

Human Body Heat Conductivity in norm and pathology : A review



Original Research Article

ISSN : 2456-1045 (Online)
(ICV-MDS/Impact Value): 63.78
(GIF) Impact Factor: 4.126
Publishing Copyright @ International Journal Foundation
Journal Code: ARJMD/MDS/V-32.0/I-1/C-3/DEC-2018
Category : MEDICAL SCIENCE
Volume : 32.0 / Chapter- III/ Issue -1 (DECEMBER-2018)
Journal Website: www.journalresearchijf.com
Paper Received: 13.12.2018
Paper Accepted: 24.12.2018
Date of Publication: 05-01-2019
Page: 12-21



Name of the Author:

A.I. Ibraimov¹

¹ *Laboratory of Human Genetics, National Center of Cardiology and Internal Medicine, Bishkek, 720040, Kyrgyzstan.*

Citation of the Article

Ibraimov A.I. (2018) Human Body Heat Conductivity in norm and pathology : A review.; *Advance Research Journal of Multidisciplinary Discoveries*.32(3)pp. 12-21

ABSTRACT

Heterochromatin is universally distributed in the chromosome of all the eukaryotes, accounting for 10% to 60% of their genome. There are two types of heterochromatin: C- and Q-heterochromatin. C-heterochromatin is found in the genome of all higher eukaryotes, while Q-heterochromatin is only in the genome of three higher primates (*Homo sapiens*, *Pan troglodytes* and *Gorilla gorilla*). However, there is a fundamental difference between them: quantitative variability of Q-heterochromatin exists only in human populations. Despite the fact that heterochromatin regions (HRs) has been studied since the 20s of the past century, its biological role remains unclear. An attempt is made to justify the view of possible participation of HRs in cell thermoregulation. HRs, being the densest domains in a cell, apparently conducts heat between the nucleus and cytoplasm when there is a difference in temperature between them. It turned out that there is a link between the amount of Q-heterochromatin and level of human body heat conductivity (BHC): the larger the amount of Q-heterochromatin, the higher of human BHC. The data on the influence of human BHC on its life activity in norm and pathology are presented.

KEYWORDS:

human body heat conductivity; cell thermoregulation; chromosomal Q-heterochromatin; human adaptation.

I. INTRODUCTION

Thermal conductivity is the process of heat energy propagation by direct contact of bodies having different temperatures. According to the second law of thermodynamics, the spontaneous process of heat transfer in space occurs under the influence of the temperature difference and is directed towards a reduction in temperature. In all materials, heat is transferred through the transfer of energy by micro particles. Molecules, atoms, electrons, and other micro particles that make up the material move at speeds proportional to their temperature. Fast-moving micro particles give their energy to more slowly ones, thus transferring heat from the zone with high to the zone with low temperature leads to equalization of body temperature.

Temperature has a fundamental influence in all chemical and biochemical reactions. It influences reaction rates, equilibrium amounts, viscosity, solubility, molecular arrangements and numerous other parameters. Temperature is important for physiological processes as well as cell maintenance and function. The body temperature is maintained at a relatively constant level because of the balance, which exists between heat production and heat loss. If there were no heat loss even in the resting subject would produce sufficient heat to raise the body temperature by 10 °C every hour; a 69-kg man produces 70 calories of heat per hour due to the basal metabolism.

It is commonly assumed that the main elements of the organ-based physiological thermoregulation are known, and at present the efforts are directed at the study of their complex interactions at the cell and molecular level (Blatties, 1997). However our long experience of studying the effects of one of the types of constitutive heterochromatin in the human genome – chromosomal Q-heterochromatin regions – inclines to the idea that, possibly, in the process of keeping the temperature homeostasis in the organism one more element participates, namely the heat conductivity of the cellular part of a body (Ibraimov, 2003, 2004).

II. THE ORIGIN OF THE IDEA OF HUMAN BODY HEAT CONDUCTIVITY.

It is found that non-coding part of human genome makes about 98% of cell nucleus DNA. Approximately 15-20% of this non-coding part of human DNA is constitutive heterochromatin (John, 1988). There are two types of constitutive heterochromatin: C- and Q-heterochromatin. Human chromosomes possess two types of constitutive heterochromatin: C- and Q-heterochromatin (Caspersson et al., 1970; Arrighi, Hsu, 1971). Chromosomal C-heterochromatin regions (C-HRs) are found in the genome of all higher eukaryotes, while Q-heterochromatin regions (Q-HRs) are only in the genome of three higher primates (*Homo sapiens*, *Pan troglodytes* and *Gorilla gorilla*) (Pearson, 1973; 1977; Verma, 1988). However, there is a fundamental difference between them: quantitative variability of chromosomal Q-HRs in the genome exists only in human populations (Paris Conference, 1971; ISCN, 1978).

Despite the fact that chromosomal heterochromatin regions (HRs) has been studied since the 20s of the past century, its biological role remains unclear on the whole. This circumstance is also reflected in variety of hypothesis, none of which is supported with necessary experimental data (for details see: John, 1988; Ibraimov, Mirrakhimov, 1985; Prokofyeva-Belgovskaya, 1986; Elgin, Grewal, 2003; Craig, 2004; Dimitri et al., 2004; Bhasin, 2007; Ibraimov, 2015). Moreover, all these hypotheses generally refer to C-HRs, but not to Q-HRs.

Based on our investigations of chromosomal HRs variability in human populations, as well as on the analysis of existing literary data on the condensed chromatin (CC), structure of interphase nucleus and redundant DNA in the genome of higher eukaryotes, an attempt is made to justify the view of possible participation of CC in cell thermoregulation (Ibraimov 2003). CC,

being the densest domains in a cell, apparently conducts heat between the nucleus and cytoplasm when there is a difference in temperature between them. The assumed heat conductivity effect of CC is stipulated by its principal features: a condensed state during the interphase, association with the lamina and the inner nuclear membrane, replication at the end of the S period of a cell cycle, formation of the chromocenter, genetic inertness, and wide variability in the quantitative contents both within and between species (Ibraimov 2003, 2004).

Chromosomal HRs is localized in the interphase nucleus along the periphery of nucleus and is in close association with nucleus membrane. The layer is often so dense that it can itself form the quite rigid structure able to maintain spatial organization of nucleus (Comings, Okada 1970). Chromosomal HRs is a basis of periphery layer of CC chromatin in cells (Lampert, 1971; Bostock, Sumner 1978; Prokofyeva-Belgovskaya 1986).

Chromosomes have both internal (repair, recombination, rearrangement, modification, restriction) and external (replication, transcription, packaging, organized movement) molecular activities, which are accompanied, inter alia, by some heat output. If for any reasons the temperature in a nucleus begins to exceed that in cytoplasm there is a need for dissipation of surplus heat outside the nucleus. To do this the nucleus has two options: increasing its volume or increasing the heat conductivity of the nuclear membrane. The first option is limited for obvious reasons. The second option is the more promising one should the heat conductivity of the nuclear membrane be increased somehow. Since the nuclear envelope consists of double-membrane extension of the rough endoplasmic reticulum, the nuclear membrane cannot essentially change its structure. But it is necessary to remove the surplus heat from the nucleus somehow. Since the proposed idea is based on cell phenomena, apparently nature 'found' a very simple and effective solution: it increased its heat conductivity through compression of the internal layer of the nuclear membrane by CC.

