Expert Views

The relevance of climate change for life and health insurance

Part 2 – The Medical Director's View

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The Art & Science of Risk

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In this second part of our series of publications, Dr. Sonal Bagul, SCOR's Head of Underwriting & Claims Asia, explains the physiological mechanisms and links between climate change impacts and human health (some well-understood by researchers and some currently less so) in detail. He will discuss the impacts on various parts of the body such as the heart, lungs, kidneys, and blood, but also on drug use, delivery, and efficacy. In the forthcoming third part of the series, with Dr. Gabriela Buffet, infectious disease specialist, and Xiao Gao, epidemiologist, will look into existing and emerging infectious diseases and their interplay with climate change and health.

Climate change as a public health emergency

Climate change is recognised by scientific bodies as a major public health emergency. The key phenomena that result from climate change include extreme events such as record heat waves, wildfires, floods, hurricanes, and droughts, as well as long-term trends such as poor air quality. Many of these aspects have direct or indirect effect on mortality and morbidity in humans and other animals. More direct consequences of climate change may include injuries, heat-related illness and sometimes death, exacerbation of respiratory and cardiovascular disease, infectious diseases, and changes in vector-related diseases. Indirect aspects may include physical and mental health effects as well as issues related to forced migration driven by climate change.

It is now well established through many studies there is a roughly U-shaped relationship between temperature and all-cause mortality. In the moderate climate of the Netherlands, the lowest mortality has been found to occur at a daily average temperature of 16.5°C,[1] while the mortality is lowest between 22.7 and 25.7°C in Athens.[2] In Taiwan, with a relatively hot summer season, a rightward shift of the optimal mortality temperature range can be observed compared to that of countries with colder climates, at 26-29°C.[3]

These variations across different latitudes can be explained by adaptation and acclimatization. Acclimatization is one of the core components of the human coping mechanism for withstanding variations in environmental heat exposure, in which heat-shock proteins and their cognates play a critical role. Beyond the capacity of these natural bodily defense systems, unless there are radical changes in public health policies regarding heat health risks to support effective planning and adaptation, humanity could indeed be exposed to excessive morbidity and mortality caused by climate change, eventually impacting actuarial assumptions for the life and health insurance industry.

Ideally, such excess deaths should be identified in death registries in a timely manner to support observations useful for public health policy makers or actuarial references. As a medical guideline, in US, a death certificate typically provides space to designate an immediate cause of death (e.g., cardiac arrest), along with up to twenty contributing causes, one of which will be identified as the underlying cause of death.[4] Hyperthermia or hypothermia are included as contributing causes and must be recorded during extreme weather events. Thus, where similar protocols of death certification are followed, it is possible to identify deaths due to extreme weather events. On the other hand, it is expected that deaths due to other outcomes of climate change, like diarrheal deaths, malaria, or dengue, will not be attributed towards climate change directly. Only through epidemiological surveys or wider studies, excess deaths compared to previous periods can be attributed to climate change.

Pathways of climate change's impact on mortality and morbidity

The WHO names several major pathways through which climate change harms human health directly or indirectly. Direct impacts include those from increased exposure to high ambient temperatures and effects mediated through natural systems, such as vector borne diseases. Indirect impacts comprise effects mediated through socioeconomic systems, such as the health consequences of increased impoverishment and mental health.

Often, it is not the climate itself that affects human health; rather, the health consequences result from the environmental, ecological, and social impacts of a changing climate.

Many climate-health models have been developed for a range of health outcomes known to be sensitive to climate change, such as heat-related mortality and morbidity, mortality and morbidity associated with diarrheal disease in children, malaria and dengue risk and mortality/morbidity, undernutrition, and mortality/morbidity associated with coastal flooding. However, medical literature is still limited due to the complexity and novelty of climate change issues. We must appreciate that climate change is a complex phenomenon that has the power to influence patterns of mortality and morbidity in unexpected, unpredictable, and unfamiliar ways.

