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Review Article

Common Cold, Cell Thermoregulation, Heterochromatin and Body Heat Conductivity

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Abstract

Common cold (CC) is an infectious disease of the upper respiratory tract. CC is considered one of the most widespread human illnesses and causes enormous harm to health and the economy. In the United States alone, CC leads to 75–100 million physician visits annually, with a conservative cost estimate of $7.7 billion per year. Americans spend $2.9 billion on over-the-counter drugs and another $400 million on prescription medications for symptomatic relief. The etiology of CC is known — over 200 virus strains are implicated in the cause of the CC, while bacterial infection is detected in only 5% of cases. As for pathogenesis, it is generally accepted that cold exposure plays an important role in the development of CC. It is believed that cooling reduces tissue resistance to infectious agents and lowers the body's defense against infection. However, it is evident that only a proportion of the people who are exposed to cold will develop an infection, and this finding suggests that there are other underlying factors that may reduce resistance to CC. Such factors can include: a) the initiation mechanisms of CC; b) the CC sickness rate (children get sick more often than adults) and gender (males are more susceptible to CC than females, regardless of age); c) not all people develop CC during the cold season, and finally, d) CC affects only humans and apes. It is proposed that at the core of these factors may lie the features of cell thermoregulation, heterochromatin and heat conductivity of human body.

Kew Words: common cold; cell thermoregulation; chromosomal Q-heterochromatin; human body heat conductivity; susceptibility to common cold

Introduction

Common cold (CC) is an infectious disease of the upper respiratory tract. CC is considered one of the most widespread human illnesses and causes enormous harm to health and the economy. In the United States alone, CC leads to 75–100 million physician visits annually, with a conservative cost estimate of $7.7 billion per year. Americans spend $2.9 billion on over-the-counter drugs and another $400 million on prescription medications for symptomatic relief. The etiology of CC is known — over 200 virus strains are implicated in the cause of the common cold, while bacterial infection is detected in only 5% of cases. As for pathogenesis, it is generally accepted that cold exposure plays an important role in the development of CC. It is believed that cooling reduces tissue resistance to infectious agents and lowers the body's defense against infection. The data available suggest that exposure to cold increases the risk of developing upper respiratory tract infections; in addition, the longer the duration of exposure the higher the risk of infection. Although not all studies agree, most of the available evidence suggests that inhaled cold air, cooling of the body surface and cold stress induced by lowering the body temperature cause pathophysiological responses such as vasoconstriction in the respiratory tract mucosa and suppression of immune responses, which are responsible for increased susceptibility to infections. (see more [1,2]). However, it is evident that only a proportion of the people who are exposed to cold will develop an infection, and this finding suggests that there are other underlying factors that may reduce resistance to CC. We believe that such factors can include: a) the initiation mechanisms of CC; b) the CC sickness rate (children get sick more often than adults) and gender (males are more susceptible to CC than females, regardless of age); c) not all people develop CC during the cold season, and finally, d) CC affects only humans and apes.

Pathogenesis of common cold.

Existing hypotheses.

It should be mentioned at once that for many years the medical science studies the initiation mechanism of CC. Yet, much remains unclear. So far, there is not commonly accepted theory explaining the mechanism of CC development. Regarding the pathogenesis, it is generally accepted that the cold plays an important role in the development of CC. Принято считать, что that cooling causes circulatory disturbance and reduces vascular permeability. Changes in blood flow decreased vascular permeability cause

malnutrition of tissue and lowering its resistance against the infectious agents. Pathophysiological mechanisms of CC have been studied generally. In the initial period of the disease, the virus replicates in the input "gates of infection": the nose, nasopharynx, and larynx. The virus then enters the bloodstream and causes the symptoms of general intoxication: fever, headache, aching back and limbs. The immune response activation results in development by an organism of antibodies to the virus, whereby the blood is cleaned from it gradually and the symptoms of intoxication diminish [1].

It is believed that the inspiration of cold air causes a decrease in the temperature of the respiratory epithelium [3]. The latter is responsible for the decrease of mucociliary clearance and the local immune responses of the airway, i.e., it compromises phagocytic activity leading to increased susceptibility to infection. The data available suggest that exposure of the body surface or the upper airways to cold temperatures may contribute to the development of upper respiratory tract infections. The inhalation of cold air or cooling of a part of the body surface is associated with an increased risk of respiratory infections.

It was once believed that colds were caused by chilling the body. Nevertheless, despite its name and the common belief about the effects of cold, a cold is caused by infections. There is no evidence that one can catch a cold due to hypothermia, overheating, or exposure to cold or rainy weather [4-6], and some studies indicate that cold exposure does not affect incidence rates [7]. However, research has shown that viruses are the cause of illnesses, and cold itself does not affect the frequency or severity of illnesses [7-9]. The only known way to completely avoid catching a cold is to live in total isolation from the rest of humanity [10]. For example, researchers visiting the Arctic or Antarctic do not catch colds during their stay, despite the freezing temperatures and harsh conditions. But, they get very sick as soon as they start interacting with their companions [11].

