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Why the Arctic Indigenous people are resistant to cardio-vascular diseases? A view on the Euro-Asian North

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ABSTRACT

Although an increasing prevalence of coronary heart disease and arterial hypertension in the indigenous northern peoples currently takes place, it was low a few decades ago. The topic of this review is a resistance of the Northern native populations to cardiovascular diseases, that is known for a long time but still remains scientifically unexplained. These multi-factor diseases of complex etiology have a genetic component that forms a predisposition to these diseases. The lack of scientific knowledge about the mechanisms of the previous resistance to the development of atherogenic pathology and the causes of its current progression hinders prevention, effective therapy and clarification of genetic and physiological prerequisites of individual and ethnic vulnerability to this pathology not only among the First Nations people of the North, but also in their historical ancestors – inhabitants of the middle and low latitudes.

Keywords: Arctic; North; Indigenous People; Arterial Hypertension; Arterial Stiffness

Introduction

This review addresses the increasing incidence of coronary heart disease (CHD) and arterial hypertension (AH) among recent northern populations. These diseases have historically been absent from the indigenous inhabitants of Arctic regions. Gaining scientific knowledge about the causes of the arctic ethnic groups' previous resistance to the development of atherogenic pathology and the factors underlying their current progression would help elucidate the genetic and physiological prerequisites for individual and ethnic tolerance to this pathology. The relevance of the above problem, or the need for its resolving at the present time, is due to the gradual but steady loss of phenotypic features of the cardiovascular system in the form of tolerance to the development of hypertension in Northern nationalities. This loss can occur for at least two causes. First, the genetic originality of Northern Minority populations is gradually being lost, "blurred" because of mixed marriages and preference of grooms of Caucasian race by aboriginal women, which originality seems to have been responsible earlier for the resistance described here. The second reason is environmental "pressure" in a broad sense, which manifests itself in changes in lifestyle and type of food, physical activity, anthropogenic transformation and technogenic pollution. Thus, science will soon lose genetically peculiar and phenotypically unique object – people historically adapted to the Arctic conditions. These people are carriers of properties that are important for getting knowledge on the resistance to AH that is one of the main causes of death in the developed countries.

One more aspect of the importance of genetic and physiological studies of the Northern Ethnic peoples is their still quite numerous populations, geographical isolation, compactness of residence in the limited areas, that facilitates expeditions and obtaining representative data. A relevant reason for elucidating ecological dependence of this phenomenon is the climatic conditions in the Asian Arctic, which are quite extreme and differ from those in Scandinavia, Greenland and the European North of Russia, experiencing the softening influence of the warm Atlantic Ocean. In addition to the above-mentioned resistance to diseases, a "northern" man likely also represents the only model of cold resistance, which model will serve as a source of genetic material for the restoration of the species in the event of a possible ex-

treme cold snap on Earth, for example, as a result of weakening solar activity, a "nuclear winter", atmospheric dustiness due to a collision of the planet with a celestial body, or a super volcanic eruption - events, some of which have already occurred in the history of the Earth. The resistance of interest has become known to readers long ago, at least in 1863, when a 484-page book has been published by German scientist Georg Hartwig in Russian language, in which he described his observation about the absence of CVD in the indigenous residents of the European North of Russia. Later, this observation was repeatedly confirmed (Brown, et.al [1-3]). A lower prevalence of CHD and hypertension was described in the Eskimos of Greenland (Bjerregaard, et al. [4]), North American Indians (Jolly et al. [5,6]), Northern Finland inhabitants (Luoma, et al. [7]) compared with the non-indigenous population. However, the frequency of vascular disorders and mortality from them increases after the 1980s (Ivanov [8]), that is especially noticeable in comparison with the decreasing mortality from these causes due to the medicine progress in mid-latitude populations.

In a population study, the Swedish Sami were found to have lower blood pressure, triglyceride and cholesterol levels than non-Sami (Edin Liljergen, et al. [9]). In the residents of the North Siberian Yamal-Nenets District, the excess of systolic blood pressure in Russian people compared to their peers of the Khanty and Mansi nationalities is manifested already in childhood (Anchugin, et al. [10]). A lower prevalence of hypertension was found in Siberian Shors (Barbarash, et al. [11]), Eskimos of Greenland (Bjerregaard, et al. [4]), Asian Arctic minorities (Burtseva, et al. [12]), North American Indians (Foulds, et al. [6]) compared with the general population, but the frequency increased after the 1980s. Later, the latter authors, measuring the pulse wave velocity in the aorta and the intima-media thickness of the brachiocephalic arteries, showed high vascular elasticity in Northern Canadian Indians (Foulds, et al. [13]). (Alekseev, [14]), observing postmortal material in doctor's dissertation, discovered more pronounced morphological signs of atherogenic lesions of the aorta and brachiocephalic arteries in immigrants to Yakutsk compared to native Yakuts.