Physiology of thermoregulation in humans

During extreme weather conditions, deaths may result not only from heat stroke and related conditions, but also from cardiovascular disease, respiratory disease, and cerebrovascular disease. Heat stroke is the most serious heat-related disorder. It occurs when the body is unable to control its temperature. When body temperature rises rapidly, the sweating mechanism fails, and the body cannot cool down. This condition may cause death or permanent disability.

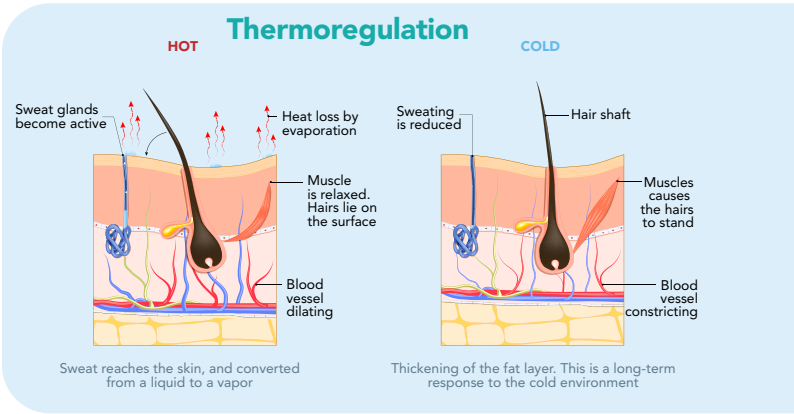
In humans, excess heat is lost from the skin surface. The four primary methods through which heat is lost from skin to surroundings are *radiation, conduction, convection,* and *evaporation*.

Loss of heat by *radiation* means loss in the form of infrared heat rays, a type of electromagnetic wave. All objects that are not at absolute zero temperature radiate such rays. The human body radiates heat rays in all directions. About 60% of total heat loss in human occurs through radiation. If the temperature of the body is greater than the temperature of the surroundings, a greater quantity of heat is radiated away from the body than is received from the surroundings by body, thus allowing for cooling.

Normally, about 3% of heat is lost from the body by direct *conduction* from the surface of the body to solid objects, such as a chair or a bed. However, about 15% of heat can be lost by conduction to air if the air is colder than the skin. Once the temperature of the air adjacent to the skin equals the temperature of the skin, no further loss of heat occurs unless the heated air moves away from the skin, and cooler air could is brought in contact with the skin. Such removal of heat from the body by air currents is commonly called heat loss by *convection*.

Even when a person is not sweating, water still evaporates from the skin, mucosal tissue, and lungs. Such *evaporation* causes continual heat loss that cannot be controlled or scaled upwards or downwards for purposes of temperature regulation, because it results from continuous diffusion of water molecules,[5] whereas loss of heat via evaporation by sweat can be controlled by regulating the rate of sweating.

When the temperature of the surroundings becomes greater than that of the skin, instead of losing heat, the body gains heat by both radiation and conduction. Under these conditions, the only means by which the body can get rid of excess heat is by evaporation. Therefore, anything that prevents adequate evaporation when the surrounding temperature is higher than the skin temperature will cause the internal body temperature to rise. Specifically, if the water content in the surrounding air is so high that it cannot absorb more water vapor, the sweating

