CONFERENCE PAPER

CLIMATE CHANGE AND DEATH INVESTIGATION: REDEFINING EXPERIMENTAL FORENSIC SCIENCE

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This paper was presented at the 10th International Congress on Advances in Veterinary and Technics (ICAVST 2025) in Sarajevo, Bosnia and Herzegovina.

How to cite this article:
Dervišević E, Spahović H, Zorlak-Čavčić A, Ajanović T. 2025. Climate change and death investigation: Redefining experimental forensic science.
Veterinaria, 74Suppl(1), 13-30.

ABSTRACT

One of the deadliest weather-related occurrences in the world is heat, and as climate change continues, heat-related mortality could rise sharply. According to The Lancet Countdown on Health and Climate Change's 2020 report, an additional 475 million heatwave incidents occurred worldwide in 2019, exposing vulnerable populations, which resulted in excess mortality and morbidity. The number of deaths from heat-related causes among those over 65 has increased by 53.7% during the previous 20 years, with a total of 296,000 deaths in 2018. The burden of heat-related mortality linked to recent decades of human-induced climate change was the subject of another multinational investigation. Human health has already been impacted by climate change and will continue to be so through direct, indirect, and diffuse pathways. Heat-related risks may get worse or get better as a result of interactions between climate change and other trends including urbanization, population growth and aging, and socioeconomic development. High uncertainty among heat-related mortality predictors (such as human behaviour and adaptation) are frequently the cause of such large variations in heat mortality; in contrast, smaller estimations of adverse health effects are produced by slower future population increase and greater adaptability.

Keywords: Climate change, death, experimental, forensic, postmortem

doi.org/10.51607/22331360.2025.74.S2.11

1. Introduction / Background

In forensic practice, the diagnosis of heat stroke-related deaths is typically one of exclusion. This is due to the fact that both gross and histological postmortem findings in heat-related fatalities are non-specific and lack pathognomonic features, while biochemical analyses do not provide definitive markers. Consequently, a comprehensive assessment of circumstantial evidence, in conjunction with detailed autopsy findings, is essential to rule out alternative causes of death, including alcohol or drug intoxication. (Dervišević et al., 2023b). Heat waves, exacerbated by climate change, have already resulted in thousands of fatalities worldwide. These deaths are often due to a combination of extreme heat and high humidity, with wet-bulb temperatures exceeding 35 °C-conditions under which the human body can no longer effectively cool itself through perspiration, leading to fatal heat stress. In addition to direct effects, climate change contributes to a range of intermediate causes of death, including crop failures, droughts, flooding, severe weather events, wildfires, and sea-level rise. These phenomena may not be immediately fatal but significantly increase vulnerability to injury, disease, malnutrition, and displacement. Climate

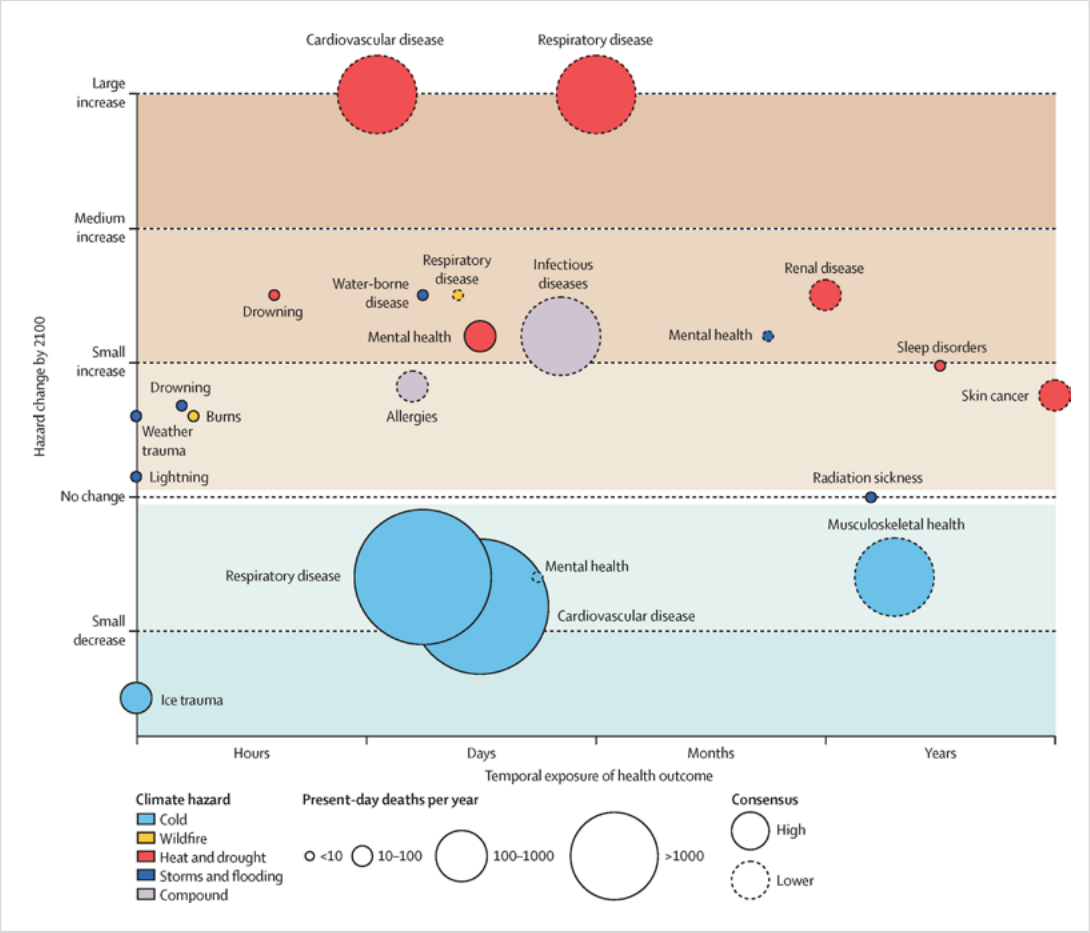

Figure 1 Synthesis of UK climatic patterns and weather-related mortality. The circle sizes show current estimates of annual human mortality from different climate hazards, categorized by the number of deaths (less than 10, between 11 and 100, between 101 and 1000, and more than 1000 annually). Certain climate hazards are represented by the colours of circles (Mitchell et al., 2024). Copyright, Elsevier 2024

change is likely the biggest threat to human life in the future, but nuclear war and biodiversity loss also present serious existential threats. The findings of this research suggest that, on average, burning about 1000 tons of fossil carbon (producing 3700 tons of CO_2) results in one premature death in the future. Measuring carbon emissions in human lives helps non-experts grasp the figures and makes energy policy goals more evident. It is obvious that it is inherently wrong to enable a policy to cause manslaughter. The large mortality tolls resulting from this research and attributable to present carbon emissions have obvious and immediate implications for energy policy. Many deaths during heat waves stem not just from body overheating, but also from heat stress, which can exacerbate pre-existing medical conditions, leading to fatal outcomes (Pearce and Parncutt, 2023). The circle's size represents the current mortality rate, which is primarily caused by heat, drought, and cold hazards, however infectious disease-related mortality is also largely caused by compound climate hazards (Mitchell et al., 2024). As they are also linked to the greatest projected increase in hazard, the largest heat and drought circles show a mortality rate of 100–1000 deaths annually, which is predicted to increase (Figure 1, y axis). Although the projected rise in the hazard is significantly smaller, there is a great deal of uncertainty in the expert replies, and the current mortality rate for several of the cold mortality causes is substantially higher (>1000 deaths annually). The risk to the elderly and heat-related cardiovascular diseases currently dominates mortality outcomes in all but the smallest category.