Animal studies have also shown that low environmental temperatures affect the severity of experimentally produced infections [12]. In the light of the foregoing, it can be argued that the question remains outside the field of view of researchers: why CC affects only humans and apes? [13]. The fact that man and apes are reputed to be the only susceptible hosts to this disease. Easily managed laboratory animals such as rats, mice, rabbits, cats and dogs are said not to catch the disease, thus making laboratory studies very difficult [14].

Own hypothesis.

To explain the essence of our hypothesis, it is necessary to clarify a few concepts that are little known to the broader medical audience. We are talking about cell thermoregulation, chromosomal heterochromatin regions, and the heat conductivity of the human body.

Cell thermoregulation.

Cell thermoregulation (CT) refers to the process of dissipating excess metabolic heat outside the interphase cell. Since the temperature of the nucleus is higher than that of the cytoplasm, the cell uses a dense layer of condensed chromatin (CC) around the nucleus as a thermal conductor to release excess heat into the cytoplasm. The heat energy is then transferred to the intercellular fluid and further into the circulation system. In this sense, CT is a unidirectional flow of heat energy directed from the nucleus to the cytoplasm through the CC, which consists of chromosomal heterochromatin regions (HRs). The layer of CC around the nucleus is the densest and, accordingly, the most heat conductive structure in the interphase cell [15-18]. CT refers to the cell's ability to effectively equalize the temperature difference between different areas, primarily between the nucleus and cytoplasm. This ability of cells is determined by the quantitative and qualitative composition of chromosomal HRs in CC.

Chromosomal heterochromatin regions.

A fundamental feature of chromosomes in higher eukaryotes, including man, is the presence of two evolutionally consolidated types of genetic material: euchromatin and heterochromatin. Euchromatin, the conservative portion of the genome, contains transcribed structural genes, while heterochromatin, the variable portion of the genome, is predominantly composed of non-coding repeated DNA sequences. Heterochromatin is universally distributed in the chromosomes of all the eukaryotes - plants, animals and man, accounting for 10% to 60% of their genome. Chromosomal heterochromatin regions (HRs) account for about 15% - 20% of the human genome. To-date two types of chromosomal HRs are recognized: Q- and C-HRs [19-21]. There are several significant differences between them: C-HRs is found in the chromosomes of all the higher eukaryotes, while Q-HRs - only in man (Homo sapiens), the chimpanzee (Pan troglodytes) and gorilla (Gorilla gorilla) [20-22]. Chromosomal Q-HRs are subject to considerably greater quantitative variability in any population. Individuals differ in the number, size, and intensity of fluorescence of these specific chromosomal regions. In individuals of a population the number of Q-HRs usually ranges from 0 to 10 [17,23-25].

Quantitative variability of chromosomal Q-HRs in human populations has been studied in detail. Regarding the issue discussed here, it is important to know that:

1) the Q-heterochromatin on the Y chromosome is the largest in the human karyotype, and its average size is twice greater than all the Q-HRs on autosomes taken together, so the overall amount of Q-HRs in females is as rule lower than in males;

2) males in the population differ from each other in the size of the Q-heterochromatin segment of the Y chromosome;

3) in individuals of a population the number of Q-HRs on autosomes usually ranges from 0 to 10;

4) different age groups have different numbers of Q-HRs, the greatest number of Q-HRs is characteristic of neonates, while the lowest – of elderly subjects;

5) in the first days, weeks, months and years of life, ceteris paribus, among healthy children the infants often die with the greatest number of Q-HRs in genome;

6) individuals capable of successfully adapting themselves to the extreme high-altitude climate (e.g. mountaineers) and of the Far North (e.g. oil industry workers of polar Eastern Siberia) are characterized by extremely low amounts of Q-HRs in their genome;

7) individuals with a lower number of Q-HRs in their genome proved to be prone to alcoholism and obesity, while those with a greater amount of Q-HRs– to drug addiction (for more details see: [23-40]).

