British Journal of Medicine & Medical Research
2(4): 647-661, 2012

SCIENCEDOMAIN *international*
www.sciencedomain.org

Where and When Human Viral Epidemics First Emerged

Sergey N. Rumyantsev[1*]

[1]*Department of Evolutionary Immunology, Andent, Inc, Jersey City New Jersey, 07302 USA.*

Research Article

Received 26th June 2012
Accepted 3rd November 2012
Published 10th November 2012

ABSTRACT

Aims: This article attempts to date the conditions, terms and places of the first emergence of the main human viral epidemics (HIV, influenza, measles, smallpox).
Place and Duration of Study: Department of Evolutionary Immunology Andent, Inc., between May 2002 and July 2012.
Methodology: The investigation was based on the integration and consequent sensing of relevant recent achievements of evolutionary branches of immunology, epidemiology and anthropology. The main focus was on the integration of up-to-date achievements of both evolutionary and historic anthropology with the data regarding inter-ethnic differences in hereditary immunity to relevant infections and the traces of the aforementioned epidemics.
Results: In contrast to rabies infection the considered epidemics emerged non-simultaneously between 14,000 and 10,000 years ago on the Eurasian territories. They were introduced into America and Australia as well as Sub-Saharan Africa during the Great Geographical Discoveries and consequent Colonization.
Conclusion: After their origin, both the epidemics and the traits of hereditary immunity against them continued to exist among humankind to the present, supported by genetically mitigated heterozygous forms of infections.

Keywords: Anthropogenesis; hereditary immunity; human evolution; HIV; influenza; measles; rabies; smallpox.

1. INTRODUCTION

Integrated results of both current and retrospective observations on epidemic processes indicate that the threat of infectious diseases has begun to diminish toward the end of the second millennium. This resulted from the combined epidemiological, hygienic,

**Corresponding author: Email: rumyan1@yahoo.com;*

immunological, and pharmacological efforts made during the 20th century as well as from the natural evolution of epidemic processes (Rumyantsev, 2008, 1997). Nevertheless, complete eradication of epidemics continues to be elusive. What is more, there has been considerable thought that we may be losing this 'war' (Abdool Karim, 2012). Many questions about the nature of infectious diseases and their varied pandemic spread have not received the answers they deserve. One of the principal uncertainties surrounding human epidemics is related to the initial source of their origin: how, where and when each of a number of infectious diseases emerged for the first time and began its epidemic spread. These questions are of great theoretical and practical interest to both scientists and non-scientists.

Although humankind's confrontation with the existing set of life-threatening microbes has a long history (Khor and Hibberd, 2011), the dating of ancient epidemics can currently make reference only to the recent past. The attempts to obtain appropriate answers have been made by various researchers. According to the first hypothesis to be proposed, infectious diseases first occurred in the relatively recent history of mankind, i.e., over the last 5,000 years (Haldane, 1949). This hypothesis was based on epidemiological extrapolations and introduced the first discoveries on the matter.

Paleo-epidemiological extrapolation studies have attempted to reconstruct the possible patterns of initial affliction of ancient humans with various infectious diseases. Such reconstructions have been undertaken through ethnic analogy – that is, by examination of disease pattern in contemporary hunter-gatherer populations followed by extrapolation of the findings into the ancient ones (Black, 1975; Cockburn, 1971; Polunin, 1953). The investigations included serological testing of currently isolated populations for relevant antimicrobial immunoglobulines as the evidential traces of foregoing infectious diseases.

This approach confirmed the initial hypothesis (Haldane, 1949) that epidemics of transmissible infections could not exist among ancient Neolithic hunter-gatherers groups whose smallness, high mobility, and isolation did not favor the transmission of infectious agents between them. Viral diseases that are infectious only in the acute phase die out quickly after introduction in a population (Black, 1975) and thus require a larger population for their maintenance than existed in any coherent group in Neolithic times. It has been suggested that the latter diseases could not perpetuate themselves before the advent of advanced cultures and did not affect humans until relatively recently (Black, 1966), i.e. not earlier than 10,000 years ago {ya) in South West Asia, between 8,000 and 4,000 ya in Europe, Egypt, India, China, but by 3,500 ya in Mexico and South America.