2. Sudden Cardiac Death (SCD)

Refers to death resulting from unexpected circulatory arrest (van den Tweel and Wittekind, 2016). Estimates of the annual occurrence of sudden cardiac death vary depending on the sources used for the case identification, applied definitions, and methods for extrapolating rates, as well as the autopsy rates conducted in each country (Illing et al., 2020). In prospective studies conducted in the USA, China, Ireland, and the Netherlands, which use standardized definitions and various surveillance sources for case identification, the rate of SCD ranges from 40 to 100 per 100,000 in the general population (Fukuda et al., 2015; P. Zhao et al., 2016). The annual occurrence of SCD grows with age, being 100 times less common in people under the age of 30 (0.001%) compared to those over 35 (Luterbacher et al., 2004). Globally, there is a rising number of deaths occurring indoors with increasing indoor temperatures, such as in bathrooms while taking a bath or in saunas. Autopsy rates are generally low and vary considerably between countries, with rates falling below 10% of all deaths in the United States, compared to 23.8% in Finland (Illing et al., 2020; Luterbacher et al., 2004). Some protocols for conducting autopsies in suspected SCD cases can differ significantly, even in the different regions of the same country (Ferron et al., 2006). These discrepancies in autopsy rates and procedures likely contribute to variations in the occurrence and the causes of SCD (Thommen, 2005). Scientific data regarding the role of hyperthermia as a cause of sudden cardiac death (SCD) remain limited highlighting the need for further research in this area (Dervišević et al., 2023a; Dervišević et al., 2023c).

The causes of sudden cardiac death (SCD) are varied, with structural heart alterations being a key contributor. Cardiac remodeling, a common feature in several cardiovascular disorders—including hypertrophic cardiomyopathy, dilated cardiomyopathy, and chronic heart failure—frequently underlies these structural changes (Iba et al., 2025a; Sacco et al., 2023) (Figure 2). SCD can also result from coronary conditions such as arterial spasms, progressive atherosclerosis, ischemia, or myocardial infarction. However, not all cases are linked to ischemic events; some occur in individuals with inherent conduction system disorders. Furthermore, external substances can act as triggers in certain instances of SCD (Sacco et al., 2023). Differentiating the underlying cause of death in such cases using standard autopsy and histopathological methods remains highly challenging. Moreover, histopathological

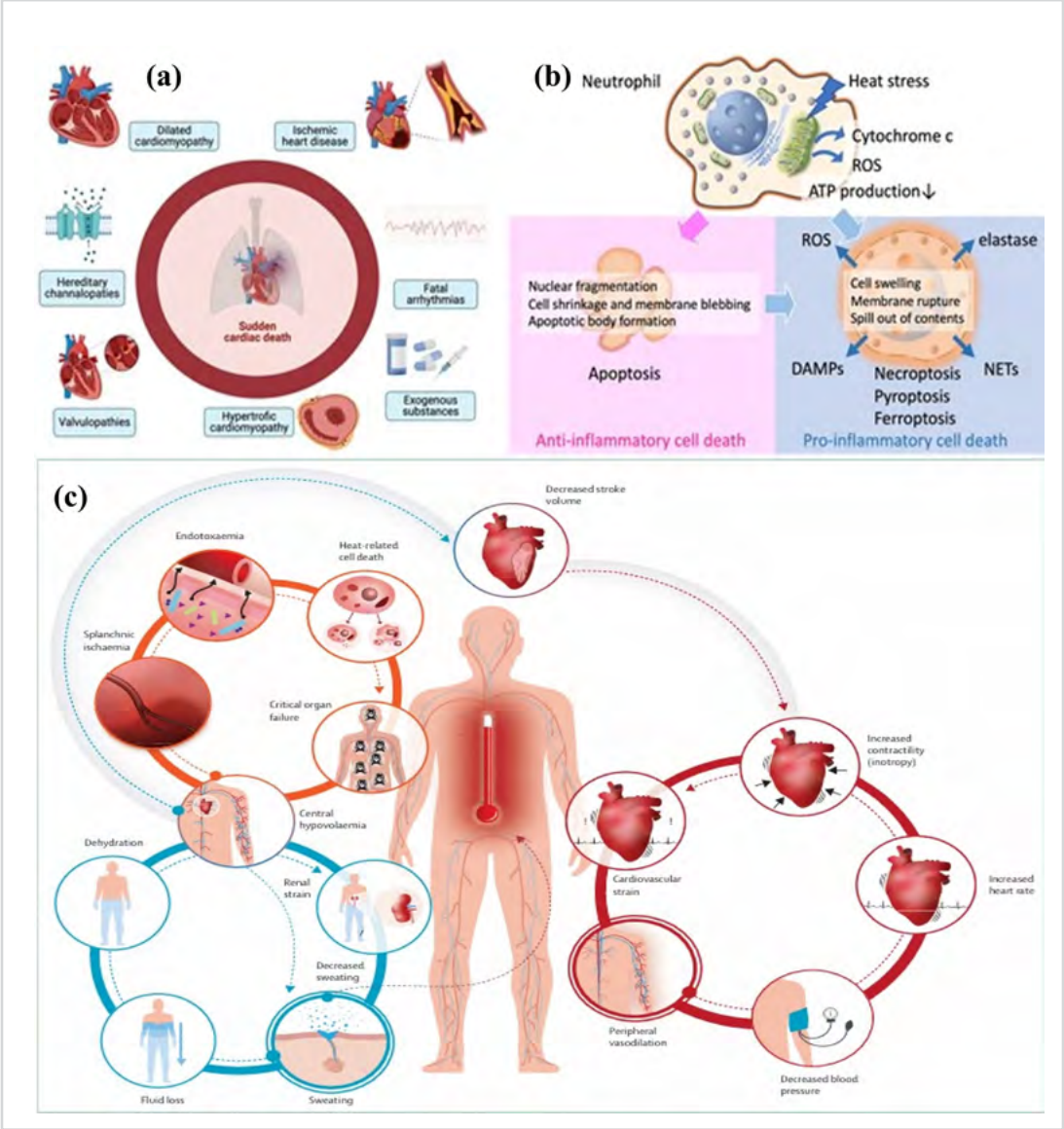

Figure 2 (a) Causes of SCD (Sacco et al., 2023). Copyright MDPI © 2023. b) Pro-inflammatory and anti-inflammatory cell death mechanisms in heatstroke (Iba et al., 2025a). Copyright BMC © 2025. (c) An example of the physiological mechanisms underlying heat stress in humans (Ebi et al., 2021). Copyright The Lancet © 2021

evaluations often suffer from interobserver variability, influenced by the pathologist's level of experience and subjective interpretation of tissue morphology. As a result, histological findings alone may not provide definitive conclusions regarding the precise cause of death. This limitation poses significant implications not only for epidemiological research and prevention strategies but also for legal contexts, where establishing the exact cause of death and any associated responsibilities is crucial (Sacco et al., 2023). The two main ways that the human body reacts to heat stress are by secreting perspiration onto the skin, which then evaporates and dissipates body heat, and by shifting blood flow towards the skin (vasodilation), which enhances heat transmission from muscles to skin and ultimately to the environment. Together with extra thermal

information from temperature-sensitive nerve cells in the skin and other parts of the body, the brain controls these physiological heat loss reactions. Non-thermal cues like cytokines, metaboreceptors (a kind of chemoreceptor that reacts to metabolic products produced by working muscles), and dehydration can also have an impact on this regulation. These physiological heat stress reactions can have varying effects on individuals and are required to restrict increases in core temperature (Ebi et al., 2021).

