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Short Communication

Why not all People die From Heat Stress?

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Abstract

In recent years, the study of the effects of heat stress on human health become quite relevant due to abnormal temperature rises in the temperate and northern regions of Europe and North America, which were accompanied by higher mortality rates among older age groups. It was generally believed that the pathogenesis of human death from heat stress was well understood. Now it turned out that our knowledge had been overstated, because people started dying even among those who had access to highly qualified medical care. From our point of view, this includes the underestimated role of human body heat conductivity (BHC) and its wide variation in the population when it comes to thermoregulation. Maybe that is why older individuals in the population turned out to be the most vulnerable group, having the lowest BHC.

Keywords: older adults; heat stress; human body heat conductivity; chromosomal Q-heterochromatin; cell thermoregulation; thermoregulation

Introduction

Until recently, the study of the effects of increased temperature on human life was mostly academic. However, in recent years, this issue has become quite relevant due to abnormal temperature rises in the temperate and northern regions of Europe and North America, which were accompanied by higher mortality rates among older age groups. It was generally believed that the pathogenesis of human death from heat stress was well understood, although this confidence was mainly based on data obtained from animals. Now, when the day of reckoning came, it turned out that our knowledge had been overstated, because people started dying even among those who had access to highly qualified medical care. According to preliminary calculations based on excess mortality, the number of victims has already exceeded 6,000 just in France alone. This is not just a statistic — it is a signal that we do not know everything about the biological factors that increase an individual's vulnerability to high environmental temperatures. From our point of view, this includes the underestimated role of human body heat conductivity (BHC) and its wide variation in the population when it comes to thermoregulation.

The thing is individuals in a population differ from each other in the heat conductivity of their bodies [1,2]. It has been found that the level of a human's BHC is linked to the number of one type of chromosomal heterochromatin regions (HRs) in their genome, specifically Q-HRs [3]. As shown in Tables 1 and 2, using the example of two racial-ethnic groups, individuals from older age groups in the population reliably show a lower number of chromosomal Q-HRs [4].

Number of Q-HRs	Ages				
	Newborns (n = 145) I	17-19 years (n = 317) II	20-39 years (n = 112) III	40-59 years (n = 67) IV	>60 years (n = 23) V
0	4	21	7	8	3
1	19	70	20	13	4
2	23	105	41	20	11
3	38	71	19	14	2
4	37	39	16	10	3
5	16	9	7	2	
6	5	2	2		
7	3				
Total Q-HRs	458	706	270	145	44

Mean Number of Q-HRs	**3.16 ± 0.13**	**2.23 ± 0.07**	**2.41 ± 0.13**	**2.16 ± 0.16**	**1.91 ± 0.24**
	$t_{I, II} = 6.0$ df = 232 P < 0.000	$t_{I, III} = 4.01$ df = 255 P < 0.000	$_{I, IV} = 4.54$ df = 210 P < 0.000	$t_{I, V} = 4.58$ df = 38 P < 0.000	$t_{II, III} = 1.29$ df = 427 P > 0.20
	$t_{II, IV} = 0.42$ df = 382 P >0.600	$t_{II, V} = 1.21$ df = 338 P < 0.200	$t_{III, IV} = 1.20$ df = 177 P >0.200	$t_{III, V} = 1.64$ df = 133 P <0.100	$t_{IV, V} = 0.81$ df = 88 P <0.300

Table 1: The distribution and mean numbers of chromosomal Q-HRs per individual in Kyrgyz samples (Bishkek, Kyrgyzstan)

Number of Q-HRs	Ages				
	Newborns (n = 37) I	17-19 years (n = 67) II	20-39 years (n = 115) III	40-59 years (n = 230) IV	>60 years (n = 211) V
0	0	4	6	14	26
1	3	9	18	50	51
2	7	21	39	74	81
3	5	19	29	58	35
4	12	9	17	27	13
5	7	4	6	6	4
6	3	1		1	1
Total Q-HRs	133	170	281	516	396
Mean Number of Q-HRs	**3.59 ± 0.23**	**2.54 ± 0.16**	**2.44 ± 0.11**	**2.24 ± 0.08**	**1.88 ± 0.08**
	$t_{I, II} = 3.8$ df = 102 P <0.000	$t_{I, III} = 4.7$ df = 150 P <0.000	$t_{I, IV} = 6.1$ df = 265 P <0.000	$t_{I, V} = 7.8$ df = 246 P <0.000	$t_{II, III} = 0.5$ df = 180 P >0.50
	$t_{II, IV} = 1.7$ df = 295 P >0.70	$t_{II, V} = 3.8$ df = 276 P <0.000	$t_{III, IV} = 1.4$ df = 343 P >0.10	$t_{III, V} = 4.9$ df = 439 P <0.000	$t_{IV, V} = 3.1$ df = 439 P <0.0.002

