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Humidity's Role in Heat-Related Health Outcomes: A Heated Debate

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Background: As atmospheric greenhouse gas concentrations continue to rise, temperature and humidity will increase further, causing potentially dire increases in human heat stress. On physiological and biophysical grounds, exposure to higher levels of humidity should worsen heat stress by decreasing sweat evaporation. However, population-scale epidemiological studies of heat exposure and response often do not detect associations between high levels of humidity and heat-related mortality or morbidity. These divergent, disciplinary views regarding the role of humidity in heat-related health risks limit confidence in selecting which interventions are effective in reducing health impacts and in projecting future heat-related health risks.

Objectives: Via our multidisciplinary perspective we seek to *a*) reconcile the competing realities concerning the role of humidity in heat-related health impacts and *b*) help ensure robust projections of heat-related health risks with climate change. These objectives are critical pathways to identify and communicate effective approaches to cope with present and future heat challenges.

Discussion: We hypothesize six key reasons epidemiological studies have found little impact of humidity on heat–health outcomes: *a*) At high temperatures, there may be limited influence of humidity on the health conditions that cause most heat-related deaths (i.e., cardiovascular collapse); *b*) epidemiological data sets have limited spatial extent, a bias toward extratropical (i.e., cooler and less humid), high-income nations, and tend to exist in places where temporal variations in temperature and humidity are positively correlated; *c*) analyses focus on older, vulnerable populations with sweating, and thus evaporative, impairments that may be further aggravated by dehydration; *d*) extremely high levels of temperature and humidity (seldom seen in the historical record) are necessary for humidity to substantially impact heat strain of sedentary individuals; *e*) relationships between temperature and humidity are improperly considered when interpreting epidemiological model results; and *f*) sub-daily meteorological phenomena, such as rain, occur at high temperatures and humidity, and may bias epidemiological studies based on daily data. Future research must robustly test these hypotheses to advance methods for more accurate incorporation of humidity in estimating heat-related health outcomes under present and projected future climates. https://doi.org/10.1289/EHP11807

Introduction

Heat extremes have dire consequences for human health, increasing mortality, morbidity, and occupational health hazards. Heat–health risks are heightened for people who are older,[1,2] have chronic diseases,[3,4] live in hot climates,[5] or are socioeconomically disadvantaged.[6] Recent heat waves have demonstrated this risk in, for example, Karachi, Pakistan, in 2015 (1,220 fatalities from heatstroke in $\sim$30 d[7]), Japan in 2018 (34,147 heat-related emergency transports in 11 d[8]), and British Columbia, Canada, in 2021 (619 heat-related deaths in 6 d[9]). About 37% of heat-related mortality in 43 countries over the past few decades has been attributed to climate change.[10]

These risks are expected to further increase with climate change, given that heat waves are robustly projected to increase in frequency, intensity, and duration.[11] In addition, as the atmosphere warms, evaporation of water increases, leading to higher humidity across much of the globe.[12] Throughout this commentary, we use the term humidity to broadly refer to mass-based measures of the amount of moisture in the air (e.g., specific and absolute humidity); unless otherwise indicated, we do not mean relative humidity (RH), as discussed in the "Understanding Humidity: Scales and Definitions" section. To the extent that higher humidity reduces the human body's ability to cool itself through sweat evaporation, increasing humidity would aggravate heat strain and the risk of adverse health outcomes. Increasing temperatures and humidity with climate change thus pose a potential compound risk for human health.

The recognition that humidity, as well as temperature extremes, may determine health outcomes led to the concept of "moist heat stress," which is projected to increase with climate change. Buzan and Huber[13] provided a comprehensive review of this work using combined temperature–humidity metrics and proposing physiological limits to human adaptive capacity. One of these metrics, wet-bulb temperature (T_w), is the temperature reached if a parcel of air cools to moisture saturation via evaporation, with latent heat supplied by the parcel; it can be measured by a thermometer covered in a wet cloth. Sherwood and Huber[14] argued that a $T_w > 35$°C for $\sim$6 h is not survivable, projecting uninhabitable regions under a global mean warming of 7°C, with these regions projected to encompass the majority of the human population under 11–12°C of warming (temperatures not projected for this century). Today, 35°C T_w exceedances are beginning to emerge in weather observations, especially near the Persian Gulf and Indus River Valley.[15]

Such projections rely on understanding the role of temperature and humidity in heat-related health outcomes. Human physiological studies using climate chambers and energy balance modeling have found that humidity strongly influences human heat strain.[16–18] However, population-scale studies of environmental drivers for mortality report strong associations with temperature but generally little or no association with humidity.[19] Resolving this disagreement is necessary to accurately assess health risks today and in a warmer future (Figure 1).

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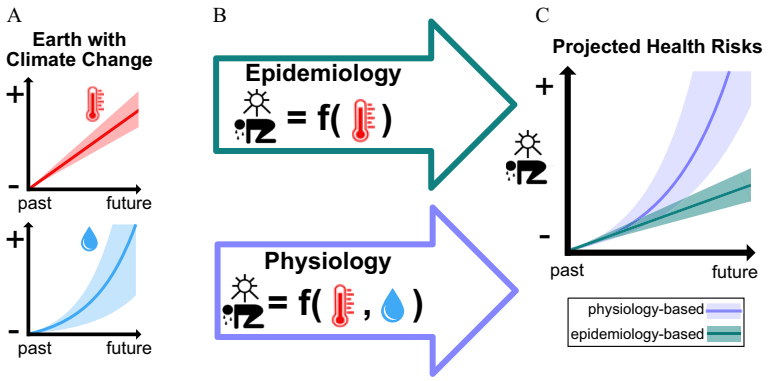

Figure 1. Flowchart illustrating the central problems proposed in this commentary. (A) With increasing atmospheric greenhouse gasses, temperature (top, red projection) and humidity (moisture in the atmosphere; bottom, blue projection) increase, with nonlinear, growing increases for humidity.[20] (B) According to epidemiologists (darker outline, turquoise arrow), heat-related health outcomes, such as excess all-cause mortality including cardiovascular collapse, should primarily follow daily temperature exposures[19]; but according to physiologists (lighter outline, purple arrow), heat strain and stroke should follow shorter-duration exposures to both temperature and humidity (among other variables).[21,22] (C) We propose that the physiologists' perspective with a strong role for humidity would result in faster increases in adverse heat–health outcomes with warming (lighter, purple projection) compared with the epidemiologists' perspective (darker, turquoise projection). Note that this is an illustration and does not incorporate actual data—for example, the light colored shading schematically illustrates uncertainty in climate change projections. Hypothesized resolutions to this discrepancy are summarized in Tables 2 and 3.

The goal of this commentary is to highlight proposed methodological and mechanistic reasons for the apparent lack of influence of humidity on heat-related health outcomes in the epidemiology literature. We present the problems posed by divergent conclusions of epidemiological and physiological studies regarding humidity, and suggest potential directions forward. We focus on temperature and humidity and, for the sake of brevity, do not discuss other environmental variables that influence human energy balance, such as radiation and wind speed. We point the interested reader to the following cited articles for information regarding these additional variables.[23–26] As in Davis et al.,[27] we examine the impacts of temperature and humidity on noncommunicable diseases, rather than infectious disease. Furthermore, we focus on nonoccupational heat–health relationships (such as the sedentary elderly dying prematurely in the heat); issues pertaining to heat-related mortality in outdoor workers require considerations of exertional heat stroke not directly addressed here.[28–30] We first present background information about humidity and its projected changes, before exploring the physiological vs. epidemiological findings regarding humidity and possible reasons for their divergence. We end by highlighting the importance of reconciling this debate for climate adaptation and presenting a summary and conclusion.