Certainly, cell thermoregulation hypothesis should be checked *in vivo* on the cell level. But we have not had such opportunity till present. Nevertheless, we have checked this hypothesis on the level of human organism assuming that CT is the basis for heat conductivity of whole cell part of body. Through trial and error we have identified areas of the body and the thermal load mode, which allows to roughly estimating the level of human body heat conductivity (BHC): high, medium and low. Results obtained show that individuals in population truly differ from each other in BHC and its level depends on the amount of chromosomal Q-HRs in human genome (for more details see: Ibraimov et al., 2014).

But some questions still remain unsolved including methodical ones in studying the human BHC variability. In particular, it is still not possible to develop a method to accurately measure the BHC of human, as it is done on homogeneous non-living objects by thermal physicists.

III. THE METHOD OF DETERMINING THE THERMAL CONDUCTIVITY OF THE HUMAN BODY.

Virtually, there is nothing new in the idea that the body of the human should possess some heat conductivity. Nevertheless, it has not drawn the attention of nor physicists, neither physiologists for the present as the important physical characteristic of a human body. Apparently, it is connected with known physical heterogeneity (in sense, density) of a human body. Probably that's why; we did not manage to find in the literature not only a special method, but even any attempt to estimate body heat conductivity (BHC) of live organisms.

In thermo physics, measurement of heat conductivity of solid bodies (f.e. metal) is carried out by determination of heat conductivity coefficient by a calorimetric method. Transfer of heat occurs through a metal rod, the ends of which are placed in a calorimeter with the water taken at temperatures T_1 and T_2 ($T_1 > T_2$). It is obvious that direct transfer of a method of measurement of the heat conductivity, applied in thermo physics is unacceptable to a human body both for technical and ethical reasons. However we have tried to approximate to the decision of this problem indirectly, by an estimation of part of a human body. For this purpose, we had to modify the standard technique of physicists so that it was acceptable to the human.

As is known inorganic and organic bodies have different mechanisms of equalization of temperature (T): in the first case it is carried out through heat conductivity (HC), in the second, besides HC, liquids circulating on all body (blood, lymph, saps) participate. It is obvious that wide variability of BHC, found out by us, in human population cannot be connected with T of blood, because its T is under the strict control of the central (hypothalamus) organ based system of physiological thermoregulation. Therefore, in our opinion it was highly probable that the possible reason of differences of individuals in heat conductivity of their bodies in a population could be any other physical factor. Under the latter, we meant human body heat conductivity (Ibraimov, Tabaldiev, 2007; Ibraimov et al., 2014). However the problem lies in an objective estimation of human BHC.

Since the literature does not have a special method for measuring human BHC, we could only use the method of trial and error to find the areas of the body, which at least allow to roughly estimating the transfer of thermal energy from environment into body and from one body part to another. For example, hand is selected from ethical and technical considerations (Ibraimov, Tabaldiev, 2007; Ibraimov et al., 2014).

Why hands? Certainly, hand tissues are not that part of the organism which makes it possible to judge unambiguously about the heat conductivity of the whole cell part of a human body. However, some physiological data testify the favor of our choice of the part of a body. For example, Aschoff (1958) has recognized that the distal parts of the body may be more important heat loss effectors of the shell. The palmer sides of the hands, plantar sides of the feet, ears, lips, cheeks and nose tip contain arteriovenous anastomoses (reviewed in: Van Someren et al., 2002). Finally, our own observations have shown that only temperature of a palm of hand has shown wide variability in population among available for measure of heat conducting parts of human body (Ibraimov, Tabaldiev, 2007). We see this fact as the most important one, as it is known that nuclear, rectal or armpit temperature is the same for all physically healthy people at rest and at room temperature.

The principle of the method is very simple. To evaluate human BHC it is necessary to: a) create a temperature gradient between a certain part of the human body and the environment. To this end, the left hand is immersed in a water bath, where the water temperature is set at 9 °C higher than the temperature of the left palm of the individual under study; b) for 20 minutes, the temperature of the right palm is measured every minute (the transition of thermal energy from one part of the body to another) using a non-contact thermometer used in medicine; c) the time of onset of the temperature maximum on the right palm (rate of transition of thermal energy) shall be recorded.

Our empirical experience has shown that to create a temperature gradient between the human body and the environment T of “hot” water in a water bath should be raised ~ 9 °C. Certainly, no fundamental physical constant of a human body is at the back of this number. But, it is also undesirable to increase the T of ‘hot’ water more than 9°C. In that case, we could face with denaturation of proteins in cells of individuals whose T of palms is close to that of armpit. In addition, some individuals experienced discomfort when their hands were immersed in water, where T of “hot” water was above ~9 °C.

After years of searching for an acceptable method to evaluate human BHC, we settled on this option. BHC estimation of individuals should be conducted indoors at a temperature of 20 °C – 22 °C. At first the individual’s oral cavity and palms temperatures were measured. Temperatures of the palm were measured using a pyrometer for non-contact temperature measurement of the human body (f.e., electronic medical infrared thermometer F-1000, B.Well, UK). A medical electronic thermometer WT-03 of the same company was used to measure the temperature of the oral cavity. Measuring the left palm temperature was necessary for preparation of ‘hot’ water. ‘Hot’ water was prepared by adding of number nine to the thermometer reading. For instance, if an individual's palm temperature was 31.0°C, then ‘hot’ water temperature for his hand should be 40.0°C. ‘Hot’ water was prepared to create a thermal gradient between the left hand and its surrounding.

The studied person sits down on a chair with his body upright, head is raised, and hands hang down naturally on both sides of his body and muscles are relaxed. Then the examinee plunges slowly his left hand up to the wrist in water bath (volume ~ 6.0 liter), which maintains water temperatures at a given mode until the end of the experiment. Water bath is placed against shin. During the heat conductivity measuring which takes 20 minutes, the individual under test should not divert his attention away, keep the hand in water and not press it against the walls of the water bath. Throughout the whole thermal load the right palm temperatures were minutely measured. Measuring the right palm temperature was necessary for determining the amount and rate of thermal energy passed from the wrist of left hand to the wrist of right hand. The assessment of the BHC is carried out as follows: if the temperature peak occurred within the first 5 minutes, it is assumed that such an individual has a high heat-conducting body; from 6 to 10 minutes middle-, from 11 minutes and above as a low heat-conducting body (for more details see, Ibraimov et al., 2014).

Relationship between the amount of chromosomal Q-HRs and the temperature difference between the surfaces of the right palm and the oral cavity at rest detected. Namely, the more the chromosomal Q-HRs in the human genome, the smaller the T difference between the oral cavity and the surface of the right palm, and *vice versa* (Ibraimov et al., 2014).