Table 2. The distribution and mean numbers of chromosomal Q-HRs per individual in Russian samples (Bishkek, Kyrgyzstan).

As can be seen from these Tables, in all cases, individuals from the older adults have the lowest values and a narrow range of variability in the distribution of the numbers of Q-HRs compared to the younger age groups. These differences are statistically significant.

Several reviews have analyzed the effects of heat stress on mortality or morbidity in general populations [5-7]. However, few have specifically addressed thermoregulatory responses and frailty in older adults. Population-based studies demonstrate that exposure to high temperatures is significantly associated with increased mortality and hospitalizations in older adults. The risk is greater in advanced age groups (≥75 or ≥85 years) and, in many cases, in women.

Excessive heat represents a significant physiological stress or capable of disrupting multiple body systems **[8].** When ambient temperature exceeds the body's capacity for heat dissipation the temperature rises, initiating a cascade of cardiovascular and metabolic responses aimed at maintaining thermal homeostasis. If these responses are insufficient or impaired, thermal imbalance ensues, potentially resulting in hyperthermia, multi-organ dysfunction, and, in extreme cases, death [9].

The mechanism of the harmful effects of heat on the human body is generally well understood [17,18]. First, heat accumulated because the body cannot get rid of the heat energy produced during metabolic processes and from external heat exposure (like high temperatures, high humidity, low wind, or the presence of heat sources). Second, when exposed to heat, the human body, in addition to adaptive physiological responses (like skin blood vessel dilation and increased sweating) [10,11], starts producing a huge amount of proteins, such as antidiuretic hormone, aldosterone, and cortisol [12,13], pro-inflammatory cytokines (interleukin 1β, interleukin 6, interferon-γ, C-reactive protein [14]), and heat shock proteins, which preserve protein integrity, prevent cell death, and increase heat tolerance [15,16]. Obviously, these metabolic processes will come with the release of extra heat in the cell and will inevitably create a problem for cell thermoregulation (see below). In addition, it has been shown that there is a connection between the level of human BHC and its ability to adapt to different climatic and geographical conditions. In particular, it has been shown that individuals with low BHC are better suited to the cold, while those with high BHC are better off in a hot climate [1].

Here it is necessary to briefly outline the essence of the concepts of cell thermoregulation (CT) and human BHC, that is, to clarify the concept of human BHC and where it came from. BHC is the physical manifestation of CT at the organism level. The essence of the CT is the elimination of the temperature difference between the nucleus and cytoplasm with the help of a dense layer of condensed chromatin (CC). The CC, the densest structure in an interphase cell, localized between the nucleus and cytoplasm, is made of chromosomal HRs. The density of CC packing, accordingly, its heat conductivity, depends on the quantity of chromosomal Q-HRs in its structure, which can affect its heat-conducting ability [1,3].

The transfer of thermal energy from the nucleus to the cytoplasm occurs through the CC layer, the density (and therefore heat conductivity) of which depends on the number of chromosomal Q-HRs in the nucleus. If an individual's genome has few chromosomal Q-HRs, it is expected that the nuclei of their cells will have trouble getting rid of excess metabolic heat. If the dissipation of excess heat from the nucleus is difficult or impossible due to insufficient CC density, this can lead to thermal death of cells, with all the negative consequences that has for the whole body. Maybe that is why older individuals in the population turned out to be the most vulnerable group, having the lowest BHC. From the same perspective, we can also explain why women made up the majority of

older adults who died from heat stress, since their genome lacks the Y chromosome, which contains the largest Q-HR block in the human karyotype.

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