As is well known, heat conductivity (HC) is the transfer of energy from warmer parts of the body to cooler parts, which leads to the equalization of body temperature (the second law of thermodynamics). All substances have HC: gases, liquids, and solids. Unlike gases and liquids, convection is impossible in solids, so heat transfer occurs only through HC. The HC of the human body, as one of the types of physical characteristics of the human

body, has never been purposefully studied by anyone. In fact, there is nothing new in the very idea that the human body must have some HC [16]. It has been established that individuals in the human population differ in the number of chromosomal Q-HRs [23-39]. It has been shown that there is a direct relationship between the number of chromosomal Q-HRs and the level of the human body heat conductivity (BHC): the more Q-HR in the genome, the higher its BHC level. BHC is the cumulative effect of the CT process at the organism level. In other words, BHC is a phenotypic manifestation of CT, allowing an indirect assessment of the heat removal capacity of the cells of a given individual [16,41,42]. Human BHC refers to the body's ability to remove (dissipate) excess heat from the organism through CT mechanisms to the circulatory system. Human BHC is the cumulative effect of the vital activity of all cells in the body, aimed at equalizing the temperature difference between the nucleus and the cytoplasm. It should be noted that CT is not regulated by the known mechanisms of the organ-based physiological thermoregulation system (the hypothalamus, sweat glands, skin, and circulatory system), but is mainly governed by the second law of thermodynamics. Therefore, we believe that the task of physiological thermoregulation is not to regulate core body temperature, but to regulate the temperature of the blood circulating throughout the body in order to preserve the structural and functional integrity of cell membranes (for details see [15,16,41,42]).

Human core body temperature.

Apparently, the concept of distinguishing people from each other by BHC is not perceived in connection with the well-known fact that in norm, all individuals in the population have almost the same core body temperature [16]. Perhaps, for this reason, it is considered unreasonable to expect a wide variability of human BHC in population. However, it turns out that the very concept of core body temperature not only in humans, but also in homeothermic organisms in general, needs to be clarified, since core body temperature does not really correspond to its name [14,15].

We believe that the human body temperature measured by thermometers is not its important physical characteristics, as this value is relatively constant, adjustable and regulated by mechanisms of organ-based physiological thermoregulation. The general opinion of experts is as follows: "the analysis of thermal homeostasis in the human body and homeothermic animals. It is shown that the temperature in the internal tissues of the body (the core of the body) is high and relatively consistent because it is maintained via heat transfer through the blood flow" [43]. This statement needs clarification. The heat cannot transfer from the blood to the cells for the following reasons: 1) in homeothermic organisms, the temperature (T) in the cells is higher than in the circulating blood. The transfer of heat from circulation system to cells is impossible, because the second law of thermodynamics; 2) cells, with rare exceptions (endothelial cells lining the inner surface of blood vessels) are not in direct contact with blood; 3) the readings of thermometers reflect T only circulating blood, where the thermometry is performed, and not T inside the cells [18]. We cannot know T inside cells because of diversity of organelles in it. There are 230 types of cells functioning in the human body, which differ in their metabolic rate, cell cycle and the amount of heat they produce. In addition, there is no method that allows measuring cell T as a whole, since the existing ones can only work with organelles (nucleus, mitochondria, ribosomes, or ER) [14,16,43]. In the same way, we cannot speak about human core body temperature because of the heterogeneity of its constituent tissues and organs. The term human body temperature (core body temperature) lacks common sense, since thermometers reflect the T of circulating blood in the capillaries of the part of the body where the thermometry is performed, but not the cells. Since it is impossible to measure the temperature of cells and bodies, the thermophysical characteristics of a human can still be judged by the thermal conductivity of their body [18].

It is accepted that the main task of thermoregulation is to maintain a relatively constant core body temperature, meaning cells, tissues and organs, to ensure their normal function. From the point of view of physiologists, this is accomplished by the transfer of thermal energy from circulating blood to cells [43], a possibility of which we question. We believe that the task of thermoregulation is not the preservation of optimal T in the body, but the preservation of structural and functional integrity of cell membranes. As indicated above, cellular T cannot be affected by heat from the circulation system. In addition, the generally accepted opinion that maintaining a temperature range of 35-41 ºC is acceptable, for example, for the normal functioning of enzymes may be controversial. This is evidenced by the following facts: 1) it is now established that the temperature of mitochondria is about 50 ºC [44]; 2) enzymes can work even at T 100 ºC and higher (in thermophilic bacteria); 3) the temperature inside cells is not regulated by generally recognized physiological mechanisms, i.e. by transfer of heat from circulating blood [43], but is determined by the laws of physics. Cell membranes are known to be very sensitive to T fluctuations and their structural and functional integrity depends on it (T). Since the T of the cell in homeothermic organisms is always higher than the T of circulating blood, it is to be expected that cell membranes are under the constant and directed action of thermal energy from the inner side of the cells. Therefore, the only protection of cell membranes from dangerous overheating is the circulating blood, whose temperature is lower than the cell temperature and is regulated by the organ-based (the hypothalamus, the sweat glands, the skin, and the circulatory system) thermoregulatory system. Since intracellular T is unregulated, the task of cell thermoregulation is only to remove excess metabolic heat outside the cell in a timely manner.