The data of more recent discoveries is scarce but controversial. For instance, the existence of HIV infection and relevant epidemics was first recognized by the Centers for Disease Control and Prevention (CDC) in 1981 (Gallo, 2006, 1246/id). The first date of the emergence of HIV epidemics was identified as 12,000 ya in EurAsia (Rumyantsev, 1992). In contrast, more advanced discoveries have indicated that HIV originated in west-central Africa during the late nineteenth or early twentieth century (Gao et al., 1999; Worobey et al., 2008), i.e. near one hundred years ago. Based on the supposition that the Vikings had genetic immunity against the infection, the emergence of HIV as a human pathogen was identified as being between 700 and 2900 ya (Gaggiotti, 2006; Lucotte and Dieterlen, 2003).

The current publication presents results achieved on the basis of a new approach: thesearch for, integration, analysis and comprehension of appropriate evolutionary, epidemiological, immunological, anthropological and paleo-genetic data. Primary attention has been focused on identifying among current humans the traces of ancient epidemics formed by natural

selection for hereditary immunity to relevant infections. This kind of evidence has not been adequately taken into account in previous studies.

The objective of this paper is not to present final results but to outline the rationale of the study as well as to describe the methods used and to report baseline data. The paper presents the initial results of an attempt to use an integrative (bio-ecological, epidemiological, genetic, immunological and anthropological and evolutionary) approach to estimate the dates and places of first emergence of four chosen viral infectious diseases (HIV, influenza, measles, smallpox) among humans.

2. MATERIALS AND METHODS

The presented integrative discovery has been based first on the results of previous long-term investigations performed by the author together with his team, as well as on a selection of various published data, derived mainly from observations not related to the matter and goals of our current search. The main emphasis was on the search for traces of ancient selection for hereditary immunity against HIV, influenza, measles and smallpox as well as on the determination of their origin in association with the ecology of *Homo sapiens* on key stages of its descent and further development. This set of four infections has been chosen because universality of their epidemiological traits and the availability of appropriate historical and anthropological data about their epidemics. Rabies infection, tick-born encephalitis and malaria have been excluded from present study because their substantially different epidemiological traits that were analyzed in our previous publications (Rumyantsev, 2012, 2008). In the present discovery, the integration of both epidemiological and evolutionary anthropological data and their integrative analysis has been taken into account for the first time in relation to consecutive periods of human evolution, from its beginnings to the modern age. The search for the traces of ancient infectious selections as well as their data gathering were performed according to (Rumyantsev, 2008). The revealed set of appropriate data of relevant observations, experiments and cumulative sensing of their hookup has been united using the approach of maximal bio-ecological, epidemiological and evolutionary integration.

3. RESULTS AND DISCUSSION

3.1 Searches for the Traces of Foregoing Epidemics

Any infectious disease arises and exists as a result of natural ecological relations between two species, in which the microbial one (the consumer) obtains the energy for its life at the expense of substances composed of the consumed organism (the victim). The action of parasitic microbes restricts the vitality of the victim, thus provoking a loss of its viability. Once filled with infectious agents, the body of the affected victim serves as a source of microbial invasion into new victims.

The intrusion of infectious agents inside the victim's body is mainly carried out by means of the victim's ecological communications, through which the regular physiological functions are provided; for example, through feeding (as an alimentary intrusion), breathing (respiratory intrusion) and self-reproduction. Of the three, the alimentary transfer of infectious agents functions most widely and effectively (Burgasov and Rumyantsev, 1974). All steps of microbial consumption are performed by means of specific microbial molecular ecological agents (either by adhesins, toxins, enzymes, cytolysins, or polynucleotide, etc.),

each of which is peculiar for separate microbial species. Each of the agents acts on its relevant molecular targets in the victim's body.