Understanding Humidity: Scales and Definitions

Many different expressions exist for the moisture content of air, developed in large part to aid understanding of meteorological and climatological processes. In studying heat–health outcomes, selection of the appropriate humidity measure is essential, given that some variables exhibit inverse correlations to temperature (e.g., RH) that can prevent a clear separation of the roles of humidity and temperature. Davis et al.[27] analyzed the humidity variable(s) used in 260 health-related publications from 2013 to 2016 and found that RH was used the majority (65%) of the time, and absolute or specific humidity only 5.3% and 1.5% of the time, respectively. The common and often inappropriate use of RH emphasizes the need for greater understanding of humidity measures among health researchers.

Table 1 defines humidity metrics for health studies and provides guidance for when each metric is appropriate. According to Davis et al.,[27,p114] the choice of humidity metric should be guided by "how humidity is fundamentally related to the process or condition of interest." In heat and health work, the skin-to-air water vapor gradient determines the drive for sweat evaporation[45] (see the "Physiological Study of Humidity in Heat Stress and Strain" section); thus, for health assessment, water vapor mass–based variables—including specific humidity, absolute humidity, mixing ratio, dew point temperature, and vapor pressure—are the most relevant expressions from the perspective of human thermoregulation.[27] Conversely, variables that have a thermal component and report humidity in relation to saturation—including RH, dew point depression, and saturation vapor pressure—exhibit strong diurnal and seasonal variations reflective of temperature and should thus be used sparingly and only when the etiology of the health condition justifies its use.[27] Although RH gives proximity to moisture saturation in the atmosphere, it is inversely proportional to temperature, is not physiologically relevant, and should be converted to a mass-based variable for most health studies.[27]

As shown in Table 1, there are a variety of metrics that combine temperature and humidity with relevance to the human heat experience. Given its close relation to human heat stress and clear meteorological interpretation, T_w is used throughout this commentary to illustrate a variety of points about physiological, epidemiological, and climate studies. Three important metrics from the physiology literature are *a*) the maximum evaporation rate achievable given a person's clothing and environment, often assuming the skin is saturated with moisture [evaporative capacity of the environment (E_{max})], *b*) the required evaporation rate for the human body to be in energy balance given metabolic heating [the evaporative requirement (E_{req})], and *c*) the actual evaporation of sweat from the skin (E).[21,46] Both E_{max} and E decrease as humidity rises and the skin-to-air humidity gradient decreases. In contrast, E_{req} depends on metabolic rate and factors other than humidity that influence human energy balance (e.g., temperature, radiation, windspeed).[21]

Moist Heat in a Changing Climate

Climate models robustly project that moist heat will intensify in response to rising greenhouse gas concentrations.[14,47–49] Here we briefly summarize the reasons for these trends.

First, climate simulations show enhanced warming of land surfaces compared with ocean surfaces, both in terms of mean and extreme temperatures. Simple theoretical arguments explain

Table 1. Humidity variables and temperature–humidity metrics commonly used in climate, epidemiological, and physiological research and recommendations for the types of study design for which they are appropriate.

Variable/metric	Definition/equation	Relevant notes/precautions for use in heat–health studies
Simple relative variables		
Relative humidity (RH) (%)	Ratio of actual vapor (e) in air to saturation vapor pressure (e_s) (i.e., proximity to saturation): $RH = e / e_s$.	Changes inversely with T_a, as the denominator varies with T_a. RH varies diurnally and seasonally; is not useful in epidemiological and environmental health studies.[27] Avoid use.
Dew point depression (DPD) (°C)	Indicates relative dryness of air; is the difference between T_a and T_d, (i.e., $DPD = T_a - T_d$).	Strongly influenced by T_a. Avoid use.
Saturation deficit	The amount by which the water vapor must be increased in a given environment to reach saturation in the air with unchanged temperature and pressure.[31]	Can be expressed in terms of a vapor pressure deficit, absolute humidity deficit, or RH deficit. Note: The physiological saturation deficit is the "difference between the amount of vapor actually present in the air (i.e., the absolute humidity) and amount that saturated air at body temperature contains (viz., ~45 gm per cubic m)" (Kendrew).[32,p189]
Simple mass-based variables—mostly conservative with T_a change; use for exposure–response studies		
Specific humidity (q) (g/kg)	Ratio of water vapor mass (g) per total moist air parcel mass (kg).	More conservative with T_a changes (i.e., less inherently diurnal).
Dew point temperature (T_d) (°C)	Temperature at which saturation occurs.	Assumes that the air's moisture content does not change as air is cooled to condensation.[27] Conservative with T_a changes (i.e., less inherently diurnal).
Mixing ratio (w)	Ratio of the mass of water vapor (g) per mass of dry air (kg).	Conservative with T_a changes (i.e., less inherently diurnal).
Vapor pressure (e) (mb)	Partial pressure of water in the atmosphere.	Conservative with T_a changes.
Absolute humidity (also called vapor density)	The density of water vapor, that is, the ratio of the mass of water vapor (g) present to the volume occupied by the mixture (m^3).	Not conservative with respect to adiabatic expansion or compression, thus, not commonly used by meteorologists.[33]
Common heat metrics/temperature–humidity indices—cannot independently examine humidity effect		
Apparent temperature (AT)/heat index (HI) (°C)	AT: adjustment to T_a based on the level of humidity to represent temperature perceived by a typical human; derived from human heat balance principles and sometimes incorporates solar radiation.[22,34] HI: A simple "hot weather" version of the AT to describe a "feels like" temperature.	HI: Over 21 approximations exist.[35] Ignores sunlight; assumes constant wind and walking speed.
Humidex (H_x) (°C)	A discomfort index derived by Anderson 1965.[36] $H_x = T_a + \frac{5}{9}\left[6.11 \times e^{5{,}417.7530\left(\frac{1}{273.16} - \frac{1}{273.15 + T_d}\right)} - 10\right]$.	No empirical basis; "… a public relations value in a weather service and conveys, in a simple way to the layman a general idea of atmospheric discomfort."[37] Used for heat wave warning and guidance, mostly in Canada.[38]
(Psychrometric) wet-bulb temperature (T_w) (°C)	Temperature at which air becomes saturated by evaporation at constant pressure. Usually measured based on moisture being evaporated from the surface of a wet muslin sleeve into surrounding air. Drier air → more evaporation → more cooling → lower T_w.	When T_w approaches skin temperature (~35°C), all heat loss avenues are eliminated, thus net heat dissipation is zero.[14] Sensitive to changing T_a.
Universal Thermal Climate Index (UTCI) (°C)	Physiologically-based thermal stress scale created to adequately reflect average human physiological perception to the outdoor thermal environment.[18,39,40] Inputs are T_a, windspeed, vapor pressure (e), and mean radiant temperature.	"Representative activity for average person": 2.3 METs (135 W/m^2); 1.1 m/s (walking); "average" person (74 kg, 1.9 m^2). These parameters are non-modifiable in the currently available UTCI version.