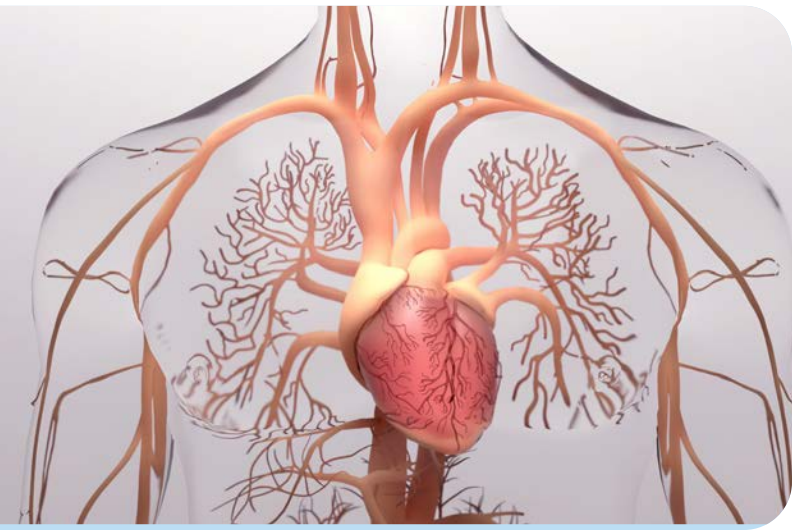


mechanism fails. Such a combination of heat and humidity is also known as wet-bulb temperature. It plays a critical role in weather warnings which we commonly see referred to as "feels like" in weather reports.

Similarly, if the surrounding temperature drops considerably, for example during a cold wave, a somewhat reverse process is initiated by the body through autonomic responses that increases the cutaneous vasomotor tone, leading to vasoconstriction and heat production (thermogenesis). These responses redirect blood from cutaneous vessels into deeper veins, and from the periphery to the core, producing a rise in mean arterial pressure and cardiac output with a consequent reduction in cardiac frequency. Any situation that diminishes the ability to generate such stimulus can lead to thermoregulatory insufficiency and hypothermia.[6]

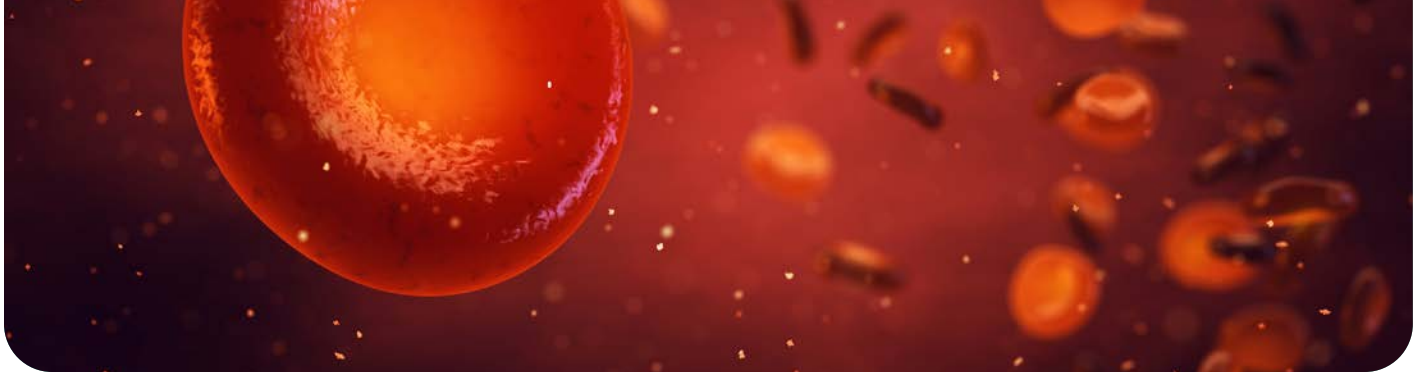
Extreme temperatures and cardiac overload

Climate change is influencing temperature distributions, leading to more extreme and unprecedented heat days. Extreme heat (but also cold) causes increased risk of illness and death by compromising the ability of the human body to regulate its internal temperature, which is primarily mediated by the autonomic system (a component of the peripheral nervous system) and cardiovascular system. The potential pathophysiology mediating the relationship between extreme heat and adverse cardiovascular outcomes has been described by researchers already. In a hot environment, the autonomic nervous system causes cutaneous vasodilation (widening of the blood vessels in the skin) to allow for greater heat transfer from the body to its surroundings. A portion of blood from abdominal organs, and in severe cases all organ systems, is redirected to the skin to accommodate the dissipation of internal heat. This decreased blood flow to central organs can be further reduced if sweating is not offset by adequate fluid intake. Maintaining a steady blood pressure during such significant vasodilation requires an increased cardiac output which is achieved through increases heart rate and myocardial contractility. Usually, the cardiovascular systems of the young and healthy can adapt to such demands due to heat. However, in the elderly and those with pre-existing cardiovascular conditions (e.g., ischemic heart disease, coronary heart disease, or heart failure), the heart is not as proficient at meeting the increased demand required to rid the body of the excess heat. Such individuals are susceptible to adverse health outcomes from extreme heat exposure.[7]