The success of microbial aggression depends strongly on the mutual chemical complementarity between relevant molecular structures of both the parasite and involved victim. In a case of intermolecular incongruence, the attacked organism appears to be constitutionally unavailable (unsusceptible) and thus cannot be affected (Rumyantsev, 1977; Rumyantsev, 1997). The incongruence created by mutant modification of target molecules makes mutant individuals constitutionally immune to the disease. They give rise to immune progeny, while susceptible homozygous individuals of the same species become affected and die without reproducing (Haldane, 1949). On repeated exposure of many generations to a given pathogen, the progeny of immune variants eventually predominate in a population (Fig. 1) and, finally, among the majority of the species. The state of immune homozygosis launches full protection, whereas heterozygosis abridges progress to full-blown disease by means of the 'wall off' of infectious agents inside restricted patches of affected tissue (O'Brien and Dean, 1997; Paxton, et al., 1996; Rumyantsev, 2002). The genetically mitigated heterozygous forms of infections are able to restrict the possibility of self-annihilation of these forms of symbiosis as well as of artificial eradication of relevant epidemics {Rumyantsev, 2008 1044 /id}.

Thus every epidemic process consists of two intrinsic accessaries mutually evolved over their shared antagonistic co-existence. Every infectious epidemic leaves its specific hereditary immunological marks on the structure of both genotype and phenotype of individuals, populations and species involved in the co-existence with the relevant infectious agent. Examination of these remains allows researchers to describe the disease patterns of ancient populations and establish their chronologies. The relicts of ancient infectious epidemics have survived from earlier periods and can now be detected in various levels of the human bodily architecture (Table 1).

The extensive presence of relevant traits of hereditary immunity among contemporary human populations is now evidenced by many approaches, including epidemiological and clinical observations, controlled experimental infections, cytological investigations, molecular biological investigations and by discovery of genome make-up (Rumyantsev, 2010a; Rumyantsev, 2008). All the revealed traits can be considered in retrospect to be evidence or traces of previous infectious selection performed by ancient epidemics. Currently, the methods of evolutionary ecological analysis allow us to estimate how and when the traces appeared over well-estimated stages of human evolution.

3.2 Emergence of Epidemics over the Descent of Humankind

Early *Homo sapiens*, anatomically, physiologically and immunologically analogous to modern humans, emerged on the African savannah (Novembre and Stephens, 2008) nearly 200,000 ya (White et al., 2003). In addition to the bipedality inherited from *Ardipithecus* through *Australopithecus*, early *Homo sapiens* differed from their ape ancestors in that they had a far bigger brain, naked body, the ability to run, could use primitive tools and elaborate on them, and also had a crude ability for conscious thought and speech. They were typical Neolithic hunter-gatherers.

The early sapient humans could inherit from their nearest ancestors the traits of hereditary immunity against rabies, tick-borne encephalitis, malaria as well as against alimentary infections associated with omnivory but especially with meat food (Rumyantsev, 2012,

2010b, 2008). On the other hand, mutual evolution of these antagonistic species inevitably led to the emergence of microbial subspecies, among which the *Clostridium botulinum* types A, B, C, D, E, F, G as well as innumerable variants of other alimentary clostridia (Burgasov and Rumyantsev, 1974) and *Salmonella* genus (Rumyantsev, 2004a) can be named, for instance. The traits for inherent protection against enumerated alimentary infections today belong to the majority of the current members of *Homo sapiens* species, too (Rumyantsev, 2004a). The features are now traced up to the levels of cellular and molecular architecture, including within the genome (Rumyantsev, 2008).

