formed when volatile organic compounds react with nitrogen oxides in the presence of sunlight. Ozone irritates the lungs at concentrations which are fairly common in urban settings, particularly in summer months. Increases in ozone are linked to asthma and other lung diseases. For those with severe asthma, symptoms increase even when ambient

ozone levels fall under the thresholds set by the EPA. Elevated ozone levels also aggravate pre-existing heart problems, like angina.

Chronic Obstructive Pulmonary Disease (COPD), chronic bronchitis and emphysema

Chronic Obstructive Pulmonary Disease (COPD) is another condition characterized by narrowing of the airways, but these changes are permanent rather than reversible. COPD is caused by exposure to pollutants that produce inflammation, an immunological response. In larger airways, the inflammatory response is referred to as chronic bronchitis. In the tiny air cells at the end of the lung's smallest passageways, it leads to destruction of tissue, or emphysema. Although current and ex-smokers account for most patients with COPD, exposure to air pollutants plays an important role in the development of COPD and the origin and development of acute exacerbations.

Lung Cancer

Lung cancer, the leading U.S. cancer killer in both men and women, is often (and accurately) associated with smoking tobacco. While that's true, there are multiple other risk factors for developing lung cancer, including air pollution. Particulate matter and ozone in particular may affect mortality due to lung cancer.

Children are Especially Vulnerable

Children are particularly susceptible to the effects of air pollution. They breathe through their mouths, bypassing the filtering effects of the nasal passages and allowing pollutants to travel deeper into the lungs. They have a large lung surface area relative to their weight and inhale relatively more air, compared to adults. They also spend more time out of doors, particularly in the afternoons and during the summer months when ozone and other pollutant levels are at their highest. And, children may ignore early symptoms of air pollution effects, such as an asthma exacerbation, leading to attacks of increased severity. Combine those factors with the adverse impact of some pollutants on lung development and the immaturity of children's enzyme and immune systems that detoxify pollutants, and you have a series of factors that contribute to children's increased sensitivity to air pollutants.

Zika Virus and Microcephaly

Aagaard KM, Lahon A, Suter MA, Arya RP, Seferovic MD, Vogt MB, Hu M, Stossi F, Mancini MA, Harris RA, et al. 2017. Primary human placental trophoblasts are permissive for Zika virus (ZIKV) replication. Sci Rep 7: 41389

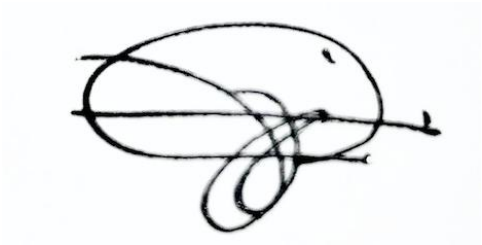
A warmer climate and changing rainfall patterns may also create hospitable environments for mosquitoes, ticks and other climate sensitive vectors that spread such diseases as malaria, chikungunya, dengue fever and the zika virus.

Compared with other flaviviruses, one striking feature of the current zika virus (ZIKV) epidemic is the association of viral infection with a marked increase of risk for congenital microcephaly and serious neurologic complications (Petersen et al. 2016), which triggered widespread efforts to understand the molecular basis of infection. Similar to its close relatives in the Flaviviridae family, such as DENV, yellow fever, Japanese encephalitis, and West Nile viruses (WNV), ZIKV has an icosahedral outer envelope and a dense inner core containing one single-strand positive sense RNA genome between 10,000 and 11,000 base pairs in length (Chambers et al. 1990; Kuno and Chang 2007). The genome of ZIKV encodes a single polyprotein that is post-translationally cleaved by host and viral proteases into three structural proteins (capsid [C], premembrane [prM], and envelope [E]) and seven nonstructural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5)

Microcephaly is a neurodevelopmental disorder, which is characterized by a marked reduction in brain size and intellectual disability caused by impaired cell proliferation and the death of cortical progenitor cells and their neuronal progeny (Barbelanne and Tsang 2014). Through January 26 of 2016, 10,441 suspected and 2366 confirmed ZIKV-associated microcephaly cases have been reported in Brazil (Zika-Epidemiological Report by the Pan-American Health Organization [PAHO] and World Health Organization [WHO]), the country that has experienced the highest ZIKV infection rates worldwide. The increase of microcephaly cases and the coincidental ZIKV outbreak led the WHO to declare a Public Health Emergency of International Concern in early 2016 (Heymann et al. 2016). This ignited tremendous interest and immediate efforts by scientists to understand the impact of ZIKV on human brain development and the mechanistic link between ZIKV infection and microcephaly. In the past year, rapid and stunning progress has been made toward developing stem cell-based cellular models, primary human tissues, and animal models to understand its pathogenesis, investigate underlying mechanisms, and develop therapeutics (Abbink et al. 2016; Cugola et al. 2016; Dudley et al. 2016; Garcez et al. 2016; Lazear et al. 2016; Li et al. 2016a,b; Miner et al. 2016a; Ming et al. 2016; Qian et al. 2016, 2017; Rossi et al. 2016; Tang et al. 2016; Wu et al. 2016; Xu et al. 2016; Hirsch et al. 2017).

Findings from these model systems have helped scientists from the US Centers for Disease Control and Prevention (CDC) to conclude that ZIKV causes microcephaly (Rasmussen et al. 2016).

Nothing further.



Dr. Jonathan Moses C. Jadloc, FPCP
Signed
August 8, 2018