The peer-reviewed literature documents increased emergency department visits in the US during extreme heat waves for three main conditions - hyperthermia, myocardial infarction (heart-attack), and general cardiovascular disease.[8] Through these studies, it was also observed that there is a deterioration of baseline symptoms in hypertrophic cardiomyopathy patients (shortness of breath, chest pain, loss of consciousness) due to a change in ambient temperature.

Conversely, a large population-based cohort study found that short-term exposure to cold temperatures significantly increased the daily rates of hospitalizations for coronary heart diseases, heart attack, and stroke.[9] In Lille, a French city with relatively cool summers, a 10°C decrease in mean temperature was associated with a 13% increase in coronary events, and this association was more pronounced in older patients.[10] Multiple studies



have demonstrated increased cardiorespiratory symptoms such as cough, dyspnea, chest pain and cardiac arrhythmias with cold temperatures, and especially during the winter period.[11] During such excess cold exposure, vulnerable sub-population such as elderly, with already diminished autonomic function, often undergo a cold stress that increases arterial blood pressure and central venous pressure, thus further reducing myocardial oxygen supply.

Effect of Extreme Temperatures on Blood Composition

With the current evidence base, experts believe that increases in blood viscosity may promote an environment that is prone to thrombosis. In combination with increased demand for cardiac output, such blood properties confer a greater risk of adverse cardiovascular outcomes during extreme heat or cold exposure. A hypercoagulable state (where the blood is more prone to clotting) was first proposed as a potential mechanism mediating the association between extreme heat and cardiovascular risk in a study following the 1976 heat wave in UK.[12] Researchers observed that exposure to a very hot and dry environment resulted in increased hemoconcentration and greater plasma cholesterol, blood viscosity, and platelet and neutrophil counts. It has been proposed that activation of the coagulation pathway is attributed to a systemic inflammatory response caused by heat toxicity due to prolonged reductions in central blood flow. However, scientists have not yet finally concluded whether the inflammatory and hemostatic responses observed in the context of heat stroke can be used to explain the association between extreme heat and a greater risk of adverse cardiovascular outcomes.

Similarly, in the 1970s, scientific research focused for the first time on dramatic rises in mortality during winter and found direct a relationship between decreased temperature and coronary/cerebral thrombosis. During exposure to excessive cold temperatures, to reduce heat loss, about a liter of blood shifts to the core from the skin surface. When the central organs of the body are overloaded with blood, the excess volume is disposed of by removal of salt and water from the blood, which in turn leaves most of the other components of the blood more concentrated. Red cells, white cells, platelets, cholesterol, and fibrinogen all increase in concentration by around 10%, and the blood viscosity increases by around 20%. Such changes in blood composition greatly increase the likelihood of a thrombus forming in the arteries, especially in sub-populations with underlying atherosclerotic plaques, ultimately leading to atherothrombotic events.[13]

Respiratory disorders in volatile climate conditions

Driven by a higher concentration of carbon dioxide and other greenhouse gases in the atmosphere, climate change will lead to shifting of precipitation patterns, more frost-free days, and warmer seasonal air temperatures. These changes are predicted to affect the start and end of the pollen season, how long it may last every year, how much pollen plants create, and how much pollen load there is in the air. In addition, non-native plants are encroaching on new territories, sometimes prolonging the pollen season by having a later blooming peak compared to native species. One example is *Ambrosia artemisiifolia*, native to the Americas, where it is already known as allergenic, but now also found in wide parts of Europe, temperate Asia and the Indian subcontinent.