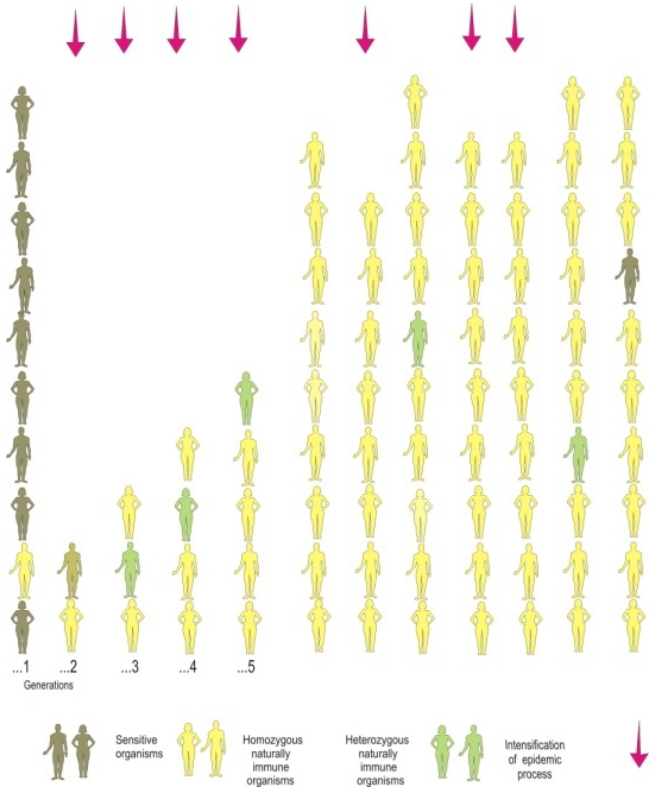

Fig. 1. Diagrammatic representation of hereditary immune state of a human population transformed over the emergence (1) and further evolution (2, 3 , 4 …) of epidemic processes. The prevalence of hereditary immune organisms beside with the minority of susceptible ones is shown (Rumyantsev 2002, updated).

Table 1. Principles, methods and means for diagnosis of hereditary immunity (Rumyantsev, 2008)

Principles	Methods	Means		Results
		Agents	Objects	
Integral	Epidemiological observations	Natural microbe population	Species, populations and persons	Integral (hereditary and reactive) immunity of species, populations and persons.
	Clinical	Natural microbe population		Integral (hereditary and reactive)immunity of persons, organs and tissues
	Experimental	Cultivated microbes or their molecules	Persons, organs, tissues	Integral (hereditary and reactive) immunity of persons, organs or tissues
Analytical	Histological	Cultivated microbes or their molecules	Cultures of organs or tissues	Hereditary immunity of organs or tissues
	Cytological	Cultivated microbes or their molecules	Cultures of cells; organoids	Hereditary immunity of cells or organoids
	Cytological		Cells of potential victim	Hereditary immunity of cells
	Cytological Molecular ecological	Microbial molecules	Molecules of the organism tested	Hereditary immunity of molecules
	Molecular anatomical		Molecules of the organism tested	Hereditary immunity of molecules
	Molecular genetic		The genome of the organism tested	Genes of hereditary immunity

In contrast, according to (Haldane, 1949) and other paleo-epidemiologists (Black, 1975, Cockburn, 1971), the epidemics of transmissible infections could not exist among ancient Neolithic hunter-gatherers groups whose smallness, high mobility, and isolation did not favor the transmission of infectious agents between them. Viral epidemics feed this condition first of all. The environments of the African savannah did not allow earliest humans catch HIV, influenza, measles and smallpox and elaborate hereditary immunity against these infections. Detailed differences in the presence/absence of genetic immunity to these diseases among different descendant subpopulations of early humans are analyzed below (see section 4).

3.3 Following the Wanderings of Ancient Humans around the World

Nearly 75,000–62,000 ya, some small (20-60 persons) groups of early *Homo sapiens* began to sweep out of the African Savannah territory where their descent and initial establishment had been accomplished. This initiated the dispersion of humankind around the world (Fig. 2).