How do we interpret the data? Statistically significant relation between the number of chromosomal Q-HRs in the genome and the T difference between the oral cavity and the right palm at rest may also characterize the heat conducting ability of the human body, the smaller the T difference, the higher the BHC, and *vice versa*. We believe that the smaller T difference between the oral cavity and the palm reflects the high thermal conductivity ability of the body, in a sense that such an organism equalizes the T difference between the different parts of the body more effectively, thereby successfully avoiding overheating of the organism in hot conditions. Temperature of the right palm at rest, presumably, also reflects the level of BHC; individuals with high T of palm may have higher BHC, and *vice versa*.

We believe that the time of occurrence of the peak temperature on the right palm reflects the rate of conductivity, while the value of T of the right palm surface at that moment seems to reflect the quantity of thermal energy in the individual's body. If the peak temperature on the surface of the palm occurs in the first five minutes after the thermal load, then such an individual is considered as a person with high BHC, and *vice versa*. In other words, we believe that a person with high BHC conducts heat through the body quicker and eliminate its excessive quantity through body shell quicker as well to maintain a constant level of inner body temperature.

ADVANCE RESEARCH JOURNAL OF MULTIDISCIPLINARY DISCOVERIES

And finally, is it possible to evaluate BHC of human without determining the number of chromosomal Q-HRs in the genome? We believe that this is possible. For this it is necessary to know: a) the time of occurrence of peak temperature on the surface of the right palm in the controlled thermal load, b) the difference of T between the oral cavity and the surface of the right palm at rest, and c) T of the surface of the right palm at rest. If: a) the peak temperature occurs in the first five minutes after a thermal load; b) the difference of T between the oral cavity and the right palm does not exceed 1.0°C , and c) T of the surface of the right palm at rest is above 36.0°C , then it is possible to expect that such a person will have high BHC, and *vice versa*.

Thus, to indirectly assess the rate of transition of thermal energy from the nucleus to the cytoplasm in humans, we use the method of assessing the BHC at the level of the whole organism. For practical purposes, to assess the level of human BHC may be not necessarily measure the rate of transition of thermal energy from the nucleus to the cytoplasm in its cells. Methodically, it may even be justified. For example, to assess the temperature of the human body, physicians are limited to measuring the temperature of the oral cavity or armpit. To solve more specific problems determine the temperature of the rectal temperature and/or eardrum. Even less often resort to direct measurement of the temperature of the internal organs. And not because it is an invasive method or technically complex, but because the temperatures of the internal organs and various parts inside the body (for example, limbs) are different during the day and time of year. To simplify the task, scientists agreed to measure the temperature of certain areas of the outer body.

There are no direct data on the temperature of different parts of the cell nucleus measured *in vivo*. However, it is difficult to expect that the nucleus temperature will be the same at all its sites. Obviously, the biochemical activity in nucleoli or chromocenters will be higher than in the rest of the nucleus. In addition, the nucleus is a heterogeneous physical mass, and a deeply structured organelle. Therefore, prior to the emergence of direct methods to determine the rate of transition of thermal energy from the nucleus to the cytoplasm could be limited to the study of BHC at the level of the organism.

IV. MAIN RESULTS

Thereby, as a whole our results show that on the population level: a) individuals in a population differ from each other on the level of BHC; b) on the average BHC of males is higher than that of females; c) individuals differ in BHC from different age groups, on the average human BHC level is steadily changed decreasing with age; d) natives of low altitude regions of southern latitude differ on the average by higher BHC than population of high mountains and northern latitude; e) weight, height, types of body constitution (normosthenic, asthenic and hypersthenic), pulse rate and level of arterial pressure do not effect on the variability of BHC in population (Ibraimov, Tabaldiev, 2007; Ibraimov et al., 2010a,b; 2014).

It is interesting that these results meet the data obtained during investigation of quantitative variability of chromosomal Q-HRs in human population, namely: a) individuals in a population differ from each other in the number of chromosomal Q-HRs in the genome; b) as a rule, amount of chromosomal Q-HRs in male karyotype is higher than in female one on the population level; c) different age groups have different amount chromosomal Q-HRs: the greatest number of Q-HRs is characteristic of neonates, while the lowest – of elderly subjects; d) a consistent interpopulation differences in the quantitative amount of chromosomal Q-HRs in their genome were established. These differences proved to be related to features of the ecological environment of the place of permanent residence, and not to their racial and ethnic composition. The amount of chromosomal Q-HRs in the population genome tend to decrease from southern geographical latitudes to northern ones, and from low-altitude to high-altitude ones (Buckton et al., 1976; Lubs et

al., 1977; Al-Nassar et al., 1981; Ibraimov, Mirrakhimov, 1982 a, b, c; 1985; Ibraimov, 1993; 2015; 2017; Ibraimov and Karagulova, 2006; Ibraimov et al., 1982; 1986; 1990; 1991; 1997; 2013; 2014; Stanyon et al., 1988; Kalz et al. 2005; Decsey et al., 2006).

V. BHC AND HUMAN ADAPTATION.

Human uniqueness in addition to all his known characteristics is that he is the only who managed to populate the whole Earth surface including such extreme areas as Far North and high altitudes remaining single tropic biological species. Moreover, all this took place in a short period of time (around 30,000 - 50,000 years), an unprecedented fact in life evolution (Stringer 1996).

It is known that of all physical environmental factors able to influence life, temperature is the most substantial. Role of temperature in biological life is obvious. And its highest form, mammals are able to maintain relatively permanent body temperature keeping high level of metabolism.

It is generally considered that the human is well adapted to hot climate. Probably this is connected with the fact that the human is a biological species developed in tropical climate of East Africa. Unlike many animal species, man is unstable to live in an extreme cold environment. He is basically a tropical homoiotherm. However, due to various reasons, human populations have to live under conditions of low or high environmental temperature (T) where maintaining the temperature homeostasis is especially difficult. Naturally, all three effector thermoregulating systems mobilize: heat production, heat loss and thermoregulatory behavior. Though being important, they cannot be effective at long-term perspective. We suppose that *H. sapiens*, besides those inherent in all mammals possesses an additional but very fine and simple mechanism of thermoregulation. In the present case, in order to preserve temperature homeostasis under different environmental conditions, in addition to physiological, behavioral and biochemical mechanisms such as wide intra population variability by BHC was used (Ibraimov, 1993, 2003, 2004; Ibraimov, Tabaldiev, 2007). Possibly, for the *H. sapiens*, BHC diversity is necessary because no single genotype can possess a superior adaptadness in all environments.