It's worth noting here that arterial hypotension is a characteristic not only of the indigenous peoples of the Arctic Circle. It also occurs briefly, initially, in people of other nationalities who arrive there (Kandror, [3])—winterers at northern and Antarctic polar stations and seasonal shift workers (Shepovalnikov, et al. [15]). The previous resistance of adult northerners to hypertension may be explained by high pressure of natural selection, which manifests itself in the ontogenetically early elimination of individuals with weak constitutions as a result of infectious diseases (Tikhonov, [16]). Such elimination enriches the population with individuals who are resistant to CVD in adulthood. The increasing frequency of cardio-vascular diseases among Arctic peoples over the past 60 years is explained (Hasnulin, et al [17]) by climatic and geographical stress (2016), while the Danish authors of the review attribute it to the westernization of lifestyle, a decrease in child mortality, an increase in actual life expectancy and aging of populations, increasing anthropogenic pollution of the environment and the increasingly widespread risk factors for this pathology (Tvermosegaard, et al. [18]).

In the context of the problem under discussion, it is necessary to note the obvious association between hemodynamics and the cold factor. It has been found that both general prolonged (10°C, 2 hours) and local acute (0°C, 11 min, upper or lower extremities) cooling increases SBP, DBP and HR (Korhonen, [19]). As early as the 1960s, many authors studying thermoregulation in the aborigines of the North established a higher temperature of the extremities during their cooling in Eskimos and Alaskan Indians than in African Americans and white Americans (Mechan, [20]), in Lapps than in Caucasian residents of Norway (Andersen, et al. [21,22]), in Athapaskan Indians in comparison with neighboring individuals of the Caucasian race (Elsner, et al. [23]).

G. Brown found greater tissue engorgement in Eskimos from Southampton Island compared to a control group of American students (Brown, [1]). Higher blood flow in the hands of Lapps during hand cooling was described as higher than in Norwegian fishermen living at latitude 69°C, and significantly higher than in recently arrived residents (Krog, et al. [21]). The latter authors explain their finding of high blood circulation during cooling by a long-term adaptation to this factor, based on a decrease in the ability of blood vessels to constrict. Thus, the results of the planned study will advance the understanding of the paradox: the effect of cold, which is associated with constriction of peripheral blood vessels, should lead to hypertension, but is nevertheless combined with a reverse hypotonic reaction of the systemic circulation.

To explain the resistance to AH of tundra and coastal inhabitants of the North, one can also propose a "salt" hypothesis about the consumption of low-mineralized water from surface river sources, which are excessively filled with melted snow and ice and are poorly enriched with minerals due to biogenic weathering and vertical transport of minerals from permafrost soils and the lithosphere. The pathogenic role of table salt in the development of AH is now well-known. It is appropriate to note here the poor study of the system of regulation of water-salt homeostasis in Arctic mammals, indigenous people of the North, and the non-indigenous population. Such work can serve as a source of a new direction in Arctic physiology with potentially interesting practical and basic findings. In northern habitats, free access to sodium chloride has historically been quite rare, which may explain the adaptive taste aversion to table salt described by ethnographers who observed the life of the northern indigenous people in natural environment (Bogoraz, [24]).

As another explanation for the Arctic immanent hypotension of the systemic circulation, one can suggest a mechanism related to the so-called "circumpolar hypoxic syndrome" (Avtsyn, et al. [25,26]) or

"northern tissue hypoxia," which is summarized in the monograph by (LB Kim [27]) and manifests itself in "polar dyspnea," a forced shallow breathing pattern that protects the respiratory tract from hypothermia. On the other hand, hypoxia indirectly causes vasodilation of muscle arteries through endothelial nitric oxide, which has been proven for both experimental (Vedam, et al. [28,29]) and high-altitude (Melnikov, et al. [30]) hypoxia. This interpretation is supported by the fact that hypotension is more pronounced in northern regions during the cold season, when freezing winds exacerbate difficulties with external respiration (Kandror, [3]).