Pollen exposure can trigger various allergic reactions and further exacerbate lung diseases. Exposure to pollen has been linked to asthma attacks and increases in hospital admissions for respiratory illness. Pollen exposure can also trigger symptoms of allergic rhinitis and conjunctivitis. In many patients, allergic response tends to persist throughout their lifetime, and curative treatment is not always successful. Studies have shown that allergic rhinitis and asthma frequently occur together. Patients with asthma and rhinitis share common physiology including heightened bronchial hyperresponsiveness and heightened reactivity to a variety of stimuli. Immunopathology of allergic rhinitis is also similar with the predominance of T-helper type 2 inflammation and

tissue eosinophilia. Clinical evidence suggest that local airway inflammation can result in a systemic inflammatory response as well. It is estimated that medical costs linked with pollen exceed USD 3 billion every year in US alone.[14]

Another factor influencing respiratory conditions is the more frequent occurrence of wildfires. Exposure to particulate matter (PM) air pollution resulting from wildfire smoke is one of the serious climate-related pathways that aggravates existing respiratory disease and where long-term respiratory health impacts may currently be underestimated. PM is a particular concern because when the particulates are inhaled, they can become lodged in the lungs or absorbed into the bloodstream and transported through the body. A growing body of epidemiological evidence documents an association between acute exacerbations of asthma or Chronic Obstructive Pulmonary Disease (COPD) and wildfire smoke exposure.[15] Multiple studies show evidence of increased physician visits, emergency department or hospital admission with those with existing respiratory diseases like asthma, COPD or for those diagnosed with acute bronchitis and pneumonia around the time of wildfire events. Although data lacks with respect to excess death linked to respiratory disorders due to wildfire smoke, there is increasing evidence that it has substantially increased short- and long-term cardiovascular mortality in those affected.[16] A meta-analysis of data from ten southern European cities between 2003 and 2010 found an increase in cardiovascular mortality especially on smoke-affected days with a higher concentration of PM with a size of less than 10 micrometers.[17]

Although a review of the current literature could not firmly establish the association between exposure specifically to wildfire smoke and cardiovascular mortality in the long-term, both short- and long-term exposure to air pollution have shown to be linked to significant increases in mortality from a number of diseases. In 2016, the WHO attributed 24% of stroke, 25% of ischaemic heart disease, 28% of lung cancer and 43% of chronic obstructive pulmonary disease cases to air pollution worldwide. Other studies link air pollution to lower birth weights, asthma, and dementia.

Another risk for respiratory health is linked to extreme weather. Once extreme precipitation events or floodings subside, residual moisture in buildings can result in mold contamination, leading to poor indoor air quality even after any standing water has been drained. Molds are microorganism that are an integral part of our world's flora and fauna. Due to their small spore size, they can aerosolize effectively and easily reach lung alveoli. When inhaled by healthy individuals, the body's natural defense mechanisms (both the innate immunity and physical anatomical barriers like cilia and lung surfactants) usually suppress growth of these microorganisms. However, molds can still trigger a wide spectrum of diseases, ranging from common allergic reactions to infectious illnesses that are less common.

Populations living in damp indoor environments experience an increased prevalence of asthma and other upper respiratory tract symptoms, such as coughing and wheezing, as well as lower respiratory tract infections such as pneumonia, respiratory syncytial virus infection, and hypersensitivity pneumonia aspergilloma; in immunocompromised individuals, invasive aspergillosis can occur. Health researchers believe that with increasing frequency of precipitation events, the incidence of such conditions as new-onset disease may rise or predispose those already living with underlying respiratory conditions.[18]