On the whole, we see efforts for maintaining temperature homeostasis under conditions different from climate of the Eastern Africa as follows: 1) an individual with less chromosomal Q-HRs in the North maintain more effectively temperature homeostasis in organism because of low BHC, permitting to preserve additional amount of produced heat in organism longer and slow down the body cooling rate from external cold; 2) an individual with high BHC in the North, constantly losing additional amount of metabolic heat through conduction which is necessary for organism in terms of cold climate and exposing to relatively fast cooling because of cold, has to produce larger amount of heat and/or consume more high-calorie food for heat production, which is not always simple and healthy; 3) an individual with low BHC in the South (where environment temperature is higher than body temperature) besides his own internal heat production receives additional heat from environment by means of conduction, which, as it is known, is not used in useful physiological work. That is why these individuals' bodies overheat faster and they have to return heat surplus (through sweating, polypnoe, forced rest, behavioral reactions and etc.) to environment at the cost of significant decrease of physical and mental activities that finally negatively influences on their adaptation to hot climate; 4) an individual with big amount of Q-HRs in genome in the South having body with high thermal conductivity perhaps adapts better to high temperature of environment, more effectively leveling temperature differences in different parts of the body and faster directing surplus heat flow from organism to environment, including the way of heat radiation.

ADVANCE RESEARCH JOURNAL OF MULTIDISCIPLINARY DISCOVERIES

Generally speaking, we wish to state that individuals equalize better and faster the T difference between different parts of body with high BHC and *vice versa*. If this is true then it would have found a simple explanation, for example, for the known resistance of South natives to high temperature of environment. Namely, they effectively equalize T difference in different parts of bodies and faster take out surplus heat in the environment due to high heat conductivity of their bodies. At that rate, aboriginals of the Far North or high altitudes could better and longer maintain the metabolic heat in the body due to low heat conductivity of their bodies with all ensuing consequences. The same way it might have been explained why do males endure better the heat load than females and the latter are more stable to cold than males.

Taking into consideration the mentioned all above, it can be explained why the amount of chromosomal Q-HRs is greater in the genome of newborns, then in senior age groups (Buckton et al. 1976; Ibraimov, Karagulova, 2006a), and the same chromosomal material is found in greater quantity in the genomes of infants died during first weeks, months, and years of their life (Ibraimov, Karagulova, 2006b). Prevalence of people with lesser quantity of Q-HRs in the genome in senior people groups may be connected with negative selection of individuals with greater amount of chromosomal Q-HRs during first years of their life. As it is well-known, infants' ratio of body surface to body capacity is higher than adults' ratio. When one more physical factor (high BHC) superimposes on this, then these infants are more vulnerable to colds and their consequences.

Our data on the temperature difference between the oral cavity and the palm could explain the data obtained in the frame of other research programs. Thus, the average difference between the oral and axillary temperatures of Indian children aged 6 - 12 was found to be only 0.1 °C (standard deviation 0.2 °C) (Chatuverdi et al., 2006) and the mean difference in Maltese children aged 4 - 14 between oral and axillary temperature was 0.56 °C (Quintana, 2004). These observations do not yet have a rational explanation. As part of our hypothesis (of a possible link between the number of Q-HRs and level of human BHC) these data could be explained by the fact that the amount of chromosomal Q-HRs in the genome of populations of India is significantly greater than that of the inhabitants of Europe (Ibraimov et al., 1997; Kalz et al., 2005).

We have also demonstrated that the natives of India are characterized by high levels of BHC, compared with the indigenous people of Central Asia (Ibraimov et al., 2010a). Indian peninsula is known for its hot climate, where the maintenance of temperature homeostasis poses serious stresses for human body. Assuming our hypothesis - the larger the number of chromosomal Q-HRs, the higher the heat-conducting ability of the human body - the low T difference between the oral cavity and axillary among Indian children could be explained by the presumed selective value of the amount of Q-heterochromatin in human adaptation to hot climate (for details see, Ibraimov, 2010). This, in turn, means that the body of Indian children has higher thermal conductivity than their Maltese counterparts, allowing them to better eliminate excess thermal energy to the environment and more effectively maintain the T difference between the different parts of the body.

Measuring the temperature of the oral cavity, we tried to learn core temperature change in the human body in the course of our experiment. It turned out that the temperature of the oral cavity does not change significantly. This may indicate that the mechanisms of physiological thermoregulation of human are developed enough so the thermal load equal to ours cannot significantly influence on the level of its core temperature.

Nevertheless, the rapid (on an evolutionary scale) and effective mastering of all the oxykumene by man is indeed a unique phenomenon, and this makes one ponder over the fact that here possibly were involved not structural genes but some mobile, non conservative part of the genome. Thus, our data suggest that

for adaptation to cold *H. sapiens* apparently used the Q-heterochromatin part of his genome (Ibraimov et al., 1982, 1986, 1990, 1991, 1997; Ibraimov, Mirrakhimov, 1982 a, b, c, 1985). However, we can hardly imagine as yet how this genetically inert material could be used in the adaptation of man to cold. Therefore, in order to relate in some way the possible mechanism of the influence of chromosomal Q-HRs to man's vital activity, including his existence under conditions of cold and hot climate, we were compelled to admit first of all that chromosomal Q-HRs in composition of CC have some heat conductive effect in cell (Ibraimov, 2003, 2004; 2015; 2017).

We suppose that *H. sapiens*, besides those inherent in all mammals possesses an additional but very fine and simple mechanism of thermoregulation. In the present case, in order to preserve temperature homeostasis under different environmental conditions, in addition to physiological, behavioral and biochemical mechanisms such as wide variability by BHC was used (Ibraimov, 1993, 2003). Possibly, for the *H. sapiens* BHC diversity is necessary because no single genotype can possess a superior adaptability in all environments.

It generally considered that the *H. sapiens* is characterized by the highest physiological plasticity, though there are no concrete data to justify availability in a man a special physiological mechanism of thermoregulation, which differs from other mammals. Of course, the mind of a man is the best creation of evolution. However for existence in conditions of high altitude hypoxia even the modern human cannot oppose anything essential, invented by his high intelligence. Nevertheless, only very few doubt that a man possesses the highest physiological plasticity.

Apparently, most likely, when speaking about this phenomenon the fact of inhabiting very different climatogeographical provinces by man is meant. But we assert that the basis of high physiological plasticity of *H. sapiens*, is possibly a wide quantitative variability of chromosomal Q-HRs in population, which through the change of the CC physical density in a cell exert modifying influence upon the level of heat conductivity of the whole human body.

Evidently, in reality one and the same person does not possess equally good adaptation to heat, cold and high altitude hypoxia. However, in any human population there are individuals able to efficient adaptation either to tropical climate, Far North, or high altitude conditions. Only in this sense it should be understood that a man as a biological species, but not as an individual, can adapt to heat, cold or high mountain hypoxia.

And as we assume, it is possible that in the basis of such high physiological plasticity of *H. sapiens*, in addition, there is a wide variability of his BHC in population. Really, selection affects not an individual, but the local population (Mayr, 1970). Thus we believe that: (1) human bodies in population significantly differ from each other, in addition, by heat conductivity; (2) organ-based physiological thermoregulation in a man is realized in different physical conditions in form of different BHC.

