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Intersecting vulnerabilities in human biology: Synergistic interactions between climate change and increasing obesity rates

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Abstract

Objectives: Increasing obesity rates and accelerating climate change represent two global health challenges shaped by lifestyle change and human environmental modifications. Yet, few studies have considered how these issues may interact to exacerbate disease risk.

Methods: In this theory article, we explore evidence that obesity-related disease and climatic changes share socio-ecological drivers and may interact to increase human morbidity and mortality risks. Additionally, we consider how obesity-climate change interactions may disproportionately affect vulnerable populations and how anthropological research can be applied to address this concern.

Results: Interactions between heat stress and cardiometabolic disease represent an important pathway through which climate change and obesity-related morbidities may jointly impair health. For example, individuals with higher body fatness and obesity-related metabolic conditions (eg, type 2 diabetes) exhibit a reduced ability to dissipate heat. The risk of poor health resulting from these interactions is expected to be heterogeneous, with low- and middle-income countries, individuals of lower socioeconomic status, and minority populations facing a greater disease burden due to relative lack of resource access (eg, air conditioning). Moreover, older adults are at higher risk due to aging-associated changes in body composition and loss of thermoregulation capabilities.

Conclusions: Few policy makers appear to be considering how interventions can be designed to simultaneously address the medical burden posed by increasing obesity rates and climate change. Anthropological research is well situated to address this need in a nuanced and culturally-sensitive way; producing research that can be used to support community resilience, promote holistic well-being, and improve health outcomes.

1 | INTRODUCTION

Environmental changes driven by human activities have contributed to the emergence of unprecedented challenges to health, including increasing obesity rates and accelerating climate change. For example, technological modifications have facilitated increased calorie availability, decreased the need for physical activity, altered sleep patterns, and created new psychosomatic stressors, all factors linked with rising global obesity rates and associated cardiometabolic disease (Chaput, Pérusse, Després, Tremblay, & Bouchard, 2014; Friedman, 2009;

Ladabaum, Mannalithara, Myer, & Singh, 2014). Similarly, human activities like deforestation and greenhouse gas emissions have been clearly identified as drivers of changing climatic conditions (Solomon, Plattner, Knutti, & Friedlingstein, 2009; Tilman & Lehman, 2001; Vitousek, Mooney, Lubchenco, & Melillo, 1997). In this theory article, we discuss the growing evidence that interactions between these two global concerns exacerbate human morbidity and mortality risks. We also explore how anthropological research can be used to shed light on the interacting mechanisms and design interventions, which address both issues simultaneously.

1.1 | The health consequences of rising obesity rates and a changing climate

Obesity represents an important global driver of morbidity and premature mortality (CDC, 2020; WHO, 2018). It is important to recognize that obesity is not linked with poor health outcomes in all cases. Still, obesity has been associated with a range of chronic diseases, including hypertension, type 2 diabetes, coronary heart disease, and some forms of cancer. Recent evidence also indicates that waist size is an independent risk factor for cardiovascular disease, exhibiting a nonlinear dose-response (Iliodromiti et al., 2018). Documenting obesity rates at the population-level can therefore help identify groups that may be at greater risk of developing cardiometabolic conditions.

Obesity patterns vary across populations, with some groups exhibiting higher rates than others. For instance, compared to non-Hispanic whites or Asians, high rates of obesity are observed among Hispanics and non-Hispanic blacks in the United States (CDC, 2020). In addition, individuals with more education and/or in higher income brackets often exhibit lower obesity rates. These trends demonstrate the complex interactions between social factors (eg, experienced racism, cheap calories, built environments that encourage inactivity, and unequal healthcare access) and obesity risk (Chaput et al., 2014; Hicken, Lee, & Hing, 2018; Hicks, 2019). To prevent the stigmatization of overweight individuals, it is therefore necessary to avoid attributing these patterns to individual choices and instead acknowledge that the modern obesogenic environment strongly shapes obesity risk in ways beyond individual control (Furlow, 2013; Lake & Townshend, 2006; Sturm & An, 2014).

Still, while population variation is evident, obesity rates have been steadily rising around the world, leading to public health efforts to improve nutrition and metabolic health. Recent global estimates indicate that approximately 40% of adults aged 18 and older were categorized as overweight in 2016, while ~13% were classified as adults with obesity (WHO, 2018). Increasing rates of overweight and obesity are also apparent in children, with over 381 million children and adolescents falling into these categories in 2016; this trend elevates the risk of long-term morbidity and poor health (WHO, 2018). In OECD countries (a group of 36 mostly high-income nations), these figures are even more stark, with an estimated one in two adults and one in six children classified as overweight or with obesity (Organisation for Economic Cooperation and Development, 2017).

Human alterations of the environment have not only contributed to rising obesity rates, but to changing climatic conditions. Human activities have been clearly linked with global climatic change, contributing to rising global temperatures, natural disasters (eg, droughts and wildfires), extreme weather events (eg, torrential rain and heat waves), detrimental environmental changes (eg, sea level rise), and losses in food production (eg, decreased agricultural and fishery outputs; Swinburn et al., 2019; Watts et al., 2018). Recent evidence indicates that human-induced warming reached ~1°C above preindustrial levels in 2017 and is on track to reach 1.5°C between 2030 and 2052, if warming continues at the current rate (Masson-Delmotte, 2018).