Link between heat and renal diseases

High external temperatures that the body cannot adapt to can result in increased core temperatures, dehydration, and blood hyperosmolality (high concentration of sodium or other electrolytes). Heatstroke is known to play major role in causing both acute kidney disease and heat-induced inflammatory injury to the kidney. As explained previously in this paper, during a heat stroke, blood from abdominal organs is redirected to the skin to accommodate the dissipation of internal heat. Such decreased blood flow to kidneys may impair the glomerular filtration process, the kidneys' main function. Clinically, it is well established that different mechanisms, such as dehydration or rhabdomyolysis (breakdown of muscle tissue), may contribute to the development of acute kidney injury. The elderly population is the most vulnerable, due to a low tolerance to high temperature, impaired sensation of thirst, already decreased glomerular filtration rate, and reduction in the tubular reabsorption of water and sodium during a state of dehydration. Such heat and dehydration, when they occur in higher frequency, can result in chronic kidney disease (CKD) and ultimately may trigger CKD of epidemic proportions in hot regions of the world, especially in people exposed to extreme heat.

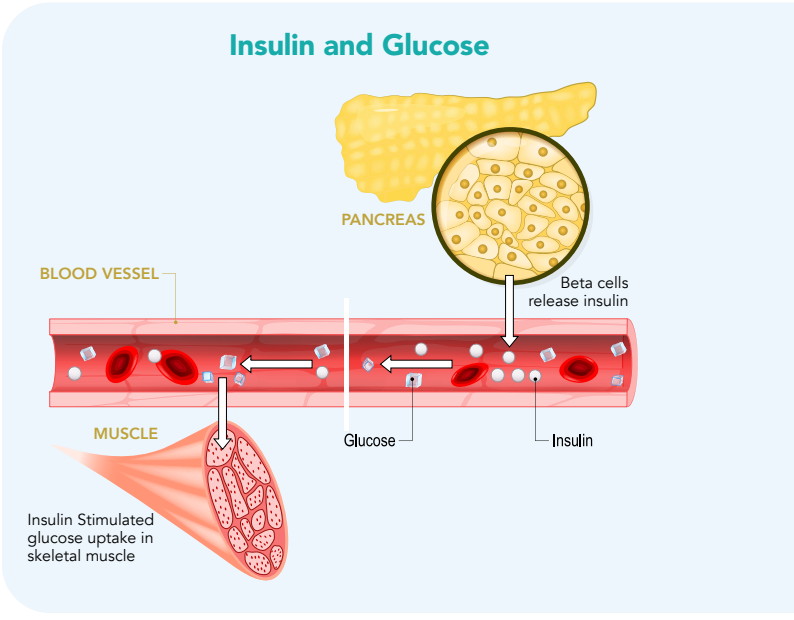
Similarly, heat stress and subsequent dehydration also have a role in kidney stone formation, and poor hydration may further increase the risk for recurrent urinary tract infections.[19] Multiple studies have been published to establish a correlation of elevated temperatures during heat waves with hospital admissions due to kidney disorders.[20]

For the past few decades, an enigmatic evolution of CKD of unknown origin has been observed in pockets of high prevalence along the Pacific Ocean coast of the Mesoamerican region, from southwest Mexico to Costa Rica, known as "Mesoamerican endemic nephropathy." The disease is common in young adult men, most often yet not exclusively from agricultural communities, and has a high mortality rate. Kidney biopsy specimens show primarily tubular atrophy and interstitial fibrosis with some glomerular changes attributed to ischemia. It is hypothesized that the causal pathway could be due to extreme heat exposure due to climatic changes.[21] A similar disease pattern is now believed to be potentially global, with similar occurrences observed in North America, the Middle East, Africa, and India.