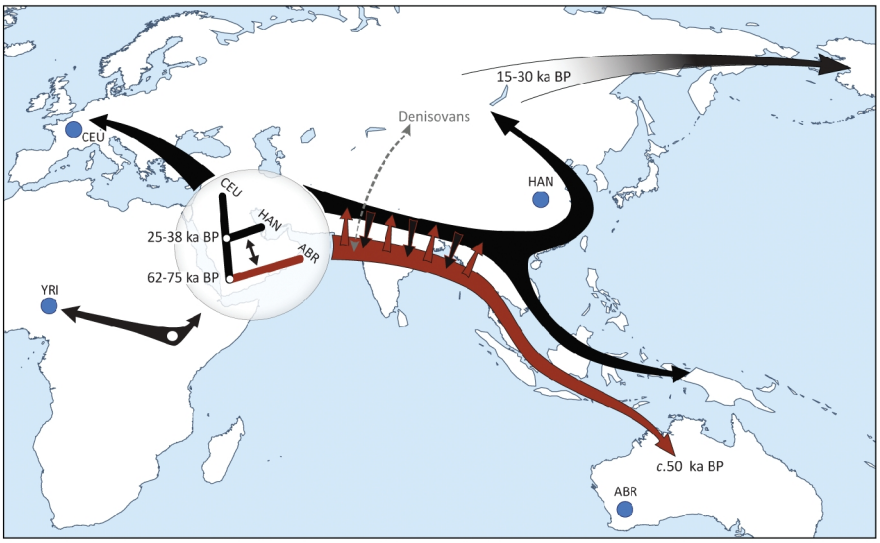

Fig. 2. Reconstruction of early spread of modern humans outside Africa (Rasmussen et al., 2012)

Some groups of migrants moved out of the former savannah's "Eden" back into the remnant of tropical forest that was the homeland of their faraway ape predecessors (Rumyantsev, 2008, 2010b). For over 60 millennia, this branch of *Homo sapiens* has been almost totally isolated from other subpopulations of humankind. Other groups moved east out of Africa along either South Asian or North Eurasian directions. All non-African populations currently living in the world probably derived from a single dispersal of early humans out of Africa (Rasmussen et al, 2012). The South Asian migration continued toward Australia and eventually reached this continent ~50,000 ya. Since then, the population lost any interrelations with other parts of humankind. For 50 millennia this very ancient branch of *Homo sapiens* has been in almost total isolation from other subpopulations of the species (Rasmussen et al, 2012). The North Eurasian dispersal divided (38,000–25,000 ya) into European and Asian directions. The last one continued (30,000 – 15,000 ya) its way toward the American continent (30,000 - 15.000 ya) and reached it nearly 14,000 ya. Since then,

the native subpopulation of the American continent has been in almost total isolation from other branches of humankind and from epidemic processes. As a result of the dispersion around the world, at least four subpopulations of early humans – Sub-Saharan, Australian, American and Eurasian - have separated from the initial population of early humans. Moreover, each of them appeared in ecologically different parts of Earth and in strong geographical isolation. Where and when could the subpopulations of early humankind meet and adopt the considered viral infections (HIV, influenza, measles, smallpox) and thus initiate appropriate epidemic processes?

3.4 Emergence of Human Viral Epidemics

The great geographical discoveries and consequent intensive colonization of the American and Australian continents disrupted isolation. Since the beginning of the 16th century, the new settlers of opened territories brought to indigenous peoples many infections they had not encountered before. Many historians argue these invasions did more to decimate isolated native populations than did warfare or enslavement, especially through epidemics of new diseases such as smallpox, influenza, and measles (Balter, 2011; Rumyantsev, 2004b; Stearn and Stearn, 1945; Thornton, 1987). The diseases had a devastating impact on many aboriginal populations that appeared to be originally highly susceptible. They did not have the traits of foregoing selection for hereditary immunity against these infections in their remote past. Nevertheless, the aftereffects of foregoing history are seen in today's events.

3.4.1 Evidence of emergence of HIV infection

During 2005, 3.1 million people (0,048% of the world population) died of HIV-related illnesses (UNAIDS, 2006). The epidemic of HIV infection remains present everywhere in the world, but varies in levels of intensity (Fig. 3).