Elevated body temperatures can negatively affect the function of mitochondrial electron transport chain components, with complex I showing particular sensitivity. This disruption compromises mitochondrial performance, diminishes ATP synthesis, and results in an energy deficit within the cell, thereby promoting cellular dysfunction and lowering resistance to physiological stress (Iba et al., 2025b). The damage to mitochondria induces apoptosis (Iba et al., 2025a). Exposure to extreme heat (typically ≥43 °C) can trigger apoptosis through mitochondrial-mediated pathways, including the release of cytochrome c into the cytosol, ultimately resulting in programmed cell death. This biological response forms the basis of hyperthermia-based cancer treatments, which exploit elevated temperatures to selectively induce apoptosis in tumor cells (Iba et al., 2025a). However, because apoptosis is an energy-dependent process requiring adequate ATP levels, cells experiencing severe ATP depletion are more likely to undergo necrosis instead, a form of cell death that often triggers an inflammatory response. In summary, hyperthermia-induced mitochondrial dysfunction-characterized by increased membrane permeability, excessive reactive oxygen species (ROS) production, and disruption of the electron transport chain-can initiate both apoptotic and necrotic pathways, ultimately contributing to multiple organ failure. Therefore, damage to mitochondria caused by elevated temperatures represents a key pathway contributing to cell death during heatstroke (Iba et al., 2025a). Heat stress can trigger multiple forms of programmed cell death.

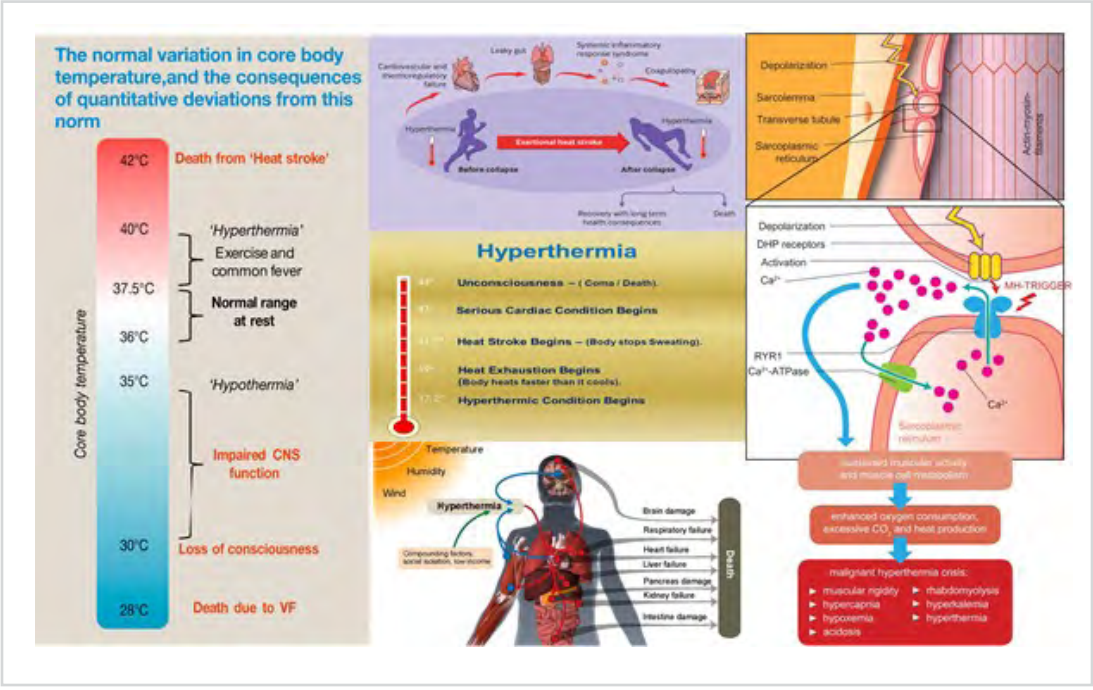

Figure 3 Summary of the main pathophysiological factors participating in exertional heat stroke, from hyperthermia to death (Iba et al., 2025a). Copyright MDPI © 2019

Among them, apoptosis is a non-inflammatory process marked by cell shrinkage and fragmentation of the nucleus. During apoptosis, cells break down into apoptotic bodies that are efficiently removed by phagocytes. In contrast, proinflammatory forms of cell death—such as necroptosis, pyroptosis, and ferroptosis—exhibit common characteristics including cellular swelling and nuclear membrane rupture. These processes result in the release of intracellular components, which in turn stimulate inflammatory responses (Figure 3).

Poor blood flow in hypoxic areas directly contributes to their ease of heating, as higher temperatures are more readily attained due to the limited heat dissipation by perfusion. Since tumour cells are far more susceptible to heat than cells in an environment with enough oxygen, it is generally believed that hyperthermia kills them specifically. Furthermore, hyperthermia at 41–43 °C enhances membrane permeability and decreases DNA damage repair, both of which improve the efficacy of medications that kill tumour cells. Lastly, perfusion and extravagation are improved even at relatively low temperatures, increasing medication delivery (van Rhoon et al., 2020a).

3. Key connections between human health and climate change

There is a correlation between elevated mortality risk and both high and low temperatures. From 2000 to 2019, it was estimated that suboptimal temperatures caused 5,083,173 fatalities annually, or 9.43% of all deaths globally. Furthermore, it is anticipated that the excess mortality linked to temperature would continue to rise until the 2050s. The temperature shift may result in a variety of illnesses in addition to an increase of fatalities, as seen in Figure 4. For instance, exposure to excessively high temperatures has been linked to an increased risk of hospitalization and ED visits for conditions affecting the respiratory, metabolic, and cardiovascular systems. Climate and environmental changes brought on by global warming may increase the likelihood of a number of health consequences (Q. Zhao et al., 2022). The vast majority of harmful chemicals affect important targets inside the body; hence the relevant "environment" is the body's interior chemical environment. This will help us reinstate human health as the main emphasis of exposure science. Lastly, biological monitoring for exposure assessment would be encouraged by concentrating on the interior chemical environment. The body's internal chemical environment is referred to as the "environment" in this context, and the amounts of chemically active substances in this internal environment are referred to as "exposures." Instead of using a bottom-up strategy that analyses food, water, air, and so forth, it makes more sense to use a top-down strategy based on biomonitoring (such as blood sampling) to investigate the exposome, current research indicates that non-genetic factors account for roughly 90% of the risks of chronic diseases (Rappaport, 2011).

Figure 5 shows the temperature ranges for the various effects. A gentle heat treatment, on the other hand, has not been linked to any toxicity and may cause a variety of alterations in cellular and molecular physiology. An increase in temperature may have an impact on a number of targets within the cell, such as the cytoskeleton, membranes, and macromolecule production. Evidence of immunological activation and the emergence of systemic immune responses is also present. Particle energy rises with temperature, making collisions more effective and accelerating chemical reactions. The temperature rises by 10 °C when the reaction rate is increased, and the temperature coefficient values differ depending on the enzyme. For the majority of enzymes found in animal cells and tissues, the ideal temperature range is 35 – 50 °C (Pereira Gomes et al., 2019).