VI. BHC AND SPORT

The example from the modern sport life can better illustrate our understanding of BHC role. More and more countries situated at southern latitudes have started taking part in the world sport movement lately. The most notable in this process is that, natives of this region achieve great success in sports, requiring (in addition to other factors) effective heat-loss (football, professional boxing and marathon race). While sportsmen from northern latitudes prevail in water and winter sports and also in mountaineering (Ibraimov et al., 1990, 1991).

It is ascertained that natives of southern latitudes have more chromosomal Q-HRs in their genome (Lubs et al., 1977; Ibraimov, Mirrakhimov, 1982 b, c, d, 1985; Ibraimov et al., 1997; Kalz et al., 2005). Since southerners bodies, as we think, have relatively high heat conductivity (Ibraimov, Tabaldiev, 2007) it is not surprising, that they are successful in sports, which require effective heat-loss. Indeed, a sportsman with high heat conductivity cannot make much progress in water sports due to the fact that their body cools rapidly. However, this sportsman can be more successful in sports which require effective heat-loss.

Comparative tests for endurance of the “whites” and “blacks” to physical load in conditions of heat and high humidity demonstrate superiority of Negroes even over those “whites” who are used to working in such conditions. On the other hand, the experience of war in Korea showed that frostbite occurs much more often with Negro soldiers than with the “whites” (Folk, 1974). Long-term experience of the Indian medical officers in the Himalayas shows that the South Indians are physiologically more susceptible as compared to Gorkhas and North Indians under identical environmental conditions and that the high altitude population is more resistant to cold injures. It was shown that South Indians are more susceptible to frostbite than other ethnic groups of this country (Mathew, 1992).

VII. BHC AND HUMAN PATHOLOGY

Since wide quantitative variability of chromosomal Q-HRs was detected only in human population but alimentary obesity, alcoholism and drug addiction relate to the group of "purely human being pathology" we thought that it would be quite logically to study peculiarities of BHC of such diseased individuals. It seemed intuitively that such research would be important because these diseases in one way or other connect with thermo regulation.

Peculiarities of individual's BHC suffering from alimentary form of obesity, alcoholism, drug addiction as well as healthy individuals were studied with the method of calorimetry. It was established that patients differ with significantly low BHC as opposed to control sample. Among patients, drug addicts had the highest BHC, then alcoholics and individuals suffer from obesity. Obtained data confirm the assumption that possibly there is certain relationship between the level of human BHC and vulnerability to "diseases of civilization" (Ibraimov et al., 2010a,b).

Alcoholism and drug addiction are a purely human pathology. Despite the obviousness of etiology, the pathogenesis of the development of these diseases is still unclear, although there never was a lack of hypotheses and theories. Therefore, not aspiring to originality, we suppose that the role of chromosomal Q-HRs in the genome cannot be fully denied. As we have shown, in the genome of patients abusing strong alcoholic beverages the amount of chromosomal Q-HRs is very small; while in drug addicts it is on the contrary great (Ibraimov, 2016b). The possible role of BHC in these situations seem to us to be as follows: frequency of taking strong alcoholic drinks has a trend for increase by latitudes (from South to North), and by altitude above sea level, whereas the amount of Q-HRs in genome has a trend to decrease as the geographical latitude and altitude of permanent residence of human population increase (Ibraimov, 1993; Ibraimov, Mirrakhimov, 1985; Ibraimov et al., 1982a,b,c; 1982; 1986).

Let's conceive the utmost example. In a sense, living in the Far North or in the high mountains, sometimes, predispose to taking strong drinks just for having thermal comfort. However, given this, we assume that one and the same dose of alcohol taken by persons with different BHC may result in different consequences. So, in the individual with low BHC the alcoholic intoxication begins after he takes a relatively large amount of alcohol for one drink because of lower leveling of temperature in different parts of body that finally leads to stronger intoxication with a hang-over syndrome than with persons with normal or high

BHC. In other words, the lower BHC of an individual, the slower the intoxication begins. It is due to the longer time needed for heating the whole body that is necessary for having a sense of thermal comfort in the whole organism.

With drug addicts, i.e. the individuals with high BHC (because of high amount of Q-HRs in genome), drug addiction also appears due to the desire to have a sense of thermal comfort as soon as possible, but this time this “pleasure” is a result of a “drug overcooling” of the body that have subsequent emotional, or other experiences. We believe that the psycho-emotional effects of alcohol and drugs on the organism are attributed to the degree of violation of the temperature homeostasis, but they are manifested in quite opposite directions. In other words, while ethanol causes alcohol intoxication increasing the body heat (oxidation of 1 gram of ethanol produces 7 kilocalories), drugs, on the contrary, lower the temperature, thus causing the state of drug stupor. Nevertheless, the notorious susceptibility of the southerners to drugs, and northerners and highlanders to strong alcohol beverages may be explained, to a certain degree, by different Q-HRs content in their genome (Lubs et al., 1977; Ibraimov, Mirrakhimov, 1982a,b,c; Ibraimov et al., 1990, 1991, 1997), and accordingly relate it with the human BHC.

Let us now consider the situation with obesity. As we have shown, individuals with lesser amount of Q-HRs in genome are more prone to develop alimentary obesity (Ibraimov, 2016a). The results of numerous epidemiological researches, which were carried out in many countries and regions have unambiguously shown that females suffer from obesity two times more often than males. We assume that alimentary obesity is not the result of lack of inner discipline in taking meals or presence of hypothetical “gene of obesity”. In individuals with low BHC that is characteristic for the female organism in general, even if they take normal amount of food, fat will be stored in more amount than in persons with high BHC. It is easy to imagine that these individuals with a good heat isolating body, when they consume food rich in calories that is easily assimilated and who are under conditions of contemporary comfortable life possibly become more vulnerable to develop alimentary obesity (Ibraimov, 2016a).

It is known that with age the number of women in a population begins to prevail over men. Such a change in the sex ratio is usually explained by the fact that men are more subject to the effects of harmful factors (smoking, alcohol, etc.) or are more frequently engaged in professional activity with increased risk for life. Without calling in question the opinion of most people we suppose that a change in the sex ratio with age in favor of women is related to the amount of chromosomal Q-HRs in their genome. It is possible that a certain advantage of women is explained by their relative resistance, as compared with men, to cold and stress, hunger and even loss of blood because they have less heat conductivity body. In order to be convinced of the relative resistance of women to cold and stress, we shall give several known examples: a) pearl divers in Korea are exclusively women – “ama” (Folk, 1974); b) women succeed best in swimming across the cold water of La-Manche (Folk, 1974); c) during the period of the Leningrad blockade during the World War II about 80% of the women survived despite the fact that being in the rear they had a lesser access to food; d) at the reproductive age women, without detriment to their health lose every month from 120 to 300 ml of blood during menses and about 300-500 ml of blood even during normal labor. Men could hardly tolerate these losses of blood without detriment to their health. Thus, the known thesis that males seem to be less resistant to environment stress is also somewhat explained from our point of view.