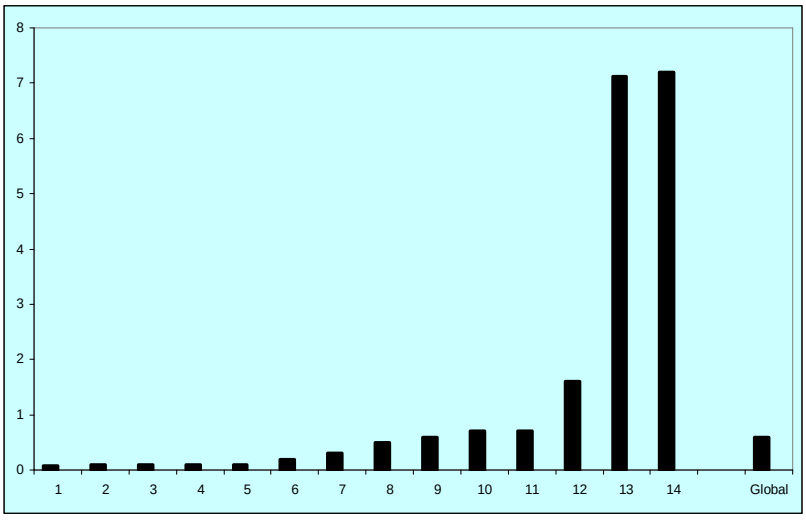

Fig. 3. HIV prevalence by ethnicity* (CDC, 2008) or region (UNAIDS, 2006): Asians and Pacific Islanders* (1), White Americans*(2), East Asians (3), American Indians*(4), Alaska Natives*(5), North Africa/Middle East (6), Western/Central Europe (7), Oceania (8), Latin America (9), South/South-East Asia (10), North America (11), Caribbean (12), Afro-Americans* (13), Sub-Saharan Africans (14)

The subpopulations of Sub-Saharan Africans are the worst affected by HIV (Fig. 3, 13 and 14). The region is home to about 10% of the world's population, but comprises 63% of all people living with HIV (UNAIDS, 2006). In contrast, the populations of North Africa north of the Sahara are characterized by very low levels of the intensity of HIV-epidemics (Fig. 3, 6). Half of all new HIV infections in the United States are among black individuals, who represent only 15% of its overall population: among 15,135 American patients with HIV, 53.6% were black (Centers for Disease Control and Prevention, 2007) and the situation has not changed (Roberts, 2012). What is more, the hope of achieving the vision of a AIDS-free generation is problematic (Abdool Karim, 2012).

The results of the first attempt to set out evolutionary and genetic reasons for this pattern of racial susceptibility were published in the early part of the 1990s (Rumyantsev, 1992, 1993a, 1993b). Nevertheless, there has been little advancement in this direction (Powe, 2003) before the conclusions have been confirmed at the genome level (O'Brien and Nelson, 2004; O'Brien and Dean, 1997).

According to data presented in Fig. 3, the rate of HIV diagnosis is 0.07% for Asians and Pacific Islanders, 0.09% for White Americans, 01% for East Asians, American Indians and Alaska Natives, 0.2% for North Africa/Middle Eastern inhabitants, and 0.3% for Western/Central Europeans, in sharp contrast to Afro-Americans (7.13%) and sub-Saharan Africans (7.2%).

The data indicates that many native inhabitants of the sub-Saharan tropical region and even their distant relatives who were transplanted to America have a sharp deficit of hereditary immunity against HIV infection. That means that their faraway ancestors did not face this kind of virus in their ancient evolutionary history and were thus not exposed to natural selection for hereditary immunity against HIV infection. The intercommunication of the sub-Saharan branch of *Homo sapiens* with other subdivisions of the species was interrupted over 60,000 ya when the ancestors of other parts of humankind moved east out of Africa. This means that the emergence of human HIV infection, as well as the launch of hereditary immunity of Eurasians against HIV, cannot have occurred earlier than 60,000 ya.

The ancestors of current Australian aborigines also left Africa 75,000–62,000 ya. They concluded their wanderings 50,000 ya (Fig. 2) and since then lost intercommunications with other parts of humankind. Colonization of the continent by Europeans began in 1780. It brought in to the aboriginal population viral infections capable of being transmitted from human to human. Now, indigenous Australians are at greater risk of HIV transmission than non-indigenous inhabitants of the same continent. There are similarities between the epidemiology of HIV infection in indigenous Australians and that observed in sub-Saharan Africa (Wright et al., 2005). For instance, in contrast to Eurasians, heterosexual intercourse is the main route for HIV transmission among both aboriginal Australians and sub-Saharan Africans, and women have a greater vulnerability of acquiring HIV. Indigenous females were 18 times more likely to be infected than non-indigenous females, and three times more likely than non-indigenous males.