It is important to note that singular severe weather events are distinct from overall climate change. Weather refers to more immediate, day-to-day conditions while climate is defined by long term weather patterns within a specific location (National Oceanic and Atmosphere Administration, 2020). Extreme weather is not a phenomenon caused only by climate change, yet there is good evidence to suggest that the frequency of severe weather events—such as extreme temperatures—is increasing due to human-driven climate change. Increasingly sophisticated statistical models have been used to document changes in the occurrence of temperature-related adverse health outcomes and determine the degree to which these changes can be attributed to climate change, as opposed to chance weather events (Ebi, Ogden, Semenza, & Woodward, 2017). Additionally, despite the difficulty of directly measuring climate change effects, researchers can assess the frequency and severity of extreme weather, as well as differential health effects linked with these conditions. Overall, this research indicates that ongoing climatic change exacerbates global morbidity and mortality rates, increasing demands on already strained medical systems (Watts et al., 2018).

For instance, warming temperatures appear to facilitate the spread of vector-borne diseases (eg, malaria) into new areas, while flooding and the displacement of communities due to extreme weather events are associated with the spread of infectious diseases (eg, cholera). The physical effects of climate change, such as heat stress,

may also overwhelm thermoregulatory processes, increasing the risk of both cardiovascular disease and associated mortality (Watts et al., 2018; Ziegler, Morelli, & Fawibe, 2017). However, little attention has been paid to how climatic changes may interact with other important global health issues, including chronic metabolic conditions. The negative health effects linked with obesity and climate change have long been studied by researchers but have only recently been considered together (An, Ji, & Zhang, 2018; Edwards & Roberts, 2009; Nichols, Maynard, Goodman, & Richardson, 2009). It is important to determine how these global health issues influence one another. Increasing temperatures and rising rates of obesity around the world are realities that are not expected to change in the near future. Understanding how obesity-related disease and changing climates interact to exacerbate health outcomes is therefore important for the coordinated and strategic design of future research studies and interventions (An et al., 2018).

1.2 | Synergistic sociocultural drivers

Climate change and obesity risk share several common drivers (Figure 1). The primary drivers of fossil fuel reliance, population growth, and economic development shape land use patterns, cultural practices, and local economies in ways that influence individual physiology and health outcomes. For example, these primary drivers interact in ways which favor increased motorized transportation, agricultural production, and urban expansion; these alterations in turn support increased access to high-calorie foods and reduced physical activity levels, elevating obesity risk (An et al., 2018; Sturm & An, 2014; Woodcock, Banister, Edwards, Prentice, & Roberts, 2007). In addition to compromising metabolic health, these same primary drivers exert several environmental impacts, including deforestation and higher greenhouse gas emissions, key anthropogenic contributors to climate change (An et al., 2018; McMichael, Powles, Butler, & Uauy, 2007). Ultimately, this type of environmental modification contributes to rising temperatures and the likelihood of more extreme weather events, consequently increasing the risk of poor health outcomes linked with heat stress (An et al., 2018; McMichael et al., 2007).

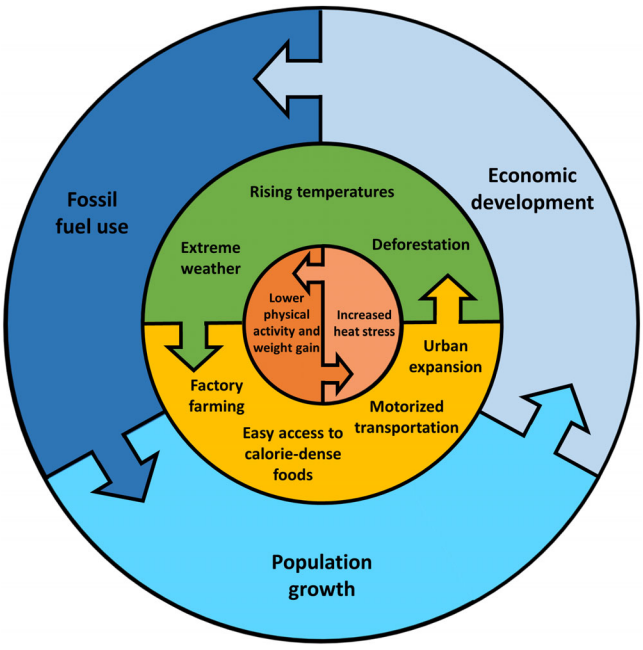

FIGURE 1 Synergistic drivers of rising obesity rates and climate change, fueled by the primary global forces of economic development, population growth, and fossil fuel use (in blue). These forces then affect national-level environmental (green) and sociocultural/economic (yellow) drivers of obesity and climate change, which in turn influence individual-level biological factors (orange)

The environmental and socioeconomic consequences of human activities are also reinforcing, interacting in ways that often negatively impact individual health. For instance, severe weather linked with anthropogenic climate change may decrease agricultural yields, driving up the prices of healthier food options (eg, produce) and leading to the consumption of cheaper but less nutritious meals (Husband, 2013; Lake et al., 2012). Climate change may also decrease the protein and micronutrient content of certain plant foods (eg, wheat, barley, rice, potato, and soy), thereby contributing to global malnutrition rates (Taub, Miller, & Allen, 2008). Climate change may also directly impact individual health by altering behaviors. To take one example, warmer temperatures may lead individuals to spend more time indoors in air-conditioned spaces. This increased reliance on cooling (and heating) to buffer individuals from extreme temperatures has been linked with minimized thermoregulation energetic demands, which may partly contribute to rising obesity rates (Moellering & Smith, 2012). Greater air-conditioner use also appears to contribute to rising CO_2 emissions, which in turn fuels accelerating climate change (eg, Isaac & Van Vuuren, 2009). Thus, an increased reliance on technology to create thermally comfortable spaces may result in a positive feedback loop, adding to global emissions, and extreme temperatures, which in turn increases the use of heating–cooling systems and decreases individual thermoregulation energetic expenditures. Ultimately, this cycle may contribute to the risk of both obesity and ongoing climate change.