Table 1. (*Continued.*)

Variable/metric	Definition/equation	Relevant notes/precautions for use in heat–health studies
Wet-bulb globe temperature (WBGT)	Indicator of heat stress on the active/working body in direct sunlight. $WBGT = 0.7T_{nwb} + 0.2T_g + 0.1T_a$, where T_{nwb} is non-aspirated, "natural" wet-bulb, T_g is black globe temperature, T_a is shaded dry bulb (air) temperature.	Intended for use in active populations outdoors; developed for military from studies in hot, humid environments. Studies, particularly climate projections, often neglect the T_g value, which is not its intended use.[41]
Simplified WBGT approximation (sWBGT)	$sWBGT = 0.567T_a + 0.393e + 3.94$. Does not account for variations in the intensity of radiation or wind speed; assumes a moderately high radiation level in light wind conditions.[42]	May overestimate thermal stress in windy and cloudy conditions or underestimate thermal stress in dry, sunny, hot conditions when required sweat rates are high due to activity levels.
Predicted heat strain	A rational index based on energy balance principles quantifying heat stress and strain.[43,44]	Physiologically relevant.
Physiologic-based humidity indicators[25,45]		
Maximum evaporation (E_{max}) (W or W/m^2)	Maximal evaporation rate possible in the given environment and clothing worn. Lower E_{max} in a more humid environment and with a higher evaporative resistance of clothing: hence, more stressful.	Directly physiologically relevant. Note: In a dry environment, E_{max} is limited by the ability to sweat.
Required evaporation (E_{req}) (W or W/m^2)	Required evaporation to achieve thermal balance. E_{req} = metabolic rate − non-evaporative heat losses.	Directly physiologically relevant.
Total/actual evaporation of sweat (E) (W or W/m^2)	Total amount of heat loss via evaporation of sweat from the body. If E_{max} is reached, then $E = E_{max}$.	Directly physiologically relevant.

Note: See McGregor and Vanos[23] and Havenith and Fiala[18] for further thermal comfort and heat stress indices. e, vapor pressure; E, evaporation; MET, metabolic equivalent of task; q, specific humidity; T_a, air temperature; T_d, dew point; T_g, globe temperature; T_{nwb}, natural wet-bulb temperature; UTCI, University Thermal Climate Index; w, mixing ratio.

the faster warming of land surfaces in the tropics and subtropics,[50,51] but this phenomenon is less well understood at higher latitudes. At the same time, warmer air can hold more water vapor, following the Clausius–Clapeyron relation, which states that at fixed RH, specific humidity increases at a rate of $\sim$6–7%/°C.[12,52] Since the earliest climate model simulations, it has been known that RH stays roughly constant over ocean surfaces when carbon dioxide (CO_2) concentrations are increased,[53] leading to large increases in specific humidity. This extra water vapor gets transported over land regions, which can see increases in specific humidity, although these increases tend to be smaller than over oceans because some of the moisture rains out as it is transported.

Although specific humidity changes over land are generally slower than the 6–7%/°C Clausius–Clapeyron scaling, they are much larger than the fractional changes in temperature.[49] The result is that increases in specific humidity dominate changes in moist heat in all but the coldest, driest climates,[49] and projecting changes in moist heat largely comes down to projecting changes in specific humidity. The driving role for humidity in recent and projected increases in moist heat is stronger in the tropics compared with the relatively drier extratropics.[49] Figure 2 illustrates this by plotting global changes in the annual 98th percentile of equivalent potential temperature (which scales with T_w) and corresponding changes in near-surface air temperature (T_a) and specific humidity, following a transient warming scenario; methods follow Lutsko,[49] which analyzed output from 14 Coupled Model Intercomparison Project Phase 6 (CMIP6) models.

Physiological Study of Humidity in Heat Stress and Strain

Physiological studies have concluded that increasing humidity along with increasing temperature should pose significant risks for human health.[14,45] It is well established in the physiology and biophysics literature that ambient humidity plays a critical role in whether human core temperature can be maintained within safe limits. To prevent accumulation of excess heat energy inside the body and the elevated risk of hyperthermia and heat stroke, the bloodstream must transport metabolically generated heat to the skin surface. To do so, the skin's vasculature dilates (cutaneous vasodilation) and then dissipates this heat to the surrounding environment.[30] Heat transfer away from the body can occur via dry transfer processes (convection and radiation), but the magnitude of these losses diminishes as T_a increases. Above $\sim$35°C T_a, the only remaining avenue for heat dissipation is evaporation, almost exclusively from sweat secreted by the 2–4 million eccrine glands distributed across the human skin.[26] Each gram of sweat can liberate up to 2,427 J of latent heat if completely evaporated from the skin.[54]As ambient humidity rises, the proportion of secreted sweat that evaporates (i.e., sweating efficiency) decreases, with the remainder either sitting on the skin or dripping off the body without contributing to latent cooling (i.e., inefficient sweating).[55] Because less heat is lost for a given sweat rate at high humidity, core temperature continues to rise until enough sweat is secreted to elicit the required rate of evaporation to attain thermal equilibrium. Heat stroke risk becomes substantially elevated when evaporative heat balance requirements necessitate a sweat rate that exceeds the physiological limit.[30]

The importance of mass-based measures of humidity (e.g., specific or absolute humidity) vs. humidity variables with a thermal component (e.g., RH, saturation vapor pressure, saturation deficit) for human thermoregulation can be understood as follows. In keeping with bulk formula for latent heat flux, evaporation from a moist surface (such as the skin) is driven by the difference in saturation specific humidity (q_s) and the specific humidity of the overlying air. q_s is in turn dictated by the temperature of that surface. For the human body, the relevant surface