To explain mechanisms of the phenomenon under review, an additional original hypothesis could be proposed that the resistance of concern is due to increased arterial elasticity and reduced responsiveness to vasoconstrictors and is associated with certain allelic variants of polymorphic genes involved in the regulation of arterial tone and blood pressure. Neither arterial elasticity nor its genetic bases have been studied in Arctic ethnic groups. This hypothesis could be resolved by measuring parameters of central hemodynamics and arterial elasticity with using applanation tonometry and analysis of pulse wave profile in expeditions to territories resided by native populations differing in time of colonization of the Arctic, geography of origin, lifestyle, type of food, and environmental conditions. To test the hypothesis, at least five polymorphic genes that are known to be associated with some pathological cardiovascular phenotypes, could be selected for exploration. Among them are those related to AH or structural disorders of connective tissue, that occur in the form of aneurysms and bundles of artery walls and prolapse of heart valves. Two belong to the renin-angiotensin system: AGT (gene that regulates the synthesis of angiotensinogen, rs699) and ACE (gene that controls the production of angiotensin converting enzyme ACE). The other two are genes encoding the synthesis of α2β-adrenergic receptor mediating vasoconstrictor action of catecholamines (ADRA2B, rs28365031), and NOS3 gene (e.g. VNTR 4a/4b) encoding the NO-synthase catalyzing the production of endothelial vasodilatator nitrogen monoxide. The fifth could be, for example, the gene COL3A1 (rs1800255), the expression of which provides the synthesis of type III collagen, that is a filament of the fibrous tunic of the arterial wall. The more effective approach could certainly be the genome-wide analysis and the subsequent comparison of the results with those obtained in a nearby nonaboriginal populations.

Modern genetic studies of northern and Siberian ethnic groups mainly focus on their origin (Huyqhe, et al. [31,32]), degree of kinship and migration routes (Lazaridis, et al. [33-36]), metabolic diseases (Zhou, et al. [37,38]), cold resistance (Hancock, et al. [39,40]). The article by authors from the University of Nebraska and the Institute of Cytology and Genetics of the Siberian Branch of the Russian Academy of Sciences is devoted to the role of natural selection in the genetic fixation of specific metabolism during the adaptation of Arctic and Siberian peoples to a protein-and-fat-based diet (Hsieh, et al. [41]. Arbour et al. [42]) review circumpolar populations, noting their previous resistance to atherogenic pathology, and analyze the genetic basis of CVD with a focus on hereditary cardiac malformations. Although only a few studies have been conducted on the prevalence and treatment of hypertension and the involvement of certain genes in the determination of this disease among Siberian and northern ethnic groups (Barbarash, et al. [11,43]), the elastic properties of arteries and their genetic markers have not been studied among the Arctic indigenous peoples. Genetic studies of arterial elasticity and central hemodynamics, frequency or association analysis, have not been conducted in the world's northern populations, and, as far as the author aware, are not currently being conducted. In a recently published pilot study, Zhou, et al. [36] evaluated the genetic basis of susceptibility to cranial artery aneurysms in the Inuit population of Canada.

The theoretical significance of studies performed for solving the discussed problem is that the elastic properties of arteries in aboriginal northerners are currently unknown. It is important to answer the question whether the historically low prevalence of CHD and AH in the Arctic inhabitants (Danishevsky, [2,4,38]) is a consequence of the high resistance of the arteries to age-related remodeling and thickening. The Northerners in this context are considered as model objects – the result of the evolutionary transformation of the genome and physiology of ethnic groups in the specific conditions of the Arctic – to learn the mechanisms of the development of AH.

To summarize, this review describes and tries to explain a paradox. Living in cold conditions, to which individual physiological adaptation is accompanied by limited superficial blood flow and permanent spasm of skin vessels, should lead to increased blood pressure (Brook, [44]). However, at the population level, the opposite pattern is observed in indigenous people as a result of historical adaptation. This fact is especially important given that hypertension in the North is more common and more severe among coming non-native populations than in mid-latitudes, which is explained by the pronounced heliogeophysical stress in the Earth's auroral zone (Hasnulin, et al. [17]).

The data presented in this review indicate that the discussed problem needs further investigations. The lack of scientific knowledge about the mechanisms of maintaining the previous resistance of northern aboriginals to atherogenic pathology and the causes of its current progression prevents development and application of effective therapeutic methods, clarification of the genetic and individual physiological prerequisites and ethnic vulnerability to this pathology not only in the inhabitants of the North, but also in their historical ancestors – residents of middle and low latitudes (Anuchin, et al. [45-50]).

The potential results of studies under discussion will expand the scientific understanding of genetic and physiological causes and arterio structural bases of predisposition to hypertension. Additionally, the discovering of genetic bases of the AH-resistant phenotype will

give physicians practically significant markers for the prognosis and selection of tactics for AH treatment, especially its pharmacoresistant forms. Scientists will get the direction for possible genome editing and genetic engineering studies aimed at obtaining AH-resistant organisms.

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