We suppose that infants with a great amount of chromosomal Q-HRs in their genome are possibly subject more frequently to over cooling, catarrhal disease, etc. due to high heat conductivity of their body. Whereas, individuals with a low heat conductivity of body possibly have a certain advantage as concerns their survival in infantile age as compared with those who have a medium and especially great amount of chromosomal Q-HRs in genome. That is how we explain the “redundancy” of individuals with a lesser amount of chromosomal Q-HRs in genome in the population of elderly subjects (Ibraimov, Karagulova, 2006; Ibraimov et al., 2010a,b).

Indirectly other known medical-biological data justify our point of view: 1) the level of infant mortality and morbidity is higher with boys than girls. This trend especially expressed in high mountain climate conditions (Baker, 1978); 2) apparently, girls are better than boys “protected” from hunger and diseases, and the trend of curve of their physical growth breaks very rarely (Harrison et al., 1977); 3) mortality from infectious diseases in males is, on average, two times higher than in females; 4) in all ethnic groups males almost two times more often are sick with tuberculosis (TBS) than females. For example, in the USA the Afro-Americans are more susceptible to TBS that determines quick progress of the disease. Vice versa, in “whites” the TBS more often acquires chronic than acute form; 5) also it is known that among the animals only apes get our common cold and stand it very badly (Ibraimov, 2016c).

Aside from effects of hypothetical sex chromosome factors, the increased disease stress in males is poorly understood (Green, 1992; Synnes et al., 1994). The increased susceptibility of males to nutritional insult in early life, reported in both human and other animals (Katz, 1980; Smart, 1977, 1986; Lucas et al., 1990, 1998), is also generally assumed to be an unresolved biological issue (Lucas et al., 1998). Both morbidity and mortality are consistently reported to be higher in males than in females in early life, but no explanation for these findings has been offered. The latest attempt to explain this phenomenon belongs to Wells (2000), who argues that “the sex difference in early vulnerability can be attributed to the natural selection of optimal maternal strategies for maximizing lifetime reproductive success” and “that whatever improvements are made in medical care, any environmental stress will always affect males more severely than females in early life.” High morbidity and mortality in infants could be explained, in addition to the known to present day medicine reasons, by a simple physical consideration. As is known, in young children the surface/volume is very high, than that in adults. When one more physical factor, such as high BHC is added, then male neonates, which genome contains more Q-HRs than in girls, become very vulnerable to the factors violating the temperature homeostasis in their immature organism, particularly to common cold and its complications with all subsequent consequences.

VIII. CONCLUDING REMARKS

The role of the circulatory system (CS) has been discussed above in maintaining temperature homeostasis of endothermic organisms. However, the CS cannot influence directly the temperature inside the cells, as those are linked with the CS indirectly - through the extracellular space. Thus, the CS influence on inner cellular temperature homeostasis is limited and its effect, in general, comes to transferring surplus heat from the extracellular space. That is why it seems that the problem of maintaining the inner cellular temperature homeostasis is solved by cells themselves, and we call it the cell thermoregulation (Ibraimov, 2003; 2017). Apparently, the physiological organ-based thermoregulation functions relatively independently from CT as evolutionally new adaptive system (Ibraimov, Tabaldiev, 2007). From our point of view, CT can be the missing link, which should fill the “gap” between the thermoregulation systems, functioning at the molecular level (f.e. heat and cold shock proteins) and the whole organism. It is likely that we

faced with physiological problem which is a new and alien for classical courses of physiology.

Basal metabolic rate could be out of many factors known from physiology being capable of explaining the nature of wide human BHC variability in population. Indeed, as in the present case, its level turns less with human age. But it is known that basal metabolic rate, as a rule, is lower in women and southern latitude natives. Apart from that, basal metabolic rate is influenced by such factors as height, weight, body constitution, pulse rate and environmental temperature, which contradicts our data.

As of possible genetic factors the most appropriate is the amount of chromosomal Q-heterochromatin in human genome. Certainly, the thickness of peripheral layer of CC around cellular nucleus depends on total amount of chromosomal C-heterochromatin in the genome. But as we suppose, packaging density (compactization) of CC itself is basically connected with the amount of chromosomal Q-heterochromatin. The point is that human populations do not differ significantly in the quantity of C-heterochromatin in their genome (Ibraimov, Mirrakhimov, 1982d; Erdtmann, 1982). Wide quantitative variability at the level of populations is found only in the amount of Q-heterochromatin. To be exact, apparently, human BHC depends mainly on the amount of chromosomal Q-heterochromatin in his genome (Ibraimov et al., 2014). As the amount of chromosomal heterochromatin does not change in ontogenesis, it is possible that the level of BHC may be a constitutional character, the same as the color of skin, eye shape, body constitution, height and other innate physical human peculiarities.

And finally, do man need to know about the level of thermal conductivity of his body? People who are afraid of developing hypertension or diabetes regularly check their blood pressure or blood sugar levels. Perhaps those who fear obesity, alcoholism or drug addiction would not hurt to have an idea of the thermal conductivity of the body. This does not require a special device and can be checked by measuring the temperature of the palm and mouth at rest. If the temperature difference between the palm and the oral cavity does not exceed 1.0 °C, then such an individual has a highly heat conductive body. If this difference exceeds 2.1 °C then such an individual has a low-heat conductive body. In addition, in contrast to the measurement of blood pressure or blood sugar to determine the level of BHC need only once in life, because the amount of chromosomal Q-HRs in the human genome does not change during ontogenesis.

In conclusion, we would like to note that the main difficulty in elucidating the biological role of chromosomal HRs is the detection of their phenotypic manifestation. It is known, unlike structural genes, the DNA in the HRs does not encode known in the science of proteins and enzymes. From our point of view, human BHC level is a physiological phenotype of the amount of chromosomal HRs that are involved in maintaining temperature homeostasis in the body through cell thermoregulation.

IX. ACKNOWLEDGEMENTS

I apologize to those authors whose work is not cited or cited only through reviews. The reason for this is only the space limitations.