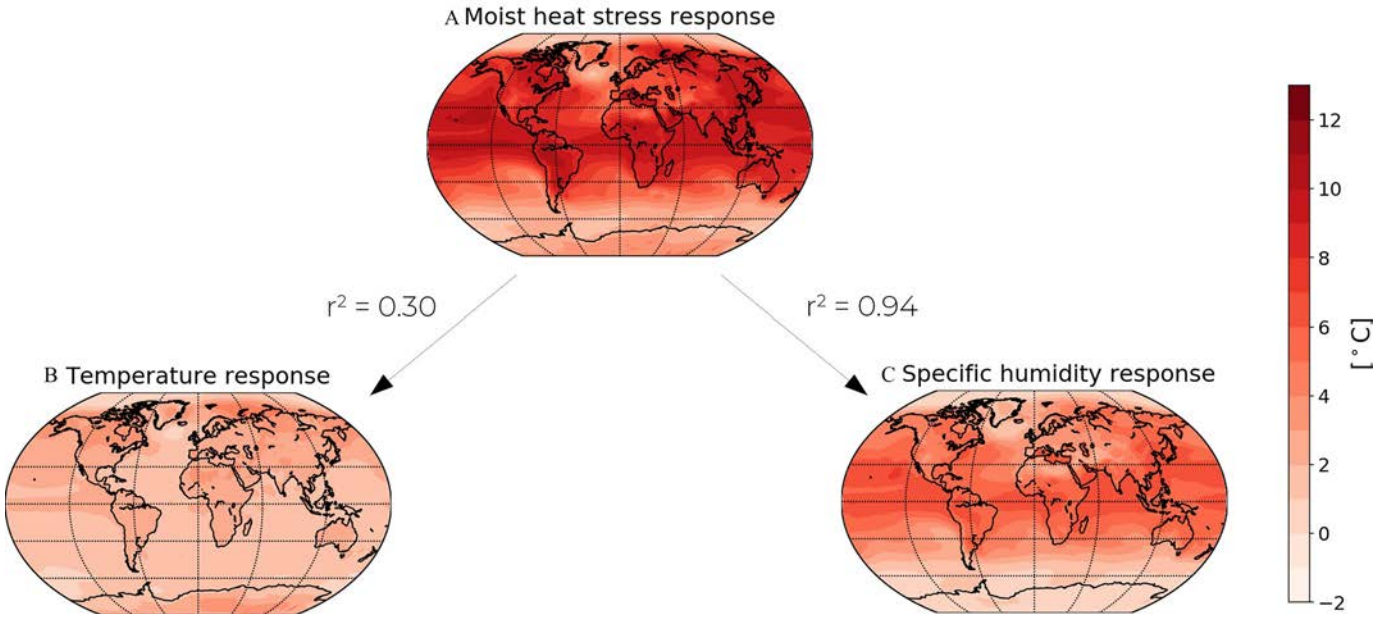

Figure 2. Changes in moist heat stress metrics following global warming are strongly correlated with changes in specific humidity. (A) Multi-model mean change in the 98th percentile of daily equivalent potential temperature (which scales with wet-bulb temperature). (B) Multi-model mean change in air temperature, conditioned on the 98th percentile of daily equivalent potential temperature. (C) Multi-model mean change in specific humidity (converted to degrees Celsius), conditioned on the 98th percentile of equivalent potential temperature. Output from transient warming simulations (CO_2 concentrations are increased at 1%/y) of 14 CMIP6 models is analyzed; each panel plots the annual mean comparing years 71–80 and years 1–10 of near-surface (2-m) atmospheric quantities. This figure uses data from Lutsko,[49] and methods and particular model simulations used are described in detail in that paper. Note: CMIP6, Coupled Model Intercomparison Project Phase 6; CO_2, carbon dioxide.

temperature is skin temperature (T_{sk}), and therefore the driving gradient for evaporation and sweat efficiency is $q_s(T_{sk}) - q_{air}$, bearing in mind that total evaporation from the human body is also influenced by the physiologically modified skin wettedness (proportion of skin covered in sweat).[18,55] A variety of physiological mechanisms, including blood flow and sweating, help the human body keep T_{sk} somewhat constant at 35°C for fully vasodilated skin, or at least varying much less than surrounding T_a.[56] T_{sk} is maintained to ensure an internal body temperature gradient that allows the flow of metabolic heat outward from the human body; when at rest, the body's core temperature is typically 37°C. Given that $q_s(T_{sk})$ is relatively constant, variations in evaporative potential from the human body, and in turn human heat strain and stress, are influenced primarily by the q_{air}. This is true in most climates, excluding very dry conditions when the skin surface is not saturated.[55,57] A further caveat is that clothing, various human body attributes, and wind speed can also influence the rate of cooling from evaporation. In contrast to mass-based metrics, metrics with a thermal component, such as RH, reflect variations in the temperature and water content of the air and do not cleanly capture drivers of evaporation from the skin surface.

The same T_a with different levels of ambient humidity (when controlling for wind and radiation) can elicit different levels of physiological heat strain. For example, young healthy men exercising for 45 min at a fixed heat production of 450 W in 37°C T_a demonstrated a 0.3–0.5°C rise in core (esophageal) temperature accompanied by sweat losses of 400–500 mL with 12 g/kg q (30% RH),[58] but core temperature increases and sweat losses were 0.9–1.1°C and 700–800 mL, respectively, when q was 24 g/kg (60% RH).[58]

The most widely used climate projections for estimating future human survivability with global warming are based on these physiological impacts of humidity. The premise of the 35°C T_w survival threshold is that both the dry heat loss and the humidity gradient between skin and air are eliminated, resulting in no cooling via convection or sweat evaporation.[14] Under these assumptions, the human body becomes an adiabatic system whereby all internally generated heat remains inside the body, leading to catastrophic overheating. Importantly, a fixed T_w can be attained with a range of different T_as.[17] For example, 35°C T_w is equivalent to 35°C T_a with 36 g/kg q (100% RH), 40°C T_a with 34 g/kg q (71% RH), and 50°C T_a with 31 g/kg q (36% RH).

Epidemiological Study of Humidity in Heat–Health Outcomes

The key role of humidity on human heat stress in physiological studies seems at odds with weak relationships between humidity and heat-related health outcomes in epidemiological studies. Although most epidemiological studies investigating the role of heat on noncommunicable health outcomes (e.g., premature mortality or emergency department visits) have relied on temperature metrics (e.g., daily maximum temperature or diurnal temperature range) as the primary exposure,[59,60] some studies also considered humidity.[19,61–63] These studies often employed differing humidity metrics (see Davis et al.[27] and Table 1) or at times combined temperature–humidity metrics [e.g., humidex, apparent temperature (AT)]. Such studies also employed various analytical approaches with distinct interpretations and implications. Typically, in epidemiological studies, variations in daily health outcomes and weather data are compared, contrasting with the shorter temporal scales explored in physiological experiments. These contrasting timescales imply consideration of different health outcomes. For example, increasing dehydration and body temperature can lead to direct impacts, such as heat exhaustion or heatstroke, over shorter time periods (hours to days) and/or indirect impacts by exacerbating underlying comorbidities, such as cardiovascular or renal diseases, that often show multiday lagged responses.[64]

Conclusions are mixed among epidemiological studies that consider humidity. One study found that high RH levels were

associated with higher cardiorespiratory hospital admissions in New York,[61] but the majority of studies diverge from expectations based on the physiology literature. For example, a recent multicountry, multicity study by Armstrong et al.[19] concluded that overall mortality was slightly reduced compared with typical mortality levels following days with higher RH, and these findings were largely consistent with mass-based measures of humidity (dew point temperature and specific humidity). Studies in Australia[62] and in the United States[63] also concluded that RH had little to no additional effect on temperature–health relationships. Such counterintuitive conclusions may be explained by the complex and heterogeneous relationship between humidity and temperature in heat and health studies, as well as critical underlying analytical and data considerations, as described below.

We distinguish three main approaches to incorporating humidity variables in heat–health epidemiological studies that may cause counterintuitive findings (i.e., little or no impact of humidity on heat–health outcomes) when compared with physiological knowledge of thermoregulation in the heat, including: *a*) the use of a composite index, *b*) considering humidity as a confounder between temperature metrics and health outcomes, and *c*) the use of an interaction term to represent potential effect modification or interaction between temperature and humidity. We also describe relevant limitations of existing epidemiological and weather data.

Composite Biometeorological Indices

Many epidemiological studies have relied on composite biometeorological indices, such as the AT, heat index, or humidex (see Table 1). Such approaches offer the advantage of simultaneously considering temperature and humidity in a single index that can then be used as the main exposure of interest. With this approach, the epidemiological interpretation can be ambiguous, given that increases in the index can be driven by temperature, humidity, or both [or other factors, as in the University Thermal Climate Index (UTCI)]. In addition, certain indices (e.g., the humidex) were developed based on human (dis)comfort,[65] which is based on an average person's subjective feelings of comfort; thus, relationships may be skewed and not driven by physiological heat strain,[66] such as heat strain during outdoor work.[67,68] The units applied to the index may also be confusing [e.g., for UTCI, Web Bulb Globe Temperature (WBGT), and humidex, "°C" is added to the output value, yet the output is not a true temperature]. Although composite temperature–humidity indices can be useful when designing heat warning systems, the interpretation ambiguity may lead to biased or misleading estimates of associated risks and thus impact the effectiveness of warning systems.[69]

For example, consider a hypothetical analysis of daily time series of health and weather data, examining a geographical context in which the correlation between temperature and humidity has a strong seasonality with a high correlation in June but low correlation during the rest of the summer. In this setting, the relative risk associated with a given value of T_w (or any other composite index) compared with a baseline value (or "benign" weather day) may be low overall because the average risk is driven by the majority of days where humidity levels are low. Such an average effect may be particularly high early in the summer season and not be detected.