Additionally, rising temperatures often lead individuals to engage in less physical activity or intentional exercise, potentially contributing to continued weight gain and poor metabolic health (An et al., 2018; Swinburn

et al., 2019). One study found that extreme seasonal temperatures were linked increased obesity risk, yet nearly all associations between obesity rates and the natural environment were mediated by decreased physical activity levels (von Hippel & Benson, 2014). At the same time, warmer temperatures may discourage people from cooking at home and increase the frequency of eating out, which has been linked with weight gain (Bezerra, Curioni, & Sichieri, 2012; Lachat et al., 2012). Thus, global warming may alter individual physical activity and dietary patterns in ways that increase obesity risk. Population-level measures of climatic variables and obesity rates also appear to translate to biological effects at the individual-level. Specifically, the cumulative effects of a changing climate, in conjunction with widespread shifts in diet and activity patterns, may lead to detrimental interactions between experienced heat stress and cardiometabolic processes, exacerbating morbidity experienced by vulnerable individuals.

2 | SYNERGY AT THE BIOLOGICAL LEVEL: INTERACTIONS BETWEEN CARDIOMETABOLIC DISEASE RISK AND HEAT STRESS

There are a multitude of potential biological pathways through which obesity-related disease and climate change may interact. This is partially due to the fact that global climate change is predicted to influence a wide spectrum of diseases, ranging from zoonotic infections to malnutrition. The specific biological pathways through which the global rise in cardiometabolic disease and climate change will interact depends on local ecological, socioeconomic, and cultural conditions. For instance, cultural norms and behavioral adaptations, such as napping during the hottest part of the day, designing clothing and habitations to effectively cool and reduce sunlight exposure, and attention to hydration status may all mitigate heat stress (Hanna & Brown, 1983; Rosinger, 2015). Additionally, individual biological factors appear to play an important role, with synergistic effects between cardiometabolic health and climatic factors that are likely to play out through multiple interrelated biological mechanisms. Here we explore one possible biological pathway: interactions between cardiometabolic disease and heat-related morbidities.

The biological mechanisms that change during adaptive responses to heat stress overlap with those involved in the etiology of obesity-related cardiometabolic dysregulation (Figure 2). These shifting biological parameters include blood viscosity, vasodilation/vasoconstriction, sweat production, plasma osmolality, and adipose tissue physiology. When the body is exposed to heat stress, sensory neurons called thermoreceptors located in the skin, muscle, and brain send a signal to the hypothalamus indicating there has been a change in temperature (Kenny, Sigal, & McGinn, 2016). The preoptic anterior of the hypothalamus then coordinates a physiological response to increase dry heat exchange and evaporative heat loss. An increase in dry heat exchange is achieved by increasing vasodilation of the blood vessels. The hypothalamus stimulates evaporative heat loss by increasing sweat production. Sweating can lead to shifts in blood viscosity and plasma osmolality, or the proportion of electrolytes to water in the plasma. Sustained heat exposure can also downregulate the expression of thermogenic genes in adipose tissue (Roh et al., 2018). Although human cooling mechanisms typically fail at higher temperatures - leading to an increased risk of heat exhaustion and heat stroke when core body temperatures exceed 37°C - evidence indicates that individuals are able to acclimatize to hotter temperatures to an extent (Glazer, 2005).

The process of heat stress acclimatization has primarily been examined in studies of exercise in hot environments (Leonard, 2015). During the first four days of acclimation, participants exhibit increased vasodilation but excessive, ineffective sweating. After about 2-4 weeks of heat stress exposure, however, participants exhibit more evenly distributed sweating, which facilitates greater evaporation, and even higher levels of vasodilation and peripheral heat conductance (Leonard, 2015).

The ability to effectively acclimatize to heat stress declines with age due to wear and tear on the biological mechanisms listed in Figure 2. Older adults have a harder time sensing and regulating their hydration status (Rodriguez et al., 2009). Sweat production becomes less efficient with age, and vasodilation of the blood vessels near the skin can become impaired (Kenny, Sigal, & McGinn, 2016). Simultaneously, the risk of cardiometabolic disease increases with age. Thus, the bidirectional relationships between cardiometabolic health and physiological responses to heat stress are particularly apparent among the elderly (Kenney, Craighead, & Alexander, 2014). Below we describe biological mechanisms through which heat stress can exacerbate cardiometabolic disease, as well as the pathways through which cardiometabolic disease can impair thermoregulation during heat stress (Figure 2).

2.1 | Pathways through which warming climates and heat stress may affect obesity-related morbidities

Shifts in blood viscosity and vasodilation during heat stress can exacerbate an individual's risk of cardiometabolic