Water makes up the majority of the human body, making up about 60% of its weight. The intracellular space contains around 40% of this aqueous proportion, followed by the extracellular space with 2%, the interstitial space with 15%, and the intravascular space with 5%. Although the human body needs a lot of water, it is unable to store much of it, so in order to keep internal equilibrium, water must be consumed. Water produces the heat

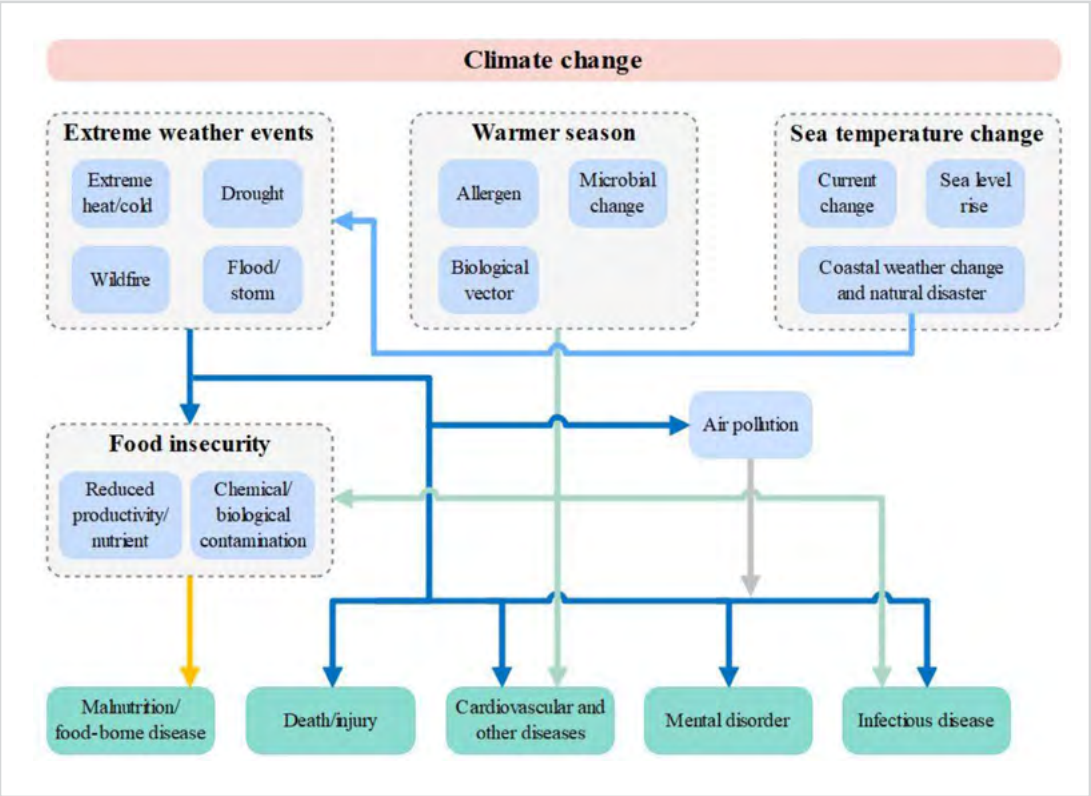

Figure 4 Key connections between health outcomes and climate change, including four groups extreme weather events, warmer season, sea temperature change and food insecurity (Q. Zhao et al., 2022). Copyright, Elsevier 2022

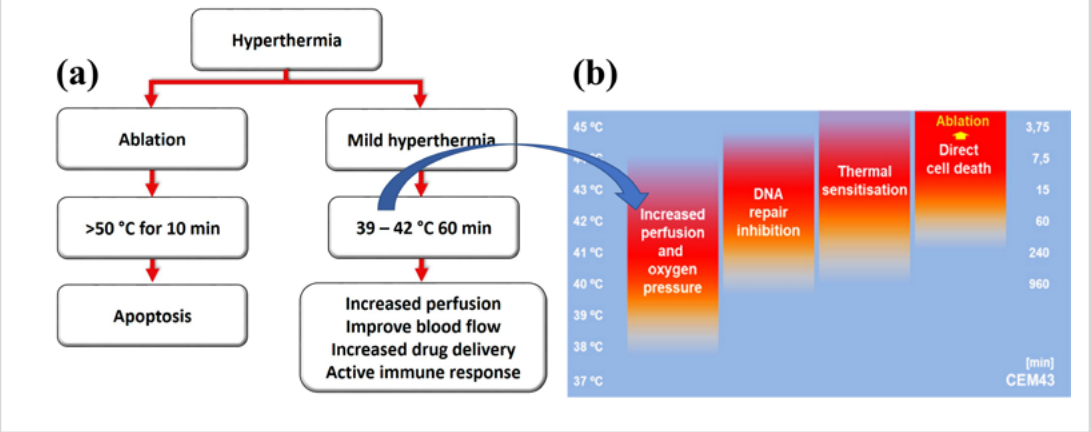

Figure 5 (a) Depending on the severity and duration, mild hyperthermia and ablation cause different types of cell damage (Gomes et al., 2019). Copyright MDPI © 2019. (b) Various biological mechanisms of hyperthermia, including the temperature at which the effect starts and, if relevant, the maximum temperature at which it stops or transitions to a higher one (van Rhoon et al., 2020a). Copyright Elsevier © 2020

energy required to break H-bonds when it is liquid at 37°C. The duration of this operation is roughly 1 to 20 picoseconds (1 ps = 10−12 s). Because a new bond is formed with a different molecule as soon as the first one breaks, molecules are in perpetual motion. This phenomenon is referred to as "flickering clusters," which are transient clusters of hydrogen bonds that form in the liquid phase of water. The patient's cardiac output is closely related to the T° variations that occur after intravenous fluid administration. An elevation of 1°C in the patient's core body temperature may occur for every 4 litres of saline solution given. Within the first 48 hours after a cardiac arrest, there is a correlation between increased mortality and each degree Celsius above 37.7°C (Robayo-Amortegui et al., 2024). As shown in Figure 6(a), lowering core body temperature in traumatic brain injury patients can reduce cerebral metabolism by as much as 6% for every degree Celsius that the temperature is lowered. This reduces cellular damage. Finally, as shown in Figure 6(b), water disease affects the integumentary system via the same mechanism of capillary leakage, venous congestion, and water extravasation into the interstitial space.

Figure 6 (c-d) summarizes the key pathophysiological mechanisms involved in exertional heat stroke. During physical exertion, hyperthermia develops when the cardiovascular system can no longer maintain effective thermoregulation. This results in altered blood flow, increasing intestinal permeability and allowing gut contents to enter the bloodstream—a process described by the leaky gut hypothesis. Both hyperthermia and translocated intestinal contents trigger a systemic inflammatory response syndrome (SIRS), which contributes to disseminated intravascular coagulation (DIC), manifesting as coagulopathy. These pathological responses often persist after collapse, continuing until the individual is cooled and consciousness is restored. The primary outcomes of exertional heat stroke are either fatality or survival accompanied by long-term health complications. The world is witnessing an increasing number of sudden deaths resulting from hyperthermia. Current scientific evidence suggests a causal link between hyperthermia and cardiac response, in line with the pathophysiological sequence of events associated with hyperthermia, although the exact mechanism of onset remains unknown (Richmond et al., 2015). From a forensic medicine perspective, there is no data indicating the exact pathophysiological mechanism that leads to sudden cardiac death. One of the significant advancements in clinical cardiology involves the identification of biochemical markers indicative of myocardial damage. In forensic medicine, researchers have been seeking a biochemical marker that would serve as the gold standard for post-mortem cardiac and non-cardiac mechanisms leading to ischemia and subsequent necrosis of cardiomyocytes (Marui et al., 2017). There is currently no data available on the exact percentage of deaths occurring during bathing, but there is an increasing amount of research focused on what precisely happens pathophysiological during such events (Mørch et al., 2017). Studies conducted thus far present unclear and unresolved pathophysiology and suggest the need for further investigation, as it is crucial to understand that death occurs during exposure to high water temperatures. Bathing in hot water has been associated with sudden death (Walter and Carraretto, 2016).