If using composite indices, we encourage exploration of effect heterogeneity (while considering the presence of nonlinear relationships with health outcomes) according to the composition of the index or the season. Given the documented physiological impacts of high humidity (see the "Physiological Study of Humidity in Heat Stress and Strain" section), it would be interesting to explore the heterogeneous effects of composite indices on more specific causes of mortality or morbidity (e.g., by exploring cardiovascular subtypes, such as hypertensive disease or cerebrovascular diseases). Overall, we recommend using alternative methods (see below) that separate out the effects of temperature and humidity, even if some caveats are needed.

Humidity as a Confounder

Many studies consider, implicitly or explicitly, humidity as a confounder in the relationship between temperature metrics and health outcomes.[70–72] Humidity is typically adjusted for in such analyses. Consider the following multivariable linear model where Y represents a given health outcome rate (e.g., daily rates of nonaccidental mortality per 100,000), t is daily mean temperature, h is daily absolute humidity, and c' is a vector of confounders (including calendar variables):

$$\mathrm{E}[Y|t,h,c'] = \theta_0 + \theta_1 t + \theta_2 h + \theta_3 c'.$$

Assuming no interaction on the additive scale and a linear dose–response effect for simplicity, θ_0 represents the intercept, and θ_1 the effect of t conditioning on both h and c'. The coefficients θ_2 and θ_3 (under some assumptions[73]) can be interpreted as the controlled direct effect, although interpreting such coefficients can be misleading.[74,75]

A first challenge is that by conditioning (i.e., adjusting) on humidity levels, we deliberately remove the influence of humidity on both temperature and the health outcome. This corresponds to a hypothetical, counterfactual world in which humidity and temperature are independent regarding health impacts, which is not the case and thus affects the interpretation of temperature's impact on health. Such results should be interpreted with caution.

A second challenge is that when temperature and humidity are highly correlated (or when such correlation is time dependent or nonlinear), including both variables in a single multivariable regression model can lead to multicollinearity issues. Such multicollinearity can lead to ambiguously estimated coefficients that may arbitrarily vary because of differential proportions of the variance explained or if one of the two variables has more missing data. If such correlation between temperature and humidity varies greatly with time, this multicollinearity may be exacerbated by the inclusion of calendar variables to adjust for long-term or seasonal trends. Multicollinearity also reduces the precision of the estimated coefficients and may lead to erroneous conclusions that humidity (or temperature) does not have an effect. This is particularly problematic when null hypothesis significance testing is used (e.g., using a p-value threshold)—a misleading practice for which there are practical alternatives.[76,77] In summary, including humidity as a confounder can omit important mechanisms linking temperature and humidity in producing negative health outcomes, especially when these variables are highly correlated.

Humidity as an Effect (Measure) Modifier

Finally, some studies consider modification of humidity on temperature's health effect, for instance by including an interaction term between temperature and humidity. In etiological studies (i.e., when the goal is to infer causal effects as opposed to descriptive and predictive studies), there is a key distinction between interaction and effect modification.[78] Although analytical approaches to estimate interaction or effect modification (i.e., interaction terms, stratification) are similar, the policy implications may differ if the aim is to address exposure to temperature, humidity, or both at the same time.

Interaction and effect modification are scale dependent, which means that they can occur on absolute and multiplicative scales.[79] Therefore, when using a multiplicative model, such as Poisson or

conditional logistic models, product terms represent deviations from multiplicative joint effects and not deviations from additivity. The choice of such a scale is important for the interpretation of interaction or effect modification and policy implications.[80,81] The absolute scale may be more suitable for inferring recommendations for targeted policies (i.e., to optimize the benefits of early warning systems). Multiple tools to quantify interaction or effect modification on the additive scale have been proposed.[82]

In a recent example, Armstrong et al.[19] explored the interaction between daily temperature and RH assuming no cross-lag interactions and no nonlinear interaction. In their study, they decided not to include such an interaction term in their final model (from which they inferred the main effect of heat on mortality) based on Akaike's information criterion (AIC). AIC is intrinsically a metric for predictive purposes that may not be relevant for etiological research questions, such as effect modification questions or to decide whether to include a variable.[83,84] When including an interaction term or stratifying between humidity and temperature, it is crucial to consider the main question of interest (etiological, descriptive, or predictive), the scale of interest, whether interaction or effect modification is of interest, and the potential policy implications.

Limitations of Epidemiological and Weather Data

Data limitations may also hinder accurate epidemiological assessment of humidity's role in heat–health outcomes. Epidemiological data and station-based weather data are not available at every location where people live. Governments and researchers in higher-income countries more often have the capacity to collect and maintain such data sources.[85,86] When data records exist in low- and middle-income countries (LMICs), they often have relatively short duration, are reported weekly or monthly, or are not publicly digitized, limiting their usability.[86] This issue has been gravely highlighted for sub-Saharan Africa, where weather data shows striking historical heat waves for which there are no impact estimates.[87]

These data limitations pose challenges for disentangling the role of humidity. Figure 3 demonstrates that the most comprehensive analyses of temperature and humidity associations with mortality lack data from many LMICs and tropical locations.[10] This omission may misrepresent humidity's role in heat stress. The tropics and subtropics experience some of the highest levels of humidity and combinations of temperature and humidity.[15] As a result of higher T_ws in the tropics, locations where humidity has the greatest impact on heat stress might not be included in epidemiological studies. Figure 3A represents this by using daily resolution T_ws from the station-based HadISD data set[88] and by plotting the average of annual maxima of that data for each station, overlaid with locations of epidemiological data from a large, global data set.[10] The predominately extratropical regions where mortality data are available also tend to have positive correlations between temperature and humidity, making it difficult to discriminate between the roles of temperature and humidity using epidemiological time-series analyses. Figure 3B illustrates this by plotting the correlations (Pearson r) between near-surface T_a and specific humidity anomalies from HadISD,[88] overlaid with locations of the same epidemiological data as Figure 3A[10]; anomalies are calculated by subtracting the corresponding daily resolution seasonal cycles from temperature and specific humidity at each station. Finally, there is a vast literature showing that lower-income populations are more vulnerable to heat.[86] Thus, the relative lack of LMIC data may lead to an underestimation of aversive effects of heat and possibly humidity.

Another challenge pertains to the temporal resolution of data. Mortality and morbidity data usually have daily resolution and are often compared in epidemiological studies to daily mean, maximum, or minimum temperature, and daily average humidity. These daily variables negate the extent to which temperature and humidity are correlated in real-time when human heat stress occurs.[27] For example, heatstroke occurring over mere hours at midday would not be well-captured by daily averages. Moreover, relatively fast meteorological processes, in particular atmospheric convection, effectively cap the combined value of temperature and humidity that can be reached, especially in hot and humid regions such as the tropics.[48,89] These temporal influences may reduce the likelihood of high values of temperature and humidity being correlated on diurnal timescales and may also reduce the discernible role of humidity in the epidemiological study of heat–health outcomes.