Interplay between climate change and diabetes

Diabetes is a public health concern that currently affects over 8% of the global population, with numbers expected to rise. With emerging changes in climatic conditions, it would be fair to assume such changes could substantially impact the diabetic population in many ways. Diabetics are found to be particularly vulnerable to both heat and cold waves as an outcome of impaired thermoregulatory mechanisms alongside impaired autonomous nervous system responses to extreme temperatures. Studies clearly document increased numbers of visits to emergency rooms, hospitalizations, and mortality for people with diabetes during both cold and heat waves.[22]

The pathophysiological pathway behind these increased rates of morbidity and mortality in diabetics is as follows: during heat exposure, heat loss in the body is enhanced by skin blood flow (vasodilation) and sweating that is stimulated



by cholinergic nerve cells. Both these actions are modulated by the sympathetic nervous system and triggered as soon as the mean body temperature surpasses a given threshold. When heat-loss responses reach maximum levels, no further cooling of the body can take place in spite of further rises in mean body temperature. Diabetes can delay or increase this onset threshold and reduce thermosensitivity as well as the maximum capacity for heat-loss responses. That, in turn, makes for greater increases in mean body temperature during exposure to excessive heat.

On the other hand, patients with diabetes are also susceptible to extreme cold temperatures. In fact, it is during winter season that higher rates of morbidity in diabetics have been observed. However, the pathophysiology pathway of cold effects on diabetes mortality is not very clear yet. Lack of physical exercise, lower intake of fresh fruits and vegetables and higher intake of saturated fat, and reduction of Vitamin D level in patients with already impaired insulin sensitivity are possible factors contributing to winter mortality peaks.

Medication use and thermoregulation

Several heat-exposure situations, such as physical exercise and work in hot weather, steam baths, or saunas are known to induce changes in hemodynamics, body fluid volume and blood flow distribution, which in turn may affect the pharmacokinetics of a drug and the therapeutic response.

Many regular medications prescribed to manage chronic conditions may interfere with the body's internal thermostat or impair sweating. Such medications, including but not limited to general anticholinergics, antidepressants, and opioids, might compromise physiological heat loss responses. However, many medications have not been yet systematically studied in the context of thermoregulation, nor have the effects of using regularly prescribed doses during heatwave conditions. Researchers believe that a group of drugs including anticholinergics, antidepressants, antiepileptics, antihypertensives, muscle relaxants, and opioids may alter the thermoregulatory center in brain, while the group of anticholinergics, antihistamines, antipsychotics, antivertigo medications, bladder antispasmodics, gastric antisecretories, and muscle relaxants may alter sweat gland and cutaneous vasculature stimulation peripherally.[23]

Certain drugs prescribed to manage hypertension, like angiotensin converting enzyme inhibitors (ACEIs), ACE inhibitors, beta blockers, and diuretics, may have the potential to exacerbate the effects of extreme heat on the body. Furthermore, studies have observed adverse drug reactions during period of heatwaves from cardiovascular medications such as diuretics, ACEIs and ACE inhibitors. For instance, an analysis of patients that were admitted to hospitals in Paris during the 2003 heatwave demonstrated that long-term use of diuretics could negatively affect the prognosis of patients that suffered from non-exertional heatstroke.[24,25]

Oral anticoagulants like heparin and warfarin are widely used to reduce the risk of blood clots, and this type of medication can also cause bleeding problems, which may result in an elevated risk of hemorrhagic stroke caused by a burst or bleeding blood vessel. A recent study observed that elderly patients who regularly took oral anticoagulants exhibited a significantly increased risk for stroke admissions on hot days compared to non-users.[26]

Effect of extreme temperatures on drug delivery and efficacy

In addition to interference with the body's thermoregulatory responses, drugs are at risk of encountering issues such as stability of preparations, equipment malfunctions, or adverse impact on pharmacokinetics due to extreme temperatures.

As an example, it is known that prolonged exposure to high temperatures can alter insulin kinetics and stability. Insulin can be stored up to four weeks at temperatures not higher than 25-30°C. Under high temperatures and exposure to light, insulin may transform by deamination and formation of dimers and oligomers, thereby reducing its biological activity.[27] A case of insulin degrading in the reservoir of a pump in sunlight and heat was published in British Medical Journal in 2009 where a young girl suffered an episode of diabetic ketoacidosis despite the correct dose having been administered by her insulin pump.