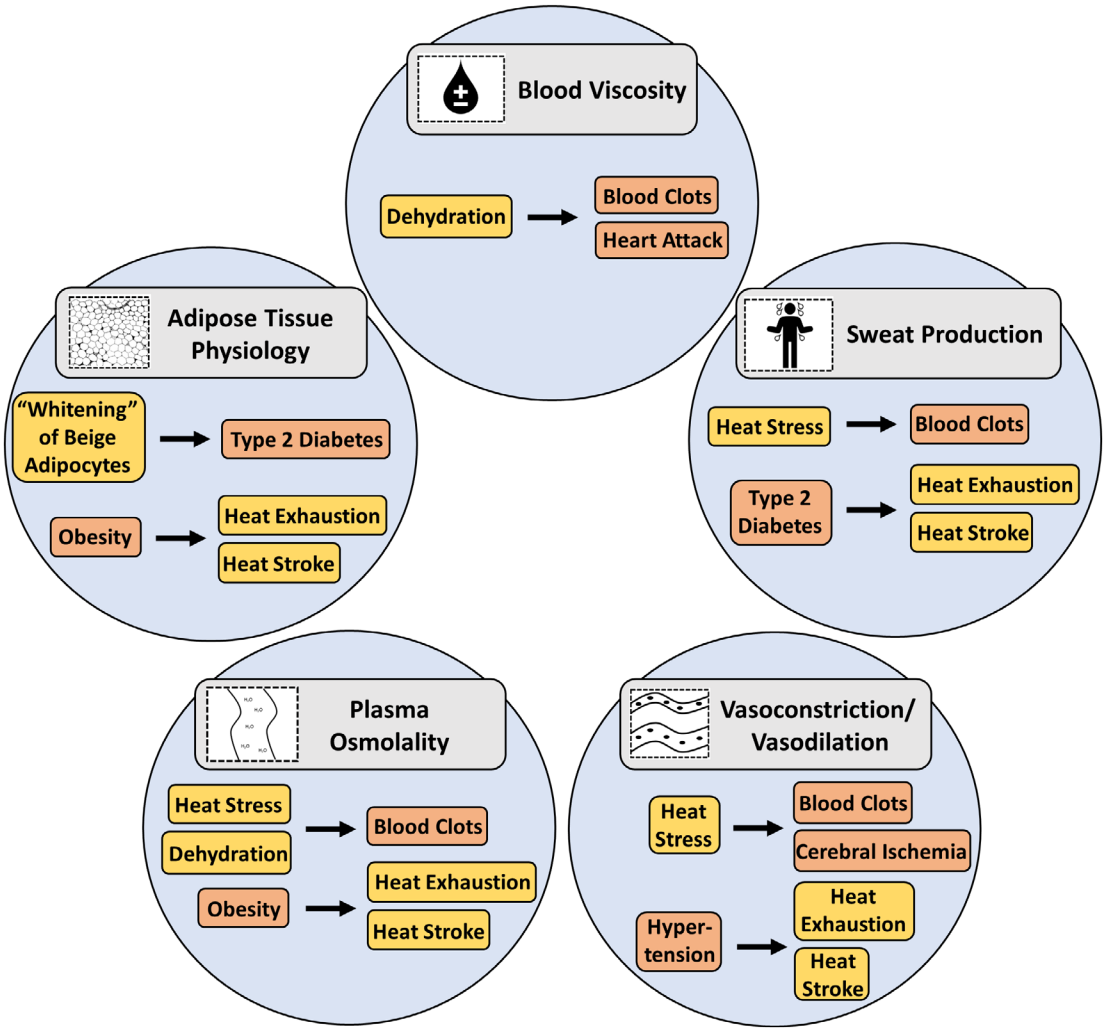

FIGURE 2 Biological pathways involved in the etiology of both obesity-related disease and heat-related morbidity. The figure highlights five biological mechanisms (large blue circles) through which heat-related conditions (in yellow) and cardiometabolic disease (in red) interact

disease morbidity and mortality. For instance, acute heat stress can increase the risk of blood clots in the heart and brain, conditions respectively known as coronary or cerebral thrombosis. Individuals that are exposed to 41°C for 6hours or more experience significant increases in red blood cell count, platelet volume, blood viscosity, and plasma osmolality, all of which increase the risk of cerebral and coronary thrombosis (Keatinge et al., 1986; Rodriguez et al., 2009). These changes are likely due to a net loss of body water and salt from sweating, even with unlimited access to water.

The risk of cerebral thrombosis during a heat wave may be even higher for adults with hypertension (Keatinge et al., 1986). A sudden drop in blood pressure in response to heat stress, even to normal levels, makes it difficult for hypertensive patients to pump enough blood to the head. This can lead to either cerebral thrombosis or cerebral ischemia (a fatal decline in blood flow to the brain) (Keatinge et al., 1986). Heatwaves are also linked to a spike in the number of hospitalizations from myocardial infarction (heart attack) (Morabito et al., 2005; Sun, Chen, Xu, & Li, 2018). The precise biological mechanisms that cause this trend remain unclear; however, it is possible that increased blood viscosity due to dehydration may play a role (Sun et al., 2018).

Another pathway through which warming ambient temperatures may influence cardiometabolic disease risk is via brown adipose tissue (BAT) development and its influence on glucose metabolism (Maurer, Fromme, Mocek, Zimmermann, & Klingenspor, 2020). White adipose tissue (WAT) is the most prevalent form of adipose tissue on the body and its primary function is to store energy in the form of lipids. Conversely, BAT is rich in mitochondria and thus highly metabolically active, functioning as a critical mammalian adaptation to low ambient temperatures. BAT is present in all human infants

and some adults (Enerbäck, 2010). During cold exposure, the sympathetic nervous system sends a signal to brown adipocytes, which triggers an increase in the breakdown of lipids and glucose for mitochondrial metabolism and thermogenesis. Recent work suggests that BAT may play an active role in the regulation of glucose metabolism and storage (Iwen et al., 2017; Matsushita et al., 2014; Maurer et al., 2020). Researchers have consequently proposed that cold-induced BAT metabolism may have protective effects against the development of type 2 diabetes mellitus (Iwen et al., 2017).