Limited spatial resolution of weather data may also misrepresent the true personal heat exposure.[90–92] Station-based weather data are often from airports on the outskirts of cities, which may not capture intra-urban heat effects and other heterogeneities associated with land surfaces. In addition, some individuals, particularly in higher-income settings, spend much of their time inside, where air conditioning, shading, and/or fans create different thermal environments from that recorded by distant weather stations used in epidemiological models. This disconnect between the recorded weather and personal heat exposure may also bias the modeled effect of humidity. Overall, a general lack of data and poor spatiotemporal resolution may result in limited knowledge of heat–health relationships in the places most affected. We emphasize that although physiological studies are conducted at the individual level, epidemiological studies are based on ecological inference (e.g., the daily change in mortality counts in relation to daily changes in weather metrics). Such discrepancy in the etiological scale of interest may fundamentally contribute to the discrepancies we describe.

Recommendations for Epidemiological Studies

In summary, we make the following recommendations for epidemiological studies examining health risks from heat:

1. When choosing a humidity variable, mass-based measures (e.g., specific or absolute humidity) are recommended and RH is not. Mass-based measures directly affect sweat evaporation, whereas RH exhibits temperature-driven diurnal cycles that do not reflect variations in heat strain (see the "Understanding Humidity: Scales and Definitions" section).
2. Avoid the use of humidity only as a confounder (e.g., via adjustment in multivariable models) and instead consider effect modification (and explore both additive and multiplicative scales).
3. Statistical methods need to be appropriate for the goals of the study (i.e., descriptive, predictive, or etiological); in particular, AIC is not suitable for addressing etiological research questions.[83,84]
4. When using composite indices, we recommend exploring the potential effect heterogeneity of a given composite index on a health outcome according to the composition of the index (i.e., driven by temperature or humidity) or the seasonality.
5. Additional data should be sought out from LMICs, the very hot and humid tropics, and locations where temperature and humidity are less correlated.
6. Given the present mismatch in scales between epidemiological and physiological studies, epidemiological studies should seek to collect additional individual-level health information to be analyzed alongside meteorological metrics.
7. Following the physiological considerations discussed in the next section, epidemiological studies should explore whether the effects of humidity vary depending on the cause of death or hospitalization.

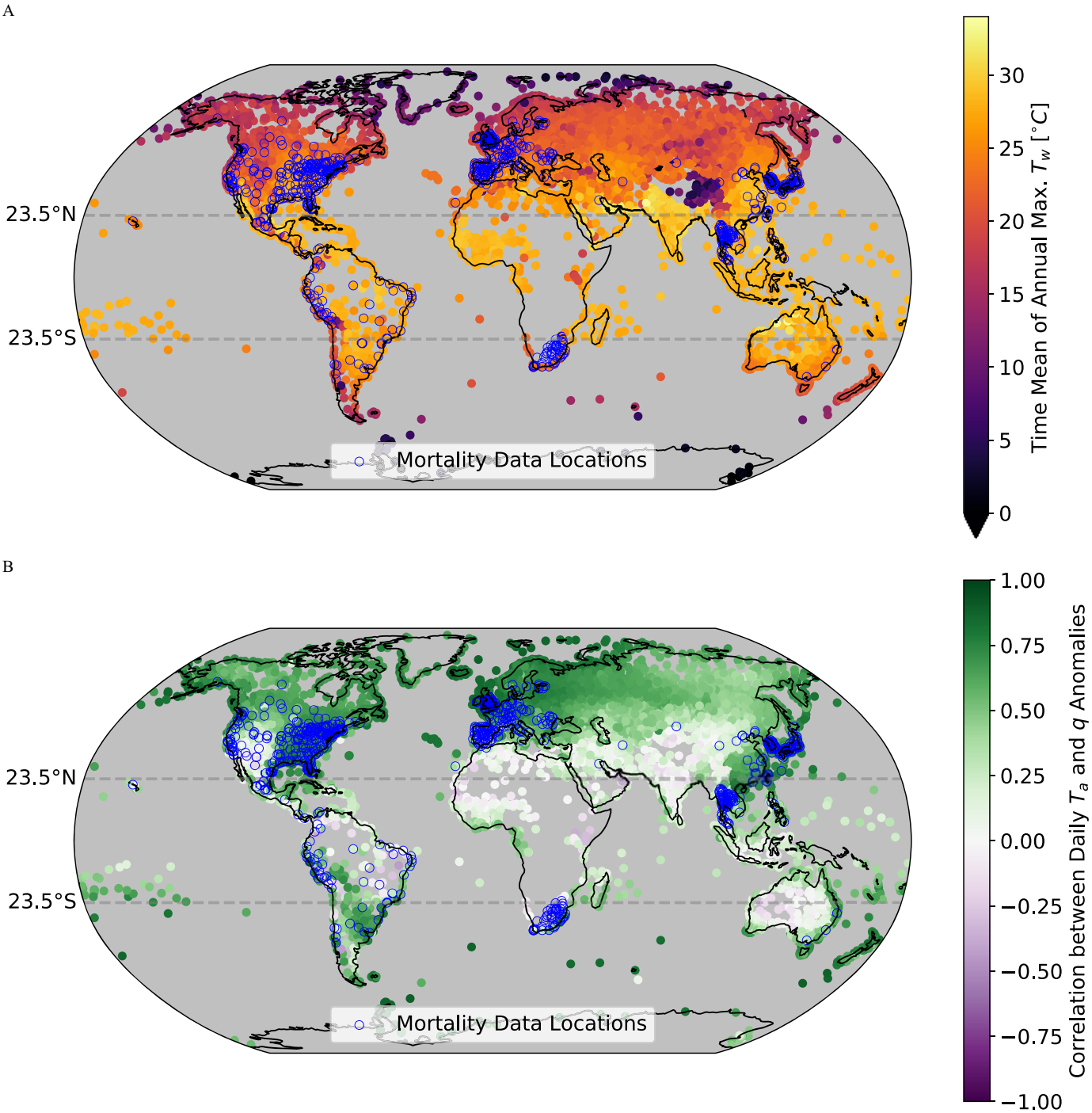

Figure 3. Historical temperature and humidity conditions compared to locations of available mortality data. (A) Time mean of annual maximum wet-bulb temperature from stations in HadISD (version 3.1.2.202104p),[88] overlaid with locations of mortality data (blue circles) used in a recent study attributing heat-related mortality from climate change,[10] (B) Same data sources as (A)[10,88] but instead plotting correlation between daily anomalies of 2-m air temperature and specific humidity. Anomalies are calculated as deviations from the daily resolution seasonal cycle for each station. In both (A and B), gray dashed lines demarcate latitude bounds of the tropics. Note: max, maximum; q, specific humidity; T_a, air temperature; T_d, dew point; T_w, wet-bulb temperature.

Possible Physiological Reasons for Epidemiological Results

Potential physiological reasons for the disconnect on humidity's role in heat stress between physiological and epidemiological studies include the following:

1. **Sweating impairments in heat-vulnerable people.** Progressive impairments in the human sweating response occur naturally with aging owing to reduced peripheral sensitivity to acetylcholine, the neurotransmitter predominantly responsible for eccrine sweating, and atrophy of the sweat glands.[93] Above the age of 60 y, thermoregulatory sweating can decline by up to 25%, although these impairments can be delayed by regular aerobic training.[94] Anticholinergic medications, such as many antidepressants and psychotropic medicines, also theoretically attenuate sweating,[95] as do other medications that alter

peripheral vasodilation, such as anti-adrenergics and beta-blockers.[96] Advanced age, poor health, and taking prescription medications are leading risk factors for heat-related morbidity and mortality.[2] Those who get sick or die during heat waves likely have reduced sweating capacity; thus, it is possible that these individuals are consequently less sensitive to impairments of heat dissipation from elevated humidity (i.e., the effects of high humidity are less).