The essence of our hypothesis.

We believe that in the pathogenesis of CC, the human BHC factor plays an important role, which is characterized by wide variability in the population. With the help of this factor, it is possible to rationally explain certain features of the pathogenesis of CC, such as: a) the initiation mechanisms of CC; b) the CC sickness rate (children get sick more often than adults) and gender (males are more susceptible to CC than females, regardless of age); c) not all people develop CC during the cold season; and d) CC affects only humans and apes. Let us start with the initiation of the common cold, that is, how viruses or bacteria penetrate the cells of the upper respiratory tract. Existing hypotheses suggest that cooling causes circulatory disturbance and reduces vascular permeability. Changes in blood flow decreased vascular permeability cause malnutrition of tissue and lowering its resistance against the infectious agents. The virus then enters the bloodstream and causes the symptoms of general intoxication: fever, headache, aching back and limbs [1,3,4].

We believe that the disruption of the functional and structural integrity of cell membranes plays an important role here. From our point of view, the biological significance of maintaining the temperature of circulating blood in homeothermic organisms within narrow limits (36-41 °C) lies in preserving the normal functioning of cell membranes, which are sensitive to temperature changes: at high temperatures, they become 'too fluid,' and at low temperatures, 'too rigid.' For example, in cold conditions, the cell membranes of the epithelium covering the surfaces of the upper respiratory tract become less mobile, allowing viral particles to attach and enter the cells. There is an additional factor to consider. As is known, the main proportion

of upper respiratory tract illnesses is caused by rhinoviruses, which reproduce best at a temperature of about 33 °C, which is possibly close to the temperature of the epithelium on the surface of the upper respiratory tract during prolonged inhalation of cold air. 9The CC sickness rate (children get sick more often than adults) and gender (males are more susceptible to CC than females, regardless of age). For example, on average, children catch a cold about 6 to 8 times a year, adults 2 to 4 times, and elderly people less than once [45]. This can be reasonably explained by the characteristics of chromosomal Q-HR distribution: 1) different age groups have different numbers of Q-HRs, the greatest number of Q-HRs is characteristic of neonates, while the lowest – of elderly subjects; 2) The Q-HR on the Y chromosome is the largest in the human karyotype, and its average size is twice greater than all the Q-HRs on autosomes taken together, so the overall amount of Q-HRs in females is as rule lower than in males.

As for 'not all people develop CC during the cold season,' it can be explained by the fact that human body heat conductivity level depends mainly on the amount of chromosomal Q-heterochromatin in his genome. Individuals in population differ in the number of chromosomal Q-HRs (the number of Q-HRs in human genome usually ranges from 0 to 10). An interesting fact is that CC affects only humans and apes, apparently related to: 1) chromosomal Q-heterochromatin is found only in man, the chimpanzee and gorilla, while C-heterochromatin - is found in the chromosomes of all the higher eukaryotes; 2) curiously enough monkeys, unlike the apes, do not seem to be liable to catch common cold. If our hypothesis is correct, then we have reason to expect that humans and the two higher primates should differ from other homeothermic animals by having the highest level of BHC. Since individuals in a population differ from each other in their BHC, the level of which depends on the amount of chromosomal Q-HRs in the genome, we believe that human CC susceptibility and frequency of disease probably is determined on how quickly and deeply his body is cooled under the influence of cold. If our hypothesis is correct, then we can expect that individuals with high BHC would be more susceptible to CC and vice versa. It is the level of human BHC that determines how quickly and deeply his body becomes cool at cold, and ultimately the CC susceptibility of an organism.

Concluding remarks.

The biological role of "redundant" DNA in eukaryotes, which consists of repeating sequences of nucleotides and does not encode proteins and enzymes known to science, remains still unclear. Part of this DNA in the interphase cell is complexed with proteins into highly compacted structures, designated as condensed chromatin, and on the metaphase chromosomes it manifests itself as heterochromatin regions. The purpose of this note to show once again that the biological role of non-coding DNAs, which form a dense layer of condensed chromatin around the cell nucleus in an interphase cell, is their participation in maintaining intracellular temperature homeostasis. Since accurate measurements of the temperature of different parts of the cell in vivo are not yet possible to verify the existence of thermoregulation at the cellular level, indirect methods for studying cell thermoregulation by evaluating the heat conductivity of the body and heterochromatin regions of chromosomes in humans are proposed. The heat conductivity as one of the types of physical characteristics of the human body has never been purposefully studied by anyone, although from a physical point of view it is impossible to deny this possibility. It is assumed that the phenotypic manifestation the part of "redundant" DNA at the level of the human body is the level of heat conductivity of his body, the role of which is to be evaluated in norm and pathology.

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