The protective effects of BAT thermogenesis against the development of cardiometabolic disease may be dependent on prior exposure to cold stress over the life course. Repeated exposure to cold stress over 10 days can stimulate the expansion of BAT mass (van der Lans et al., 2013). Evidence from rodent models indicates, however, that when mammals are exposed to neutral temperatures for long periods of time, brown adipocytes undergo morphological "whitening," increasing the amount of lipids stored in fat vacuoles (Cui et al., 2016; Roh et al., 2018). Additional work is needed to test how climate-controlled environments and indoor heating contribute to this phenomenon in humans. Recent work examining developmental plasticity in BAT in an indigenous circumpolar population, the Yakut of northeastern Siberia, suggests that adult BAT thermogenesis may be sensitive to climatic conditions early in life. Yakut individuals who were exposed to colder winters and reported greater participation in outdoor winter activities during early childhood exhibit greater BAT thermogenesis in adulthood (Levy, 2017). These findings cumulatively suggest that time spent outside during development strongly influences human thermogenesis; these processes may, therefore, be sensitive to changes in climate conditions. However, the pathways through which climate change and heat exposure may promote type 2 diabetes by altering the development of BAT deposits have yet to be thoroughly explored.

2.2 | Pathways through which obesity-related morbidities affect biological responses to heat stress

On average, the risk of heat stress is elevated among children and adults with obesity (Chung & Pin, 1996; Dougherty, Chow, & Kenney, 2010), such that individuals with clinically high BMIs tend to maintain higher core body temperatures during heat exposure (Habibi, Momeni, & Dehghan, 2016; Zhang, Noda, Himeno, & Liu, 2016). It has been hypothesized that individuals with higher body fat maintain warmer core temperatures because the thickness of fat causes an increase in thermal resistance between the core and periphery, thus restricting heat dissipation (Zhang et al., 2016). The insulative value of human body fat, however, is currently under debate (Brychta, Cypess, Reitman, & Chen, 2019; Fischer, Cannon, & Nedergaard, 2019).

Alternatively, higher body fatness may maintain warmer core temperatures during heat stress due to a higher body mass-to-surface area ratio, which may lower the capacity for heat dissipation (Kenny, Yardley, Brown, Sigal, & Jay, 2010). Additionally, the risk of heat exhaustion during physical activity may be elevated among individuals with high BMIs since carrying more body weight during weight-bearing exercise requires additional energy expenditure and leads to greater heat production (Bar-Or, Lundegren, & Buskirk, 1969). Individuals with high BMIs also have a higher prevalence of dehydration and more concentrated urine despite greater water intake (Kant, Graubard, & Atchison, 2009; Rosinger, 2020). It has been hypothesized that obesity may disrupt the homeostatic mechanisms that regulate electrolyte balance within and between cells due to alterations in body fluid distribution, causing an increase plasma osmolality, and this may exacerbate the risk of heat stress (Rosinger, 2020; Thornton, 2010).

Evidence also indicates that adults with diabetes mellitus exhibit a heightened vulnerability to heat stress due to impairments in their capacity to dissipate heat (Kenny et al., 2016). This pattern is found among both type 1 and type 2 diabetes mellitus (T2D) patients, and these patients account for a disproportionate number of hospitalizations and deaths during extreme heat waves (Schwartz, 2005). Since the etiology of type 2 rather than type 1 diabetes is linked to obesity and metabolic dysregulation, our discussion will focus on the two main biological pathways that link T2D with thermoregulatory impairments.

First, patients with T2D exhibit lower skin blood flow in response to local skin heating and whole-body heating, which limits their capacity for dry heat exchange (Kenny et al., 2016; Petrofsky, Lee, Patterson, Cole, & Stewart, 2005). The biological mechanisms that underlie this pattern remain unclear. It has been hypothesized that patients with T2D might have lower blood nitric oxide, a signaling molecule known to stimulate vasodilation. Support for this hypothesis, however, has been mixed (Kenny et al., 2016; Sokolnicki, Roberts, Wilkins, Basu, & Charkoudian, 2007). Additional work is needed to determine the mechanisms driving reduced skin blood flow in diabetic patients.

Second, T2D patients have a limited capacity for evaporative heat loss due to an impaired sweating response. In particular, T2D patients display a reduced sweating response on the lower half of the body and a hyperactive

sweating response on their upper body (Kenny et al., 2016; Petrofsky et al., 2005). Again, the biological mechanisms that underlie this pattern are unclear. Reductions in lower-body sweating are likely tied to diabetic neuropathy (pain or numbness from nerve damage), which begins with the nerves of the feet and progresses up the body. Greater upper-body sweating may be a compensatory reaction in response to reductions in lower body sweating (Kenny et al., 2016). The inability to maintain glycemic control, or healthy blood glucose levels, is associated with the severity of the impairments to thermoregulation. Thus, it appears that individuals with T2D can delay the onset of impairments to heat loss by maintaining good glycemic control through a healthy diet and exercise (Kenny et al., 2016).

Hypertension can also put individuals at an elevated risk for heat exhaustion and heat stroke (Carberry et al., 1992; Kenny et al., 2010). Adults with hypertension have restricted blood flow due to increased vascular smooth muscle activity and structural changes in the vasculature system (Folkow, 1982). This can impede the passage of heat from the core toward the periphery of the body as well as dry heat exchange through the skin (Kenny et al., 2010). Compared to nonhypertensive adults, adults with hypertension exhibit significantly less blood flow in the skin in response to either local or whole-body heating (Carberry et al., 1992; Kenney, Kamon, & Buskirk, 1984).