Notification rates for HIV infection in indigenous Australian people have been higher than in non-indigenous people. Indigenous males were twice as likely as non-indigenous males to be infected with HIV (Guthrie et al., 2000). Indigenous Australians did not show the decline in HIV that occurred among non-indigenous Australians (Wright et al. 2005). Nevertheless, the Australian HIV epidemic situation was complicated by very specific ethnic peculiarity: the aboriginal sexual network strongly identifies who is having sex with whom, how often and

where (Bowden, 2005). This means that the emergence HIV infection as well as the launch of hereditary immunity against HIV was not occurred among aboriginal Australians over all 50,000 years long period of their geographical isolation. The appearance of HIV epidemics among Australian aborigines and the launch of appropriate selection for hereditary immunity against HIV can be induced by European colonization only over 200 years ago.

The first settlers of America separated from their Eurasian relatives between 30,000 and 15,000 ya (Fig. 2). Currently, their direct inheritors (American Indians, Alaska Natives, Fig. 3, positions 4 and 5) reveals the same high grade of hereditary immunity against HIV infection as their Eurasian, Polynesian and Latino American relatives (Fig. 3). The infection could not emerge after 14000 ya; otherwise indigenous Americans would not demonstrate very expressive immunity against HIV. The set of above integrated data indicates the emergence of human HIV infection, as well as the launch of hereditary immunity against HIV among Eurasians, Polynesians, indigenous Americans and so on between 30,000–15,000 ya and locates the event on the Eurasian continent.

3.4.2 Evidence of emergence of influenza

The Spanish influenza H1N1 (1918–1919) was the deadliest human pandemic in recent history, killing more people than any other disease of similar duration. It annihilated close to 2% of the global population of the time (Ungchusak et al., 2005). According to a WHO estimate (Stohr, 2002), only 0.008% of the current world population dies of influenza per year. The recent 2009 pandemics of H1N1v2009 influenza had a hundred times fewer victims (Lipsitch et al., 2009). This strain of influenza, which was feared to cause severe respiratory distress and illness, did not have as severe effect as predicted (Laurent et al., 2012). At the same time, a significant proportion of humans were saved from the cruel disease by their hereditary traits of specific innate immunity to influenza (Rumyantsev, 2008, 2006).

The painful selection of humans for inherent immunity against influenza can be performed beginning from the first appearance of influenza virus among ancient humans who were initially extremely susceptible to the infection. The selection transformed cardinally the immune state of Eurasian part of ancient humankind. But indigenous populations of African, American and Australian continents did not possess such hereditary protective traits.

Since reaching Australia ~50,000 ya (Fig. 2), aboriginal Australians have been in almost total isolation from other subpopulations of the species (Rasmussen, et al., 2012). Colonization of the continent by Europeans began in 1780 and interrupted the geographical isolation of Australians. The first colonists introduced to the aboriginal population viral infections, including influenza. The consequent epidemics laid waste to the indigenous populations of the continent and revealed their extreme susceptibility to influenza in contrast to the inhabitants of the Eurasian continent. These facts evidence the virginity of the Australian branch of *Homo sapiens* in relation to influenza that existed over ~50,000 years and was only broken near 200 years ago. This can be taken as evidence that influenza could not have appeared among the Eurasian part of humankind earlier than 50,000 ya.

The indigenous peoples of North and South America had begun to settle these continents nearly 15,000 years ago and since then had them only to themselves. The sizes of their populations have remained essentially constant for many centuries, but that changed dramatically when Columbus's discovery of the New World launched relentless waves of European colonization. The newly arrived infection's effect on the Amerindians, originally a

highly susceptible, non-immune race, was devastating. It was a weapon so powerful that Native Americans feared it more than bullets and swords (Stearn and Stearn, 1945; Thornton, 1987).

Both archaeological and historical records indicate that European contact and colonialism initiated a significant reduction in the indigenous population through warfare, enslavement, societal disruption, and especially widespread epidemic disease (Stearn and Stearn