REFERENCES

- [1]. **Al-Nassar, K.E., Palmer, C.G., Connealy, P.M. & Pao-Lo Yu. (1981).** The genetic structure of the Kuwaiti population. II. The distribution of Q-band chromosomal heteromorphisms. *Hum Genet*, 57: 423-427.
- [2]. **Arrighi, F.E. & Hsu T.C. (1971).** Localization of heterochromatin in human chromosomes. *Cytogenetics*, 10: 81-86.
- [3]. **Acshoff, J. (1958).** Die extremitäten als effektoren der physikalischen temperaturregulation. *Wien Med Wochenschr*, 108: 404-409.
- [4]. **Baker, P.T. (1978).** The Biology of High Altitude Peoples. Cambridge University Press.
- [5]. **Bhasin, M.K. (2005).** Human population cytogenetics. A review. *Int J Hum Genet*, 5(2): 83-152.
- [6]. **Blatties, C.M. (1997).** Thermoregulation: Tenth International Symposium on the Pharmacology of Thermoregulation. *Ann NY Acad Sci USA*, March, Vol. 813(1): 1-863, Wiley.
- [7]. **Bostock, C.J. & Sumner, A.T. (1978).** The eukaryotic chromosomes. North-Holland Publ. Company. Amsterdam-New York-Oxford.
- [8]. **Buckton, K. E., O’Riordan, M. L., Jacobs, P. A., Robinson, J. A., Hill, R., & Evans, H. J. (1976).** C- and Q-band polymorphisms in the chromosomes of three human populations. *Ann Hum Genet*, 40, 90-112.
- [9]. **Caspersson, T., Zech, L. & Johansson C. (1970).** Differential binding of alkylating fluorochromes in human chromosomes. *Exp Cell Res*, 60: 315-319.
- [10]. **Comings, D.E. & Okada, T.A. (1970).** Association of chromatin fibers with the annuli of the nuclear membrane. *Exp Cell Res*, 62: 293-302.
- [11]. **Craig, J.M. (2004).** Heterochromatin – many flavours, common themes. *BioEssays*, 27: 17-28.
- [12]. **Chaturvedi, D., Vilhekar, K.Y., Chaturvedi, P. & Bharambe M.S. (2004).** *Indian Pediatrics*, 41(6), 600-603.
- [13]. **Ciminelli, B.M., Jodice, C., Scozzari, R., et al. (2000).** Latitude-correlated genetic polymorphisms: selection or gene flow? *Hum Biol*, 72: 557-571.
- [14]. **Decsey, K., Bellovits, O., & Bujdoso, G.M. (2006).** Human chromosomal polymorphism in a Hungarian sample. *Int J Hum Genet*, 6(3), 177-183.
- [15]. **Dimitri, P., Corradini N. & Rossi F. (2004)** The paradox of functional heterochromatin. *BioEssays*, 27, 29-41.
- [16]. **Elgin, S.C.R., & Grewal, S.I.S. (2003)** Heterochromatin: silence in golden. *Curr Biol*, 13(13), R895- R898.
- [17]. **Erdtmann, B. (1982).** Aspects of evaluation, significance, and evolution of human C-band heteromorphism. *Hum Genet*, 61: 281-294.
- [18]. **Folk, G.E. (1974).** Textbook of Environmental Physiology. Lea & Febiger. Philadelphia.
- [19]. **Green, M.S. (1992).** The male predominance in the incidence of infectious diseases in children: a postulated explanation for disparities in the literature. *Int J Epidemiol*, 21: 381-386.
- [20]. **Harrison, G.A., Weiner, J.S., Tanner, J.M. & Barnicot, N.A. (1977).** Human Biology. Oxford, Oxford University Press.
- [21]. **Ibraimov, A. I. (1993).** The origin of modern humans: a cytogenetic model. *Hum Evol*, 8(2): 81-91.
- [22]. **Ibraimov, A.I. (2003).** Condensed chromatin and cell thermoregulation. *Complexus*, 1: 164-170. doi:10.1159/000081065
- [23]. **Ibraimov, A.I. (2004).** The origin of condensed chromatin, cell thermoregulation and multicellularity. *Complexus*, 2: 23-34. doi:10.1159/000087851
- [24]. **Ibraimov A.I. 2010.** Chromosomal Q-heterochromatin regions in populations and human adaptation. In: MK Bhasin, C Susanne (Eds.): *Anthropology Today: Trends and Scope of Human Biology*. Delhi: Kamla- Raj Enterprises, pp. 225-250.
- [25]. **Ibraimov, A.I. (2015).** Heterochromatin: The visible with many invisible effects. *Global Journal of Medical Research (C)*, Volume 15, Issue 3, Version 1.0, pp. 7-32.
- [26]. **Ibraimov, A.I. (2016a).** Chromosomal Q-Heterochromatin Polymorphism in Patients with Alimentary Obesity. *Biol Med (Aligarh)*, 8: 275. doi:10.4172/0974-8369.1000275
- [27]. **Ibraimov, A.I. (2016b).** Chromosomal Q-heterochromatin Regions in Alcoholics and Drug Addicts. *Biol Med (Aligarh)*, 8:346. doi:10.4172/0974-8369.1000346
- [28]. **Ibraimov, A.I. (2016c).** Why only people and apes are ill with common cold? The possible role of chromosomal Q-heterochromatin. *J Mol Biol Res*, Vol. 6, No. 1, pp. 11-19. doi:10.5539/jmbr.v6n1p11
- [29]. **Ibraimov, A.I. (2017).** Cell Thermoregulation: Problems, Advances and Perspectives. *J Mol Biol. Res*, 7(1): 58-79. doi:10.5539/jmbr.v7n1p58
- [30]. **Ibraimov, A.I. & Karagulova G.O. (2006a).** Chromosomal Q-heterochromatin regions in individuals of various age groups. *Int J Hum Genet*, 6(3): 219-228.
- [31]. **Ibraimov, A.I. & Karagulova G.O. (2006b).** Chromosomal Q-heterochromatin variability in neonates deceased during first year of age. *Int J Hum Genet*, 6(4): 281-285.
- [32]. **Ibraimov, A.I., Mirrakhimov M.M., Nazarenko, S.A., Axenrod, E.I. & Akbanova, G.A. (1982).** Human chromosomal polymorphism. I. Chromosomal Q-polymorphism in Mongoloid populations of Central Asia. *Hum Genet*, 60: 1-7.
- [33]. **Ibraimov, A. I., & Mirrakhimov, M. M. (1982a).** Human chromosomal polymorphism. III. Chromosomal Q-polymorphism in Mongoloids of Northern Asia. *Hum Genet*, 62: 252-257.