Hypertension is also hypothesized to aggravate heat stress-induced cognitive dysfunction and brain pathology (Muresanu & Sharma, 2007). Research in a rodent model indicates that (compared to nonhypertensive mice) hypertensive mice are at significantly higher risk of developing motor and cognitive impairments, breakdown of the blood–brain–barrier, and accumulating liquid in the brain (cerebral edema) after exposure to 4 hours of heat stress (Muresanu & Sharma, 2007). Thus, hypertension may magnify the risk of brain damage in the wake of a heat stroke. These findings cumulatively demonstrate the various pathways through which metabolic dysregulation may also increase the risk of heat-related morbidity; however, the biological mechanisms through which these effects occur likely vary across individuals.

3 | GROUPS MOST VULNERABLE TO INTERACTIONS BETWEEN A CHANGING CLIMATE AND CARDIOMETABOLIC DISEASE

We hypothesize that the consequences of the synergistic effects of climate change and obesity-related disease are likely to be heterogeneous across groups at both the country-level, as well as across individuals within a community (An et al., 2018). Specifically, groups that are experiencing the most dramatic ecological shifts due to climate change and the greatest burden of cardiometabolic diseases will be more susceptible to their interacting effects. Here we highlight several factors that may heighten vulnerability to the effects of climate-obesity interactions in order to guide future research into relevant social and ecological determinants of health.

At the country level, low- and middle-income countries (LMICs) are predicted to be more dramatically impacted by climate change than high-income countries, despite generally producing a lower portion of greenhouse-gas emissions (Swinburn et al., 2019). For instance, the consequences of climate change for agricultural production, food insecurity, and infectious diseases are projected to be more severe in LMICs (IPCC, 2014). Additionally, the prevalence of obesity-related diseases is increasing at a faster rate in LMICs. By 2025, nearly three quarters of illness and deaths from noncommunicable diseases, such as the metabolic syndrome, are projected to occur in LMICs (GBD, 2016; Risk Factors Collaborators, 2017). This is in part due to the "Dual Burden of Disease," an epidemiological trend characterized by the existence of over- and under-nutrition within the same community, household, or even individual (Tzioumis & Adair, 2014).

The prevalence of the dual burden of disease is also growing among many indigenous communities. Previous work highlights how colonization, disenfranchisement, and the loss of land are at the root of high rates of cardiometabolic diseases among many indigenous populations (Valeggia & Snodgrass, 2015). Sea-level rise, floods, and forest fires due to the local effects of global climate change further threaten the land of indigenous communities (Snodgrass, 2013). Many indigenous groups are experiencing shifts in lifestyle and nutritional transitions characterized by a decline in physical activity levels and an increase in high-calorie, high-fat, high-salt, and low-fiber foods (Valeggia & Snodgrass, 2015). Global climate change is predicted to accelerate these current shifts in lifestyle and diet. Thus, indigenous communities are likely to be disproportionately impacted by the interacting consequences of declines in cardiometabolic health and climate change (FAO, 2020; United Nations Permanent Forum on Indigenous Issues, 2009).

Similar to predicted trends in indigenous populations, interactions between cardiometabolic disease and climate change may exacerbate existing racial health disparities. Recent research also highlights how implicit bias in medical practitioners perpetuates race disparities in health (Chapman, Kaatz, & Carnes, 2013). For instance, Green et al. (2007) used implicit association tests to examine the implicit biases of 287 internal medicine and emergency medicine residents in Boston and Atlanta. The study found

significant implicit bias favoring white Americans among the physicians. Furthermore, implicit bias against black patients was negatively correlated with likelihood of recommending treatment for symptoms of acute coronary disease. Doctors may also have similar racial biases when they encounter patients with symptoms of heat-stress.

In addition to medical barriers, other forms of discrimination may exacerbate obesity-climate change interacting effects. A recent study explored the relationship between historical redlining policies and present-day summer surface temperature in U.S. cities (Hoffman, Shandas, & Pendleton, 2020). Redlining is the practice of refusing home loans or insurance to neighborhoods based on racially motivated perception of investment risk. Land surface temperatures in redlined areas were around 2.6°C warmer than nonredlined areas on average, with greater inequities in cities in the southeastern and western U.S. Neighborhood disparities in surface temperature appear to be linked to less tree cover in conjunction with more highways and building complexes in redlined areas (Hoffman et al., 2020).

Similarly, epidemiological studies examining the health consequences of heat waves in three North American cities found that the rate of heat stroke and heat-related deaths was disproportionately higher among black residents compared to white residents (Applegate et al., 1981; Jones et al., 1982). Thus, recent research suggests that urban segregation, income-inequality, and institutional racism may be driving the relationship between race and heat-related morbidity and mortality, in part because exposure to heat stress appears to be dependent on access to better temperature-controlled spaces (Vargo, Stone, Habeeb, Liu, & Russell, 2016). Access to these spaces is in turn shaped by social and economic factors.