2. **Environmental thresholds at which differences in humidity impact thermoregulatory strain are rarely attained.** Humidity's impact on heat strain is most evident when the E_{req} to maintain balance between net heat dissipation to the environment and internal metabolic heat production exceeds $\sim 50\%$ of the E_{max}. Above this threshold, sweat begins to drip off the body or remain unevaporated on the skin.[25] Heat loss from the skin for a fixed sweat rate is subsequently reduced, and a person must sweat more to achieve requisite heat loss, which is physiologically generated by higher body temperature. Below this threshold, differences in the E_{req}/E_{max} ratio only have a modest effect on the proportion of sweat that evaporates and, therefore, on physiological heat strain.[25]

   For a given person, E_{req} predominantly increases with metabolic rate (i.e., activity level) and ambient temperature, whereas E_{max} is an inverse function of humidity. With the exception of exertional heat illness/stroke, extreme heat victims are usually minimally active,[2] so ambient temperature and/or humidity must be very high for E_{req} to approach the 50% E_{max} threshold at which a higher humidity is more likely to impair sweat evaporation. Figure 4 illustrates the threshold temperature–humidity combinations for a sedentary man and woman at which further increases in humidity would theoretically increase thermoregulatory strain, compared with historical peak heat wave conditions. Also shown in Figure 4, major heat waves tend to fall close to these thresholds, suggesting that environmental conditions in which humidity can impact the thermoregulatory strain of sedentary people could be relatively rare. The threshold temperature–humidity levels of Figure 4 are based on biophysical modeling,[25,97] with different parameter values from empirical studies for men vs. women.[98] The weather data were prepared for a prior publication, Morris et al.[99]; it includes the hottest 1-h T_a and accompanying humidity from 2007 to 2019 for 108 airport weather stations from global cities. Cities were selected based on their large populations and varying geographical locations across all six habitable continents to represent a wide range of hot weather conditions. Data underlying this figure can be found in Excel Tables S1 and S2.

3. **Most heat-related deaths and hospitalizations may be due to causes unaffected by humidity.** High humidity is argued to pose health risks in extreme heat by reducing sweat evaporation, thus elevating internal body heat storage and core temperatures. However, most heat-related deaths and hospitalizations are not due to conditions directly associated with critically high core temperatures, such as heatstroke. Rather, the leading causes of death are conditions such as cardiovascular disease.[100] There is little evidence that these diseases directly impair thermoregulation; but there is a strong rationale for heat exposure, somewhat independent of humidity, aggravating these disease states such that catastrophic failure of a major physiological system is more likely.[101] Using cardiovascular disease as an example, exposure to high ambient temperatures initially elicits cutaneous vasodilation triggered by rising skin temperature.[102] As more blood flows toward the skin, cardiac output is elevated to maintain central blood pressure, primarily by increasing heart rate.[103,104] This greater cardiovascular work requires more oxygen to be delivered to cardiac myocytes by the blood flowing through the coronary arteries.[105] Indeed, sedentary exposure to simulated heat waves increased the heart rate and rate–pressure product (an index of the heart's oxygen requirements) by up to $\sim 50\%$.[106] Underlying infirmities, such as atherosclerosis of the heart's major blood vessels, then create clear mechanistic pathways for heat exposure to cause potentially deadly cardiovascular events. Sweat evaporation moderates skin temperature, and therefore high humidity could exacerbate the trigger for this cascade of physiological events, but T_a remains the primary environmental driver of skin temperature changes in the heat.[107]

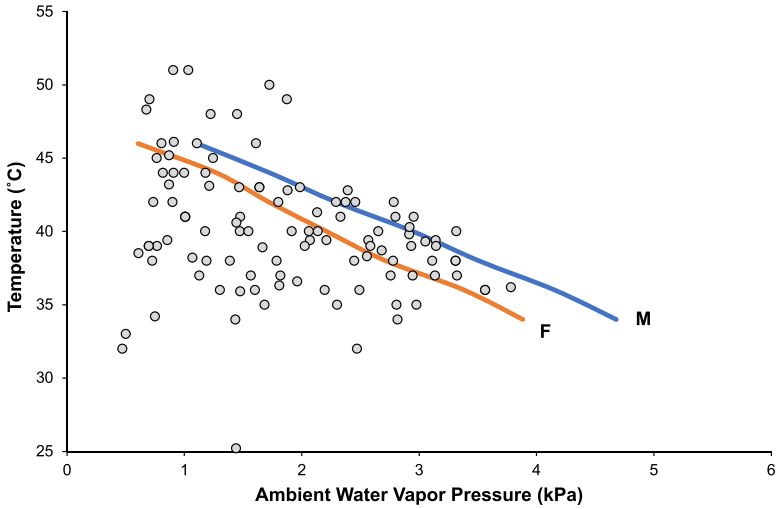

Figure 4. Environmental constraints on sweating efficiency compared to historical extreme heat data. Threshold combinations of temperature and humidity for a sedentary man (blue) and woman (orange) at which further increases in humidity will theoretically increase thermoregulatory strain because of reductions in sweating efficiency (i.e., the proportion of sweat that evaporates[26]). For context, the hottest single 1-h temperature and accompanying ambient water vapor pressure from the airport weather stations of 108 global cities across a 13-y period (1 January 2007 to 31 December 2019) are plotted (solid black circles). Five cities with the largest populations were selected in specific countries across all six habitable continents (North America, Europe, Asia, South America, Oceania, and Africa) to represent a wide range of hot weather conditions. Data underlying this figure can be found in Excel Tables S1 and S2. Note: F, female; M, male.

Table 2. Hypothesized causes of bias in epidemiological studies that may lead to underestimation of the role of humidity in heat–health outcomes.

Possible causes of bias in epidemiological studies	Location in text (section)
Analytical	
Equivalent values of composite biometeorological metrics (e.g., T_w) can lead to heterogeneous health effects depending on underlying temperature and humidity values.	Composite Biometeorological Indices
Considering humidity as a confounder effectively ignores the combined role of temperature and humidity on heat–health outcomes.	Humidity as a Confounder
High correlation of temperature and humidity may make it difficult to distinguish their effects in time-series analysis with traditional analytical tools.	Humidity as a Confounder
The use of metrics such as the Akaike information criterion (AIC) to guide the inclusion (or exclusion) of humidity in a model used for etiological purposes may be inappropriate.	Humidity as an Effect (Measure) Modifier
Data-related	
There are limited epidemiological and weather data from LMICs and the tropics, where people are likely most vulnerable and moist heat tends to be high.	Limitations of Epidemiological and Weather Data
Temperature and humidity data are at daily resolutions that negate complexities of the diurnal cycle, such as atmospheric convective processes making high temperature and humidity unlikely to occur simultaneously or for long duration.	Limitations of Epidemiological and Weather Data
Weather station data misrepresents individuals' true experiences with heat due to indoor vs. outdoor temperatures, and land surface heterogeneities, such as urban thermal island effects.	Limitations of Epidemiological and Weather Data

4. **Dehydration-related reductions in sweating.** Dehydration is another major cause of heat-related death.[100] Hydration status changes owing to a mismatch between fluid losses and intake. In the heat, fluids are mainly lost from sweating, yet fluid intake is poorly regulated by thirst alone, leading to a tendency toward voluntary dehydration.[108] Blood volume reductions from dehydration worsen cardiovascular strain in the heat. In addition, body fluid deficits exceeding $\sim 2\%$ of total body mass attenuate sweat production[109]—every 1% mass reduction was shown to increase core temperature by 0.15°C.[110] Although hydration status of the most vulnerable during heat extremes remains largely unknown, it is plausible that the prevalence and magnitude of dehydration limits sweating, thus reducing the potential for high humidity to affect evaporation and worsen heat strain.