- [34]. **Ibraimov, A.I., & Mirrakhimov, M. M. (1982b).** Human chromosomal polymorphism. IV. Q-polymorphism in Russians living in Kirghizia. *Hum Genet*, 62: 258-260.
- [35]. **Ibraimov, A.I., & Mirrakhimov, M.M. (1982c).** Human chromosomal polymorphism. V. Chromosomal Q-polymorphism in African populations. *Hum Genet*, 62: 261-265.
- [36]. **Ibraimov, A.I. & Mirrakhimov M.M. (1982d).** Human chromosomal polymorphism. II. Chromosomal C-polymorphism in Mongoloid populations of Central Asia. *Hum Genet*, 60: 8-9.
- [37]. **Ibraimov, A.I., & Mirrakhimov, M. M. (1985).** Q-band polymorphism in the autosomes and the Y chromosome in human populations. In: "Progress and Topics in Cytogenetics. The Y chromosome. Part A. Basic characteristics of Y chromosome". A. A. Sandberg (Ed). Alan R. Liss, Inc., New York, USA, pp. 213-287.
- [38]. **Ibraimov, A.I. & Tabaldiev, S.K. (2007).** Condensed chromatin, cell thermoregulation and human body heat conductivity. *J Hum Ecol*, 21(1): 1-22.
- [39]. **Ibraimov, A. I., Mirrakhimov, M. M., Axenrod, E. I. & Kurmanova G.U. (1986).** Human chromosomal polymorphism. IX. Further data on the possible selective value of chromosomal Q-heterochromatin material. *Hum Genet*, 73: 151-156.
- [40]. **Ibraimov, A.I., Kurmanova, G.U., Ginsburg, E.K., Aksenovich, T.I. & Axenrod E.I. (1990).** Chromosomal Q-heterochromatin regions in native highlanders of Pamir and Tien-Shan and in newcomers. *Cytobios*, 63: 71-82.
- [41]. **Ibraimov, A.I., Axenrod, E.I., Kurmanova, G.U. & Turapov, O.A. (1991).** Chromosomal Q-heterochromatin regions in the indigenous population of the Northern part of West Siberia and in new migrants. *Cytobios*, 67: 95-100.
- [42]. **Ibraimov, A.I., Karagulova, G.O., & Kim, E.Y. (1997).** Chromosomal Q-heterochromatin regions in indigenous populations of the Northern India. *Ind J Hum Genet*, 3: 77-81.
- [43]. **Ibraimov, A.I., A.K. Kazakova, I.K. Moldotashev, M.T. Sultanmuratov, & K.S. Abdyev (2010a).** Variability of Human Body Heat Conductivity in Population. I. Methodological and Theoretical Approaches. *J Hum Ecol*, 32(1): 1-22.
- [44]. **Ibraimov, A.I., A.K. Kazakova, I.K. Moldotashev, M.T. Sultanmuratov & K.S. Abdyev. (2010b).** Variability of Human Body Heat Conductivity in Population. II. Diseases of Civilization. *J Hum Ecol*, 32(2): 69-78.
- [45]. **Ibraimov, A.I., Akanov, A.A., Meymanaliev, T.S., Karakushukova, A.S., Kudrina N.O., Sharipov K.O., & Smailova R.D. (2013).** Chromosomal Q-heterochromatin polymorphisms in 3 ethnic groups (Kazakhs, Russians and Uygurs) of Kazakhstan. *Int J Genet*, 5(1), 121-124.
- [46]. **Ibraimov, A.I., Akanov, A.A., Meimanaliev, T.S., Sharipov, K.O., Smailova, R.D. & Dosymbekova, R. (2014).** Human Chromosomal Q-heterochromatin Polymorphism and Its Relation to Body Heat Conductivity. *Int J Genet*, 6(1), 142-148.
- [47]. **ISCN. (1978).** An international system for human cytogenetic nomenclature. Report of the standing committee on human cytogenetic nomenclature. *Cytogenet Cell Genet*, 21: 313(1)-404(92).
- [48]. **John B. (1988).** The biology of heterochromatin. In: "Heterochromatin: Molecular and Structural Aspects". Verma R.S. (Ed). Cambridge University Press, Cambridge, New York, New Rochelle, Melburn, Sydney. pp.1-147.
- [49]. **Kalz, L., Kalz-Fuller, B., Hedge, S., & Schwanitz, G. (2005).** Polymorphism of Q-band heterochromatin; qualitative and quantitative analyses of features in 3 ethnic groups (Europeans, Indians, and Turks). *Int J Hum Genet*, 5(2), 153-163.
- [50]. **Katz, H.B. (1980).** The influence of under nutrition on learning performance in rodents. *Nutr Abstr Rev.*, 50: 767-783.
- [51]. **Lampert, F. (1971).** Attachment of human chromatin fibers to the nuclear membrane as seen by electron microscopy. *Humangenetik*, 1971; 13: 285-295.
- [52]. **Lubs, H. A., Patil, S. R., Kimberling, W. J., Brown, J., Hecht, F., Gerald, P., & Summitt, R. L. (1977).** Racial differences in the frequency of Q- and C-chromosomal heteromorphism. *Nature*, 268, 631-632.
- [53]. **Lucas, A., Morley, R., Cole, T.J., Gore, S.M., Lucas, P.J.et al. (1990).** Early diet in preterm babies and developmental status at 18 month. *Lancet*, 335: 1477-1481.
- [54]. **Lucas, A., Morley, R. & Cole, T.J. (1998).** Randomised trial of early diet in preterm babies and later intelligence quotient. *BMJ*, 317: 1481-1487.
- [55]. **Mathew, L. (1992).** Severe Cold at High Altitude and Methods of Prevention of its III Effects. In: SK. Machanda, W. Selvamurthy, V. Mohan Kumar (eds): *Advances in Physiological Sciences*. New Dehli, Macmillan India LTD, pp. 338-345.
- [56]. **Mayr, E. (1970).** Populations, Species, and Evolution. The Belknap Press of Harvard University Press, Cambridge, Massachusetts.
- [57]. **Paris Conference, (1971), and Supplement (1975).** Standardization in human cytogenetics. *Birth Defects: Original Article Series*, XI, 1-84. The National Foundation, New York.
- [58]. **Pearson, P. L. (1973).** The uniqueness of the human karyotype. In T. Caspersson, & L. Zech (Eds.), *Chromosome identification techniques and application in biology and medicine* (p. 145). New York, London. Academic Press.
- [59]. **Pearson, P. L. (1977).** Pattern of bands, polymorphism and evolution of primates. In J. J. Yunis (Ed.), *Molecular structure of human chromosomes*. Acad. Press.
- [60]. **Prokofyeva-Belgovskaya, A. A. (1986).** Heterochromatic Regions of Chromosomes (in Russian). Moscow, Nauka.
- [61]. **Quintana, E.C. (2004).** How reliable is axillary measurement? *Emergency Medicine*, 43(6): 797-798.

- [62]. **Smart, J.L. (1977).** Early life malnutrition and later learning ability: a critical analysis. In A Oliviero (Ed): Genetics, Environment and Intelligence. Amsterdam, Elsevier/Noth-Holland, pp. 215-235.
- [63]. **Stanyon, R., Studer, M., Dragone, A., De Benedictis, G., & Brancati, C. (1988).** Population cytogenetics of Albanians in Cosenza (Italy): frequency of Q- and C-band variants. *Int J Anthropol*, 3, 19-29.
- [64]. **Stringer, C.B. (1996).** Evolution of early humans. In: The Cambridge Encyclopedia of Human evolution. S. Jones, R. Martin and D. Pilbeam (eds.). Cambridge University Press, Cambridge, pp. 241-251.
- [65]. **Synnes, A.R., Ling E.W., Whitefield M.F., Mackinnon M., Lopes L., Wong G. & Eiffer S.B. (1994).** Perinatal outcomes of a large cohort of extremely low gestational age infants (twenty-three to twenty-eight weeks of gestation). *J Pediatr*, 125: 952-960.
- [66]. **Van Someren, E.J.W., Raymann, R.J.E.M., Scherder, E.J.A., Daanen, H.A.M, & Swaab, D.F. (2002).** Circadian and age-related modulation of thermoreception and temperature regulation: Mechanisms and functional implications. *Aging Res Rev*, 1: 721-778
- [67]. **Verma, R.S. (Ed). (1988).** Heterochromatin. Molecular and Structural Aspects. Cambridge Univ. Press. Cambridge, New York, Sydney.