Although forensic medicine primarily addresses whether a death is violent, it is also very useful to help identify the cause of sudden deaths and the markers that can provide post-mortem evidence of the most likely cause of death. Among sudden deaths, it often turns out that cardiac aetiologies are the most common (Walter and Carraretto, 2016). Normal body temperature is approximately 37 ° (33.2–38.2 °C). This range becomes even narrower when rectal measurements are used instead of oral, tympanic, or axillary methods (Yang et al., 2017). Normal fluctuations in body temperature occur throughout the day, throughout the month, and across a lifetime.

Even slight deviations in core temperature can activate thermoregulatory mechanisms, and variations outside the physiological range can be life-

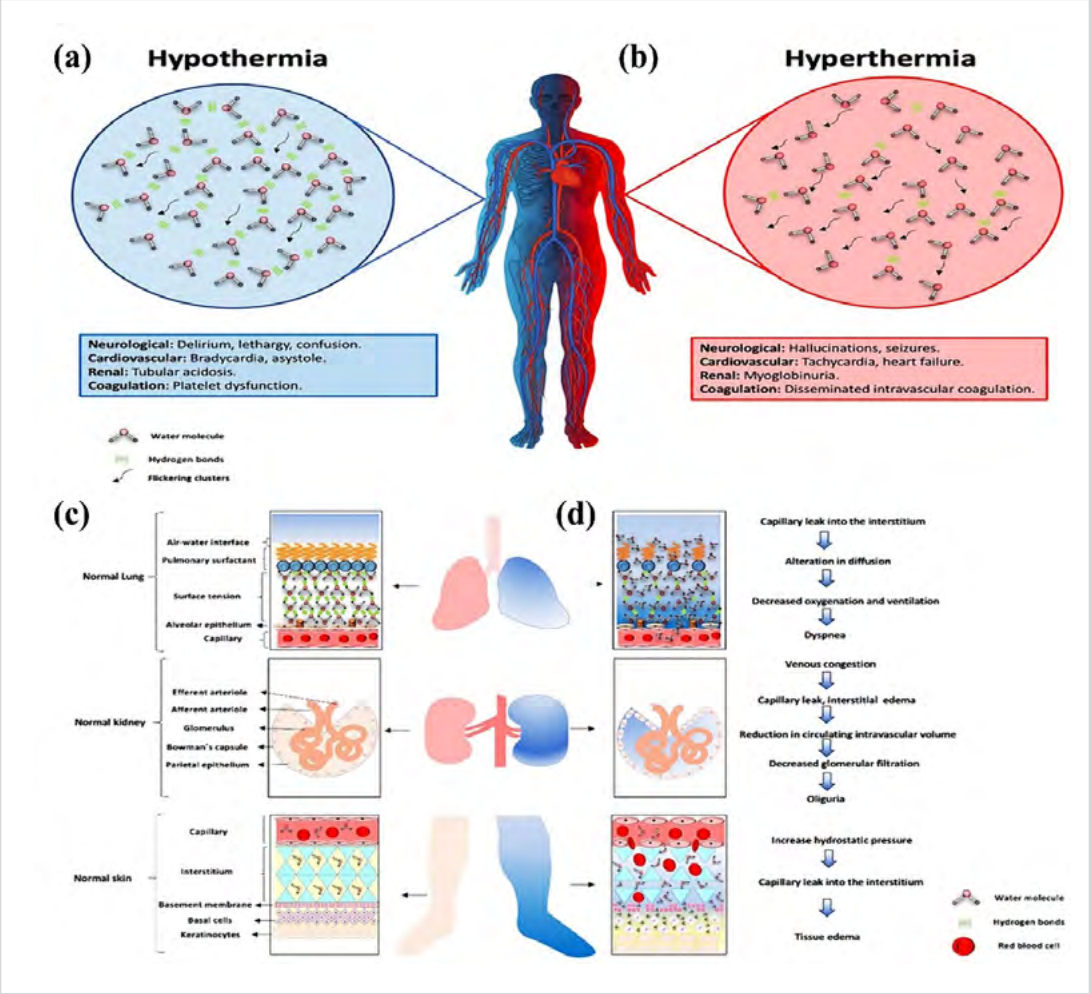

Figure 6 (a-b) The effects of water behaviour on the human body during hypothermia and hyperthermia. (c-d) The human body, which is mostly made of water, has amazing biochemical qualities and is essential for functions like thermoregulation and protein transport [22]. Copyright Frontiers in Medicine © 2024

threatening. A measured body temperature that goes above 41,9 °C can cause cytotoxicity, protein denaturation, and damaged DNA, potentially leading to organ failure. Conversely, if the body temperature falls in hypothermia, changes in cardiovascular, haematological, neuromuscular, and respiratory functions associated with this can also be fatal (Lepock, 2003).

Core body temperature is preserved within a range of ±6 °C (10-55°C) in environments, meanwhile the skin temperature fluctuates with the external environment. Oral temperature usually falls between 36.5 and 37 °C, whereas the rectal measured temperature is typically 0.5 °C higher (Natarajan et al., 2015). The normal range of body temperature is from 36 to 37.5 °C (Natarajan et al., 2015). During physical activity, body temperature can reach 38-40°C, while exposure to extreme cold can cause it to drop to 35°C.

Body temperature varies depending on the measurement site. n thermoregulation studies, the body is generally divided into two regions:

the skin, which fluctuates with environmental conditions, and the inner core (internal organs), which maintains a more stable temperature. The preoptic area of the anterior hypothalamus is essential for regulating temperature. Nerve receptors are typically more sensitive to heat than to cold, with stimulation of certain brain areas promoting sweating, while cooling can impair heat dissipation mechanisms.

Additionally, there are significantly more receptors for detecting cold than for heat, all of which signal to the hypothalamus.

Hyperthermia can be caused by heat stroke, infectious diseases, hypothalamic damage, necrosis, malignity and any stimulus that could activate immune cells to release endopyrogens (Heled et al., 2013; Lassche et al., 2019). Pathohistological findings in cardiovascular causes of sudden death vary depending on whether the event is acute, subacute, or chronic, leading to specific and nonspecific signs on the heart. In acute and subacute cases, such as those caused by hyperthermia, nonspecific unclear fibrosis or endocardial fibrosis with focal necrosis and eosinophilic infiltrates is often observed.

Previous studies of heart tissue exposed to heat have identified focal areas of necrotic fibers, which show fragmentation of microfilaments and create gaps between cardiomyocytes (Dervišević et al., 2023a).

Signs of relaxation and blood extravasation during hyperthermia have also been recorded, resulting from increased cardiac output and vasodilation as a response to high temperatures. Cardiac tissue has shown significant oedema with thinning of muscle fibres surrounding haemorrhagic areas. Classic microscopic examination of the heart following exposure to high temperatures has revealed moderate atherosclerosis, accompanied by necrotic foci and oedema (Quinn et al., 2014). A multidisciplinary approach that integrates basic medical sciences—such as medical biochemistry, pathology, and pathophysiology—through clinical cardiology can aid forensic medicine in identifying the cause of death more precisely and simply. This could be a significant study in the realm of fundamental research, contributing to clarifying the causes of death in forensic practice.