For example, individual socioeconomic status (SES; a measure of individual income, education, and social standing) will likely moderate any interactions between a changing climate and cardiometabolic disease. A growing body of work highlights a positive association between an individual's SES and better health (García, Gurven, & Blackwell, 2017; Howe et al., 2012). Previous work suggests a significant association between low SES and higher heat stress (Harlan, Brazel, Prashad, Stefanov, & Larsen, 2006), heat-related morbidity (Jones et al., 1982), or heat-linked mortality (Curriero et al., 2002). Access to air-conditioned (thermo-neutral) spaces, green space with shade, and safe water sources may be limited for low-income individuals, which can increase the risk of heat-related morbidities (Ito, Lane, & Olson, 2018; Nayak et al., 2018). Climate change may also magnify issues of food insecurity among low-income populations by jeopardizing subsistence farming and large-scale agricultural industry. Further, occupational heat stress is projected to increase for many blue-collar professions where workers spend their time predominantly outdoors away from air-conditioned workspaces, such as construction, agriculture, forestry, fishing, and mining (Spector & Sheffield, 2014). The burden of cardiometabolic diseases is consequently shifting quickly toward lower socioeconomic groups, especially in wealthy countries (Monteiro, Moura, Conde, & Popkin, 2004).

Additionally, multiple economic, social, and biological risk factors are often compounded in elderly populations, likely making individuals over 60 years old more vulnerable to the synergistic effects of cardiometabolic disease and climate change (Kenny et al., 2010). The risk of developing obesity and cardiometabolic diseases such as diabetes and cardiovascular disease increases with age in most populations. At the same time, older individuals are strongly affected by severe heat. A study of the 2003 heat wave in France found that a majority of excess mortality occurred among individuals over 60 years old (Fouillet et al., 2006). Research in both France and the United States has found that most deaths caused by heat waves occur among elderly people living in institutions, confined to bed, or living alone (Semenza et al., 1996).

Even among otherwise healthy older adults, the ability to sense heat stress, regulate fluid intake, and maintain a constant internal body temperature can become compromised with age (Kenny et al., 2010). Key biological pathways that are involved in both the etiology of cardiometabolic diseases and heat-related morbidity, such as blood flow to the skin, efficient sweat production, and cardiovascular function, can deteriorate in older individuals. The proportion of elderly people in the global population is growing, leading to calls for investigations into the health concerns of an aging public (Winker & DeAngelis, 2010). Future work should consider healthy aging within the context of interactions between global climate change and elevated cardiometabolic disease risk. The following section highlights the advantages of applying an anthropological perspective to these kinds of investigations.

4 | POTENTIAL CONTRIBUTIONS OF ANTHROPOLOGISTS TO RESEARCH TESTING ASSOCIATIONS BETWEEN CLIMATE CHANGE AND OBESITY-RELATED DISEASE

Efforts to effectively address the global health crisis posed by accelerating climate change, rising obesity rates, and their interactions would benefit greatly from the integration of research foci from diverse fields, including

anthropology. Anthropologists are interested in many topics which likely impact any obesity-climate change interactions, including the effects of socioeconomic inequalities on human health (eg, Minos et al., 2016; Puhl & Brownell, 2006; Roncoli, Crane, Orlove, Crane, & Orlove, 2016; Swinburn et al., 2019), links between cultural/technological change and well-being (eg, Butaric, Light, & Juengst, 2017; Godoy & Cárdenas, 2000; McDade & Nyberg, 2010), human resilience (Huntington et al., 2019; Roncoli et al., 2016), and biological responses to climatic stressors (eg, Snodgrass, Sorensen, Tarskaia, & Leonard, 2007). Existing anthropological perspectives and methodologies are therefore well suited to address several pressing concerns associated with interactions between rising obesity rates and climate change.

Unlike most medical research, anthropologists often use biocultural methods and cross-cultural study designs to assess health patterns across diverse socio-ecological settings, testing the role that social structure and lifestyle factors play in health patterns (Lock & Nguyen, 2018; Manderson, 1998). This enables anthropologists to more comprehensively analyze the complex cultural, socioeconomic, political, physiological, and behavioral factors, which interact to drive increasing obesity rates and climate change, both on a local and global scale. In other words, anthropological work using a biocultural approach can highlight how socioeconomic factors within a specific cultural context interact with underlying physiological responses to shape health outcomes.

To take one example, work among indigenous Siberians indicates that humans may effectively respond to extreme climatic conditions through a suite of biological adaptations (eg, body shapes which reduce heat loss, elevated basal metabolic rate, and high metabolic turnover) (Snodgrass et al., 2007). Yet, these biological traits may also increase the risk of developing cardiometabolic disease in some instances as lifestyle factors, such as diet and activity patterns, change with increased participation in a market economy. This same kind of biocultural perspective could be applied to identify how evolved physiological responses to novel challenges (eg, obesogenic environments and rising global temperatures) are shaped by local cultural practices (eg, population-specific resource extraction or dietary patterns).

Additionally, anthropologists are practiced in using cross-cultural comparisons to explore human variation, an approach which may provide important insights from affected communities to better document the full range of experiences linked with changing climatic and health patterns (Huntington et al., 2019; Roncoli et al., 2016). Cross-cultural studies can also help anthropologists identify how physiological and social factors interact to put certain groups at greater risk for health complications from the interactions between heat stress and obesity-related conditions, data useful in protecting vulnerable populations. Importantly, anthropological work can also be used to bring typically ignored groups into the wider conversation (Huntington et al., 2019; Roncoli et al., 2016). In addition, this type of dialogue can highlight how certain social inequalities intensify interactions between obesity-related disease and climate change.