Importance of Reconciling This Debate

1. **Projections of health risks with climate change.** Robustly projecting health risks of climate change serves many purposes. At a macro scale, such projections help quantify the overall costs of climate change and inform expectations of carbon mitigation benefits.[111] At a local scale, health projections help inform interventions to anticipate, adapt to, and reduce adverse impacts of climate change.[112] Rigorously understanding the role of humidity in heat–health outcomes is necessary to be confident in these projections. Temperature and humidity (by which we mean mass-based measures, not RH) both increase with global warming, but details of how they change are different and consequential for combined temperature–humidity extremes. Recent work demonstrates that humidity plays a key role in driving T_w extremes in the present climate and results in faster increases in T_w with global warming than changing temperature alone.[113,114] Thus, if humidity does not matter in heat–health outcomes, physiology-based projections (e.g., based on 35°C T_w thresholds[14]) likely overestimate future risks, but if humidity does matter, epidemiology-based projections[115] likely underestimate future risks (Figure 1). In addition, for any interval of warming, regional patterns of change in combined temperature–humidity metrics (e.g., T_w) will be dominated by humidity changes; Figure 2 shows that across state-of-the-art climate models, changes in moist heat extremes are higher in the tropics, a pattern highly correlated with change in specific humidity ($r^2 = 0.94$) and much less correlated with change in ambient temperature ($r^2 = 0.30$).[49] This suggests that projected heat–health outcomes across the world will substantially differ depending on the role of humidity, with potential equity implications given the large vulnerable populations in the tropics where moist heat changes are most exaggerated.[116]
2. **Heat–health adaptation strategies now and in the future.** Humidity's ambiguous role in heat-related health outcomes makes it difficult to optimize adaptation strategies, now and under future warming. At the individual level, the efficacy of personal cooling strategies may be affected by humidity.[117] For example, electric fans, which require a fraction of the energy and cost of air conditioning, are effective cooling devices in high humidity conditions, but at the same T_a (if T_a is greater than skin temperature) can prove detrimental under low humidity.[106] Reasons why humidity matters (or not) could also influence adaptation effectiveness. For example, if humidity does not play a large role in heat–health impacts because of sweating

Table 3. Hypothesized physiological reasons that could explain the limited role of humidity in heat–health outcomes.

Physiological reasoning for limited effect of humidity	Location in text (section)
Highly vulnerable people (elderly, medicated) have reduced sweating responses, so their mortality risk during heat waves may be less sensitive to humidity.	Sweating Impairments in Heat-Vulnerable People
Humidity only strongly affects human heat strain when the evaporative efficiency of sweat begins to decline substantially, which may rarely happen when people are sedentary, even at unusually high levels of temperature and humidity.	The Environmental Thresholds at Which Differences in Humidity Impact Thermoregulatory Strain Are Rarely Attained
Many common deaths or hospitalizations due to heat are from causes such as cardiovascular disease that are affected by temperature but presently have ambiguous mechanistic connections to high humidity.	Most Heat-Related Deaths and Hospitalizations May Be Due to Causes Unaffected by Humidity
During heat waves, dehydration risk increases, which at its extremes can reduce sweating capacity and plausibly limit individual sensitivity to humidity.	Dehydration-Related Reductions in Sweating

impairments in vulnerable populations (i.e., the elderly), then self-dousing (applying water to the skin) to increase heat transfer from the skin surface during heat waves could improve health outcomes. Self-dousing could also overcome the lessened efficacy of fans at low humidity observed in older populations in climate chamber experiments.[118]

An improved understanding of humidity's role would also influence city-level adaptation measures. At present, some heat wave early warning systems only consider temperature, whereas others consider both temperature and humidity, or composite metrics (e.g., AT).[119] If humidity matters for heat–health outcomes, and is increasing with global warming, warning systems should perhaps be revised to more consistently include humidity or use composite temperature–humidity metrics. The intensity of the urban heat island (UHI), and thus effective methods to mitigate UHIs, also depend on humidity.[120] For example, green roofs and street vegetation may enhance humidity, whereas white roofs and reflective pavement would likely have a neutral effect on humidity.[121] At a range of scales, from personal to regional, we assert that better understanding humidity's role in heat–health outcomes would clarify effective heat adaptation strategies.

Summary and Conclusions

We sought to understand the disconnect between physiologists and epidemiologists regarding humidity's role in health outcomes of elevated heat, and hypothesize possible reasons for the differing conclusions. In climate chamber experiments and human energy balance modeling, physiologists find a key role for humidity in driving human heat strain; at high levels of humidity, efficiency of sweating—the main mechanism by which the human body cools itself—decreases.[18,22,122] In contrast, epidemiologists conducting time-series analyses comparing health outcomes (i.e., morbidity and mortality) with environmental drivers typically find a negligible role for humidity.[19] We argue that reconciling these diverging views of humidity is critical in a changing climate. As concentrations of atmospheric greenhouse gasses continue to rise, ambient temperature will increase, as will specific humidity over most land regions.[20,123] As a result, both the rate and pattern of the increase in moist heat with global warming are distinctly different from that of temperature alone: Although temperature generally exhibits polar-amplified warming, moist heat exhibits tropical-amplified increases.[49,116] Humidity's role in heat–health outcomes thus could substantially alter projections of health burdens from climate change. Disentangling humidity's impacts on heat-related health outcomes is also necessary to select appropriate adaptations to present and future extreme heat.

As an interdisciplinary team covering epidemiology, physiology, biometeorology, public health, and climate science, we hypothesize reasons for the physiology-epidemiology disconnect around humidity (summarized in Tables 2 and 3). Our explanations span issues with epidemiological analyses, health and weather data limitations, and physiological reasons that may limit the influence of humidity in actual heat waves as opposed to idealized thermal chambers. These hypotheses are intended to galvanize research to bridge these knowledge gaps. Addressing this disconnect will require multidisciplinary research. Climate scientists can develop a nuanced understanding of how temperature and humidity extremes covary on diurnal and longer timescales, of the meteorology of moist heat waves in the present, and of how such events could change with global warming. Epidemiologists can carefully consider how humidity is incorporated into their models, determine whether humidity's discernible role depends on the cause of mortality or hospitalization, explore robustness of conclusions to higher temporal resolutions of data, and seek to incorporate more records from the tropics, LMICs, and places where temperature and humidity are less correlated. Physiologists (in concert with medical doctors) can work to better characterize the mechanisms and timescales of death during heat waves, and humidity's relevance to these mechanisms. Heat is the most direct way climate change impacts human health— resolving these cross-disciplinary debates around humidity is necessary to ensure that future heat–health projections and adaptation measures are robust and effective.

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The Coupled Model Intercomparison Project Phase 6 (CMIP6) data used to generate Figure 2 are available for download from the Earth System Grid Federation at https://esgf-node.llnl.gov/projects/esgf-llnl/.

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