4. Discussion

Global warming is expected to have a greater impact on health due to temperature, although it is still unknown how population aging may affect these patterns. According to the findings, heat-related mortality in 800 locations across 50 countries/areas will rise by 0.5%, 1.0%, and 2.5%, respectively, at 1.5°C, 2°C, and 3°C of global warming; of these, 1 in 5 to 1 in 4 heat-related deaths can be ascribed to population aging. Even though progressive warming alone is predicted to reduce cold-related mortality, population aging will largely reverse this trend, resulting in a net increase in cold-related mortality of 0.1%–0.4% at 1.5–3 °C global warming (Chen et al., 2024). When global warming hits 1.5°C, 2°C, and 3°C, population aging alone causes an average increase in future temperature-related excess mortality of 0.8% (95% eCI: 0.6% to 0.9%), 1.7% (1.2% to 2.1%), and 2.6% (0.9% to 3.5%) for the non-optimal (cold and hot) temperature-related mortality, as seen in Figure 7. By contrast, under the maximum amount of warming (3 °C), climate alone was responsible for 3.0% (i.e., 0.08%/2.67%) of the net changes in non-optimal temperature-related mortality. Approximately two-thirds of the countries or regions analyzed (36 across all warming scenarios) showed a significant increase in mortality burden attributable to population aging. In contrast, only a few countries exhibited a significant decrease in mortality linked to aging—specifically 5, 3, and 2 countries at warming levels of 1.5 °C, 2 °C, and 3 °C, respectively.

One of the main causes of the rise in temperature-related mortality is aging.9. According to Yang et al. (2021), between 128,000 and 229,000 people died in China in the 2090s as a result of heat-related causes. The historical exposure-response relationship between temperature and the old population across various age categories is shown in Figure 8. In particular, the senior

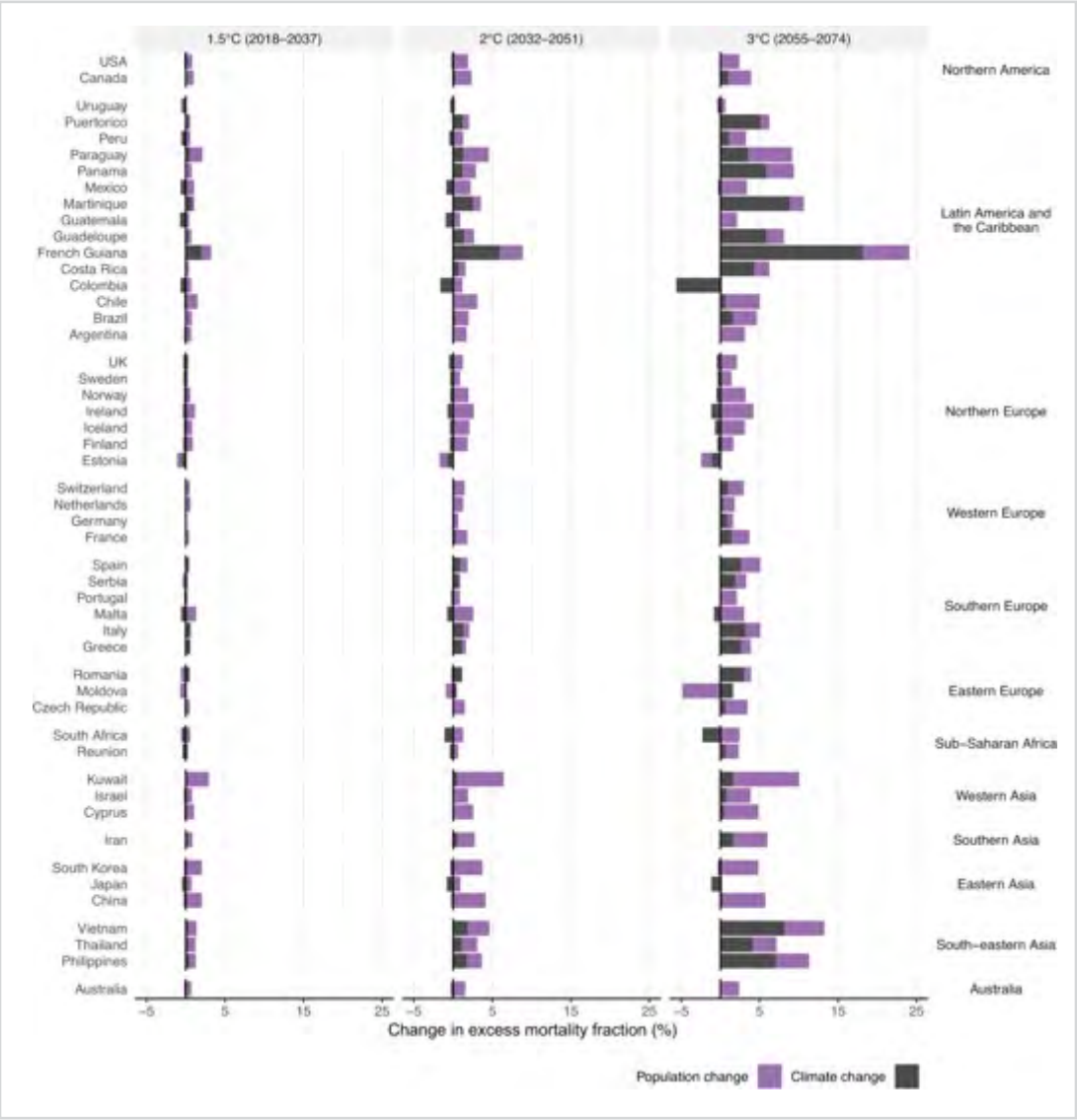

Figure 7 Changes at the country or area level related to climate change and population aging are presented for global warming scenarios of 1.5°C, 2°C, and 3°C, using 20-year periods compared to the historical baseline from 1995 to 2014. The 20-year moving average of global mean temperature is projected to first exceed 1.5°C, 2°C, and 3°C above pre-industrial levels (defined as 1850–1900) during the intervals 2018–2037, 2032–2051, and 2055–2074, respectively (Chen et al., 2024). Copyright, Nature © 2024

population in Nantong experienced an increase in the minimum mortality temperature (MMT) as they became older. The MMT was 24, 25, and 26 °C for the elderly (65–79, 80–89, and 90+, respectively). Compared to MMT, the mortality risk (RR) at the 97.5th percentile (31 °C) was 1.192 (95% CI:1.139–1.246), 1.340 (95% CI:1.283–1.399), and 1.492 (95% CI:1.402–1.588), respectively, and at the 2.5th percentile (1 °C) was 1.280 (95% CI:1.209−1.355), 1.708 (95% CI:1.583−1.844), and 2.217 (95% CI:1.981−2.480), respectively. When selected a maximum lag time of 10–21 days for temperature, 4–6 days for relative humidity, and 4–8 days for air

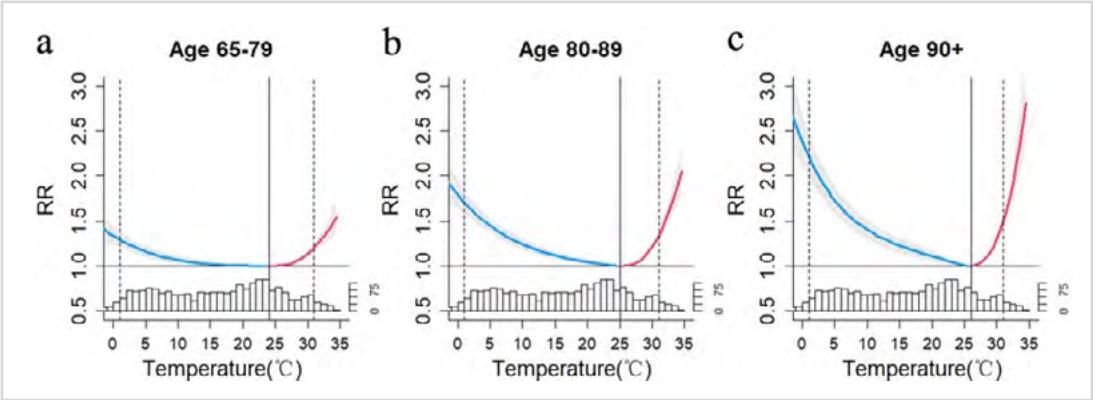

Figure 8 (a) ages 65–79; (b) ages 80–89; and (c) ages 90+. The bars show the frequency of temperatures from 2012 to 2017 at intervals of 1 °C. The associated 95% confidence intervals are indicated by the error band. The vertical dashed lines show the 2.5th and 97.5th temperature percentiles, whereas the vertical solid line shows the minimal mortality temperature (Huang et al., 2023). Copyright, Nature © 2023

pollutants, the temperature–mortality relationships were comparable (Huang et al., 2023).

Annual hot and cold degree days were used to regress the annual age-standardized mortality rates (ASMRs). Furthermore, people 75 years of age and older are predicted to see greater declines in ASMR as a result of less cold weather. When the analysis was stratified by the two most common causes of non-accidental and non-cancer deaths, it was found that, over the years under all scenarios, the increases in cardiovascular ASMR due to heat were greater than the increases in respiratory ASMR due to heat, especially during the 2090s (e.g., the increases for cardiovascular and respiratory deaths in the 2090s were 135.99% and 113.85%, respectively), while the decreases in respiratory ASMR due to less cold weather were larger over the years under all scenarios (Wang et al., 2022). In the current projection, age was anticipated to be a significant effect modifier (Figure 9). The increase in both hot and net effect under representative concentration pathways (RCP) was remarkably steep, with the net change from 0.12% in 2030s to 89.25% in 2090s (Table 1).

Table 1 Projected percentage increases in age-standardized mortality rates for Hong Kong are shown for the 2030s, 2050s, 2070s, and 2090s, relative to the baseline period of 2014–2018

RCP	2030s Hot	2030s Cold	2030s Net	2050s Hot	2050s Cold	2050s Net	2070s Hot	2070s Cold	2070s Net	2090s Hot	2090s Cold	2090s Net
2.6	3.20	−3.37	−0.29	4.69	−4.06	0.44	5.01	−4.52	0.27	5.15	−3.80	1.15
4.5	3.62	−3.78	−0.29	12.28	−5.27	6.36	17.54	−6.62	9.76	19.18	−7.12	10.69
6.0	−1.33	−1.91	−3.21	2.78	−4.30	−1.64	13.93	−6.71	6.29	23.42	−8.25	13.24
8.5	3.81	−3.55	0.12	21.30	−7.30	12.44	54.13	−9.82	38.99	116.49	−12.58	89.25

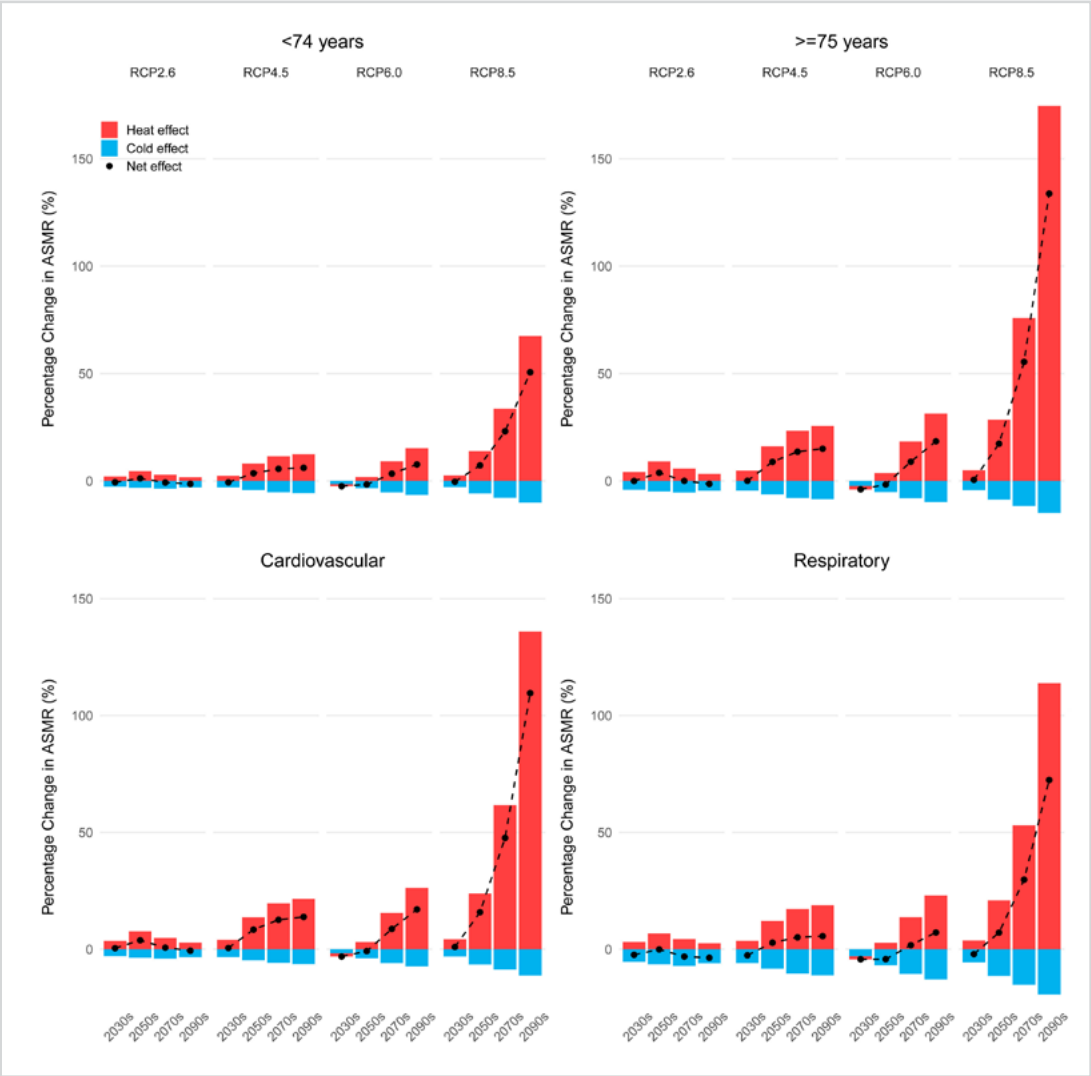

Figure 9 Using 2014–2018 as a reference baseline, calculate the average percentage changes in ASMR by age and cause of death in Hong Kong during the 2030s, 2050s, 2070s, and 2090s under various climate change scenarios. Age-standardized mortality rate, or ASMR (Wang et al., 2022). Copyright, Elsevier © 2022

5. Challenges and limitations in the implementation of technologies in forensic sciences

The forensic science specialist must confront and resolve a number of issues brought about by the exponential advancement of technologies, the creation of disruptive technologies, and the solutions that are developed on a regular basis. Although there are certain benefits to using new technologies, there are also technical drawbacks that need to be addressed. In the case of cameras, which have limited precision and resolution in images and 3D models, the technical constraints that are shown are contingent upon the features of the technological equipment utilized. Despite advancements in technology, challenges still exist in getting past technical constraints to guarantee the accuracy of forensic findings. Financial difficulties arise from the substantial investment needed to deploy cutting-edge

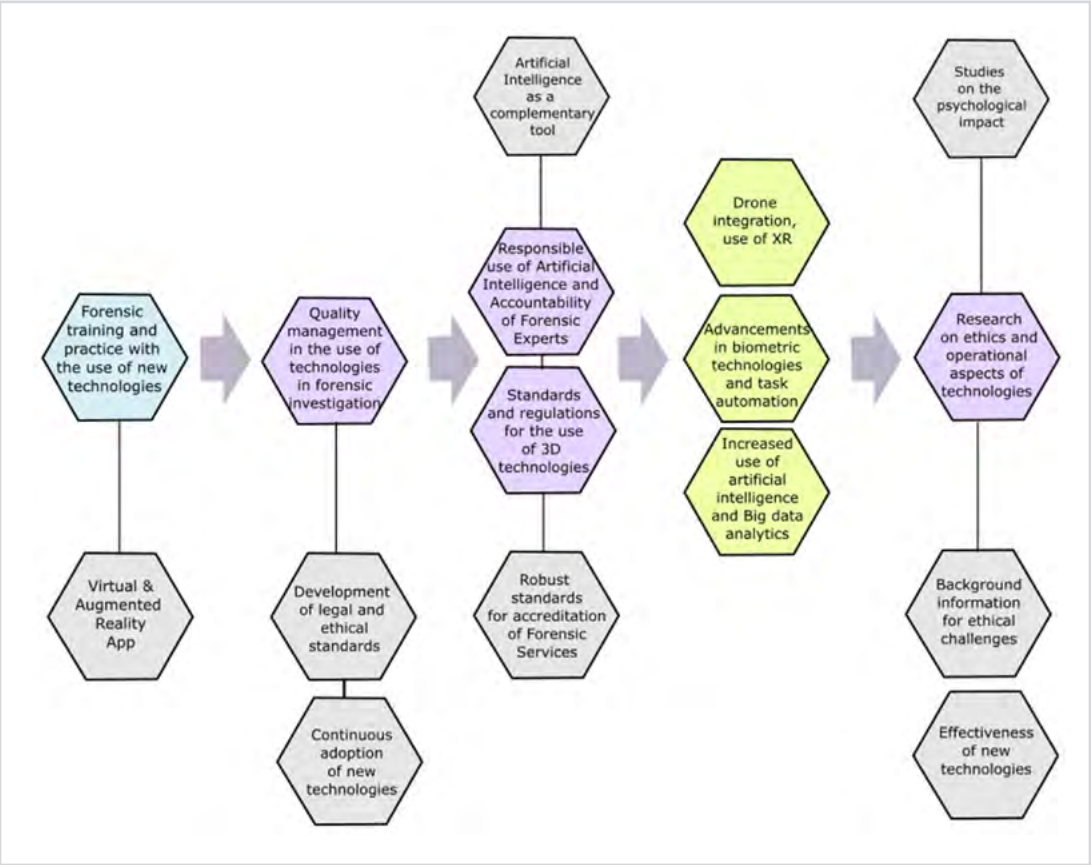

Figure 10 Upcoming advancements in forensic investigation technology use (Chango et al., 2024). Copyright, MDPI 2024

technologies. There are moral and legal issues with artificial intelligence use. Strict ethical standards have been implemented as a result of numerous inquiries. The significance of considering AI as an additional tool rather than a replacement for human judgment is highlighted by worries about the possibility of false information. As a result, taking preventative action is essential. To reduce these hazards, safety procedures must be followed and the detrimental effects must be recognized. Even with better sensors and more processing power, some forensic systems still struggle with accuracy. Before widespread adoption, thorough validation is necessary to guarantee dependable outcomes. Large-scale data-generating technologies, such 3D laser scanning, make information management difficult (Chango et al., 2024). The term "climate change" then describes how those components have changed throughout time. This includes, among other things, natural changes brought on by variations in the sun's activity or the tilt of the earth's axis. The phrase has become frequently used to describe manmade climate change in recent years. One crucial component of the job done by forensic pathologists and forensic anthropologists is estimating the post mortem delay. Although most people believe that temperature is the most significant factor influencing decomposition, some formulations also include relative humidity for a more thorough approximation. Anthropogenic climate change affects both of these factors (Strack and Smith, 2023). It is well recognized that various techniques used in forensic investigations can pose risks to both human health and the environment. This is often due to the chemical composition of the powders and liquids applied, as well as their

environmental impact. As a result, identifying safer alternatives—particularly for materials like fingerprint developers—has become a priority. Natural substances such as seaweed, certain spices, and chalcones have been suggested as more eco-friendly and cost-effective options. Digital forensics is one of the forensic and crime scene investigation fields that is expanding, as seen in Figure 10. Both the sharp rise in the amount of data being captured and the growing complexity of the digital environment are proving to be problems in this field (Thompson, 2024).

CONCLUSION

There is an urgent need for collaborative, multidisciplinary research to better understand the pathophysiological impacts of climate change on immunological disorders. To accurately measure how climate change influences immune function and disease patterns, inform mitigation and adaptation strategies, and evaluate their effectiveness, innovative data science approaches, novel biomarkers, and economic modeling are essential. Efforts to tackle disparities in climate change impacts must be grounded in the principles of justice, equity, diversity, and inclusion (JEDI). The health of people, animals, and the ecosystem as a whole are all at existential risk due to climate change. The danger of serious diseases has increased as a result of the shifting exposuresome. Linked to immunological dysregulation, which includes autoimmune disorders, cancer, allergies, and asthma. Although there has been progress in creating treatment options for these conditions, these strategies are insufficient to address the problems brought on by climate change.

ACKNOWLEDGEMENTS

This review is a part of Special Issue in journal Veterinaria as one of the topic tht was exhibited at the International Congress ICAVST, that hold in Sarajevo, July 2025.

The authors extend their gratitude to colleagues and institutions that supported this work.

CONFLICT OF INTEREST

The authors declared that there is no conflict of interest.

CONTRIBUTIONS

Conception: ED; Design: ED, TA; Supervision: HS, AZČ; Materials: TA, ED; Data Collection and/or Processing: TA; Analysis and/or Interpretation of the Data: TA; Literature Review: HS; Writing: ED, TA, AZČ; Critical Review: HS

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KLIMATSKE PROMJENE I ISTRAGA SMRTI: REDEFINISANJE EKSPERIMENTALNE FORENZIČKE NAUKE

SAŽETAK

Toplota je jedan od najsmrtonosnijih vremenskih fenomena u svijetu, a kako klimatske promjene napreduju, smrtnost uzrokovana toplotom bi se mogla naglo povećati. Prema izvještaju The Lancet Countdown on Health and Climate Change iz 2020. godine, tokom 2019. godine zabilježeno je dodatnih 475 miliona slučajeva toplotnih talasa širom svijeta, koji su izložili ranjive populacije i doveli do povećane smrtnosti i obolijevanja. Broj smrtnih slučajeva uzrokovanih toplotnim udarom kod osoba starijih od 65 godina povećan je za 53,7% u posljednjih 20 godina, sa ukupno 296.000 smrtnih slučajeva u 2018. godini. Teret smrtnosti povezan s visokim temperaturama, a koji je povezan s decenijama klimatskih promjena izazvanih ljudskim djelovanjem, predmetom je brojnih naučnih istraživanja. Klimatske promjene su već uticale na ljudsko zdravlje i nastaviće se njihovo djelovanje rezultirajući povećanjem broja smrtnosti od toplotnih udara. Rizici povezani s vrućinom mogu se pogoršati ili poboljšati kao rezultat interakcije između klimatskih promjena i drugih trendova, uključujući urbanizaciju, rast i starenje populacije te socioekonomski razvoj. Visok stepen nesigurnosti među prediktorima smrtnosti povezanih s vrućinom (kao što su ljudsko ponašanje i prilagodba) često je uzrok velikih varijacija u procjenama smrtnosti; nasuprot tome, sporiji rast stanovništva i veća sposobnost prilagodbe proizvode niže procjene štetnih zdravstvenih efekata.

Ključne riječi: Eksperiment, forenzika, klimatske promjene, postmortum, smrt