For instance, biocultural research can highlight the ways in which many communities are unable to access goods or services (eg, healthy food or resources to mitigate extreme weather), resulting in biological effects (eg, poor metabolic health or increased heat stress exposure) which may exacerbate obesity risk and the effects of climate change (eg, Minos et al., 2016; Puhl & Brownell, 2006; Roncoli et al., 2016; Swinburn et al., 2019). For example, anthropological perspectives on the sociopolitical and environmental factors shaping water insecurity could highlight the various ways that certain populations face a disproportionate risk of potable water loss and heat exposure due to climate change (Rosinger & Brewis, 2020; Wutich, 2019). This elevated water insecurity risk in certain populations may also be coupled with pre-existing metabolic health conditions, cumulatively exacerbating the negative effects of dehydration and heat stress. Thus, rather than victim blaming marginalized groups for their perceived undesirable choices or lack of resource access, anthropological research can contribute information needed to better identify and target socio-political drivers of poor health. Moreover, this type of anthropological approach can highlight localized responses to these challenges which exemplify human resilience, information that could perhaps be applied to improve health outcomes in other contexts.

Finally, anthropologists have a history of studying health outcomes in a range of changing cultural and environmental conditions, including examinations of how urban development and technological inventions impact lifestyle patterns and local ecologies (eg, Butaric et al., 2017; Godoy & Cárdenas, 2000; McDade & Nyberg, 2010). Thus, the anthropological toolkit can be used to connect ongoing cultural and environmental change to health consequences linked with interactions between climate change and metabolic health. Common anthropological methods, such as the measurement of minimally-invasive biomarkers (eg, anthropometric data, dried blood spots, saliva, etc.) will also be useful in quantifying how interactions between metabolic processes and climate change become embodied cross-culturally, especially when combined with direct measures of environmental conditions (eg, water insecurity level, individual hydration status,

direct humidity and heat exposure as measured by wet bulb globe temperatures or other techniques, etc.).

Anthropological work therefore has many applications in testing health patterns related to climatic and metabolic factors. Anthropologists interested in biocultural factors contributing to the obesity-climate change interactions could contribute important data to ongoing research exploring determinants of relevant health patterns. Understanding these biocultural factors could also help policy makers identify existing barriers to sociopolitical and medical action aimed at addressing the poor health effects of rising obesity rates and a changing climate.

5 | UNANSWERED QUESTIONS AND FUTURE DIRECTIONS

The biological effects of an interaction between climate change and obesity-related conditions remain largely hypothetical at this point. Research by human biologists is needed to test specific hypotheses related to predicted obesity-climate change interactions. Here we outline some questions of interest relevant to anthropological research foci:

1. *Measurable health outcomes*: Is there an association between hospitalizations for heat-related illnesses (eg, heat exhaustion or heat stroke) and patient body composition? How do extreme weather events (eg, heat waves) impact this relationship? If such a relationship exists, which measures of body composition or cardiometabolic health might serve as the best proxies of increased risk to the effects of heat stress? Do improvements to metabolic health result in increased physiological heat tolerance?
2. *Adaptation to thermal stress*: How does the process of acclimatization to heat stress influence cardiometabolic disease risk? Do warmer temperatures across development significantly influence individual baseline thermoregulation patterns? For example, are warming temperatures and/or climate-controlled living spaces in historically cold environments linked with reduced BAT development? How do seasonal shifts in diet, physical activity, and thermal stress exposure interact to shape disease risk?
3. *Impact on vulnerable populations*: Does body composition moderate the relationship between age and vulnerability to heat stress? Does resource access (eg, ability to afford air conditioning) mitigate the negative physiological effects of heat stress? In other words, are any existing links between body composition and physiological heat stress influenced by individual SES? Do marginalized groups exhibit a higher dual-burden of obesity-related and heat stress-related medical conditions relative to higher-income populations?
4. *Sociocultural change*: How do shifts in social, political, and economic contexts influence the health consequences of obesity-climate change interactions at a local level? For instance, how does market integration, migration, or political upheaval influence interactions between cardiometabolic disease risk and heat-related morbidity? What local conditions are protective against any negative health consequences resulting from interactions between climate change and cardiometabolic conditions? How do coping strategies vary between communities or individuals exposed to similar conditions? In other words, how well do various adaptive mitigation approaches insulate individuals from heat stress and associated cardiometabolic health effects?

6 | CONCLUSION

By considering interactions between climate change and rising obesity rates, it may be possible for health researchers (including anthropologists) and policy makers to design interventions, which more effectively address both issues. For example, policies that place restrictions on fossil fuel use, promote sustainable energy sources, and improve cardiometabolic health will likely mitigate the medical and economic burdens linked the obesity-climate change interactions through addressing both issues at once (An et al., 2018; Bloomberg & Aggarwala, 2008; Husband, 2013). Similarly, certain food options associated with improved metabolic health (eg, whole grains, legumes, and leafy greens) have also been generally linked with lower environmental impact relative to less healthy food items (eg, red meats) (Clark, Springmann, Hill, & Tilman, 2019). Efforts to facilitate healthier food options may therefore decrease disease rates, with the added benefit of reducing the environmental degradation and green-house gas emissions linked with certain forms of food production (Lake et al., 2012; Springmann et al., 2016).

However, few policy makers appear to be considering how interventions can be designed to simultaneously address the medical burden posed by increasing obesity rates and climate change. Collaborations between human biologists and environmental researchers should be conducted to address this health challenge by finding areas of overlap where joint interventions can be implemented. Future research is needed to determine which groups are most likely to experience the harmful effects of any obesity-climate change interactions, and therefore may benefit the most from this more holistic intervention

approach. Anthropological research is well-situated to address this need in a nuanced and culturally sensitive way; producing research that can be used to support community resilience, promote holistic well-being, and improve health outcomes.

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AUTHOR CONTRIBUTIONS

Theresa Gildner: Conceptualization; investigation; writing-original draft; writing-review and editing. **Stephanie Levy:** Conceptualization; investigation; writing-original draft; writing-review and editing.

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