Normally, the package leaflet for approved medicinal products must contain information regarding the storage conditions under which the drug should be stored. When a temperature limit is specified on such leaflet, the stability of the drug must have been demonstrated through long-term testing up to the specified value. For drugs where no particular storage conditions are noted, usually the drug would have been tested at a storage temperature of 40° C with 75% humidity. However, when ambient temperature surpasses these limits, the risk of drug degradation is elevated.

Heat can affect pharmacokinetics, and that in turn can affect a patient's exposure to the active substance. For example, increased cutaneous blood flow due to surrounding heat may increase the systemic availability of transdermally or subcutaneously administered drug substances like insulin, nitroglycerin, or opioid patches. Similarly, due to circulatory changes during increased ambient temperature, absorption and elimination of certain drugs may also be impacted due to compromised renal or hepatic filtration.[28] Such occurrence of increased concentration of drugs in systemic circulation would lead to potential side-effects or adversely affect desired therapeutic response.

Care providers, medical personnel and patients might be unaware of the impact of changing climatic conditions on drug delivery and efficacy, leading to an unnoticed decline in the quality of medication properties. Hence patients may be required to be advised for monitoring the timing and dose of drugs to be taken, and stakeholders need to put more emphasis on storage and transportation conditions.

Vulnerable population groups

Through the topics covered so far in this paper, unvaryingly we touched upon people with pre-existing conditions such as diabetes or high blood pressure who depend on regular medication and elderly who are particularly exposed to impacts of climate change. However, emerging research now establishes that children are, in fact, most vulnerable to the effects of climate change.

Whilst non-governmental groups are now actively highlighting how the intergenerational impacts of climate change are infringing on children's rights to life, education, and protection, the threat to their health is also very real. It is estimated that children will bear the greatest burden of disease as the effects of climate change may start in utero are anticipated to continue long into the future.

Prenatal mental stress is known to be associated with poor perinatal and respiratory outcomes. Studies have established that wildfire smoke exposure, severe ice storms, and hurricanes are associated with poor pregnancy outcomes including reduced birth weight and an increased risk of prematurity, thereby leaving a lifelong impact on children. Researchers have also found that not just gestational exposure to particulate matter (especially with a size of less than 2.5 micrometers) but also co-exposure with extreme heat may independently increase the risk of pre-term birth and hence associated congenital anomalies like septal heart defects.[29]

The vulnerability of children includes aspects of heat stress, respiratory diseases, dehydration, malnutrition, and water- or vector-borne diseases. Additionally, due to more time spent outdoors, children also tend to have a higher exposure to physical injuries, air-borne allergens, air pollution in general, disease-carrying vectors, and to UV light. The impacts of climate change on mental health are another aspect especially relevant for children and will be covered in another part of this series.

Closing remarks

At SCOR, we see it as imperative to partner with our clients to better understand emerging trends and risks, and to develop tailored insurance solutions that help people to remain resilient in this evolving risk landscape. This series aims to contribute to a deeper understanding of the relevance of climate change for life insurance and to facilitate the development of suitable actions.

As shown in this paper, there are several links between climate change impacts and human health and wellbeing, some well understood and others currently less so. What is required is more research in this area and decisive action towards managing public health in context of temperature–health effects when preventing and treating diseases.

Furthermore, amongst the most vulnerable parts of the population, in addition to the elderly and those with pre-existing diseases as described in this paper earlier, those who are confined to bed, living alone, unable to care for themselves or with mental health conditions are also expected to be disproportionally impacted by climate change. All these elements need to be well understood by insurers to develop meaningful solutions for society.

As a global independent reinsurance company, SCOR contributes to the welfare, resilience and sustainable development of society by bridging the protection gap, increasing insurance reach, helping to protect insureds against the risks they face, pushing back the frontiers of insurability and acting as a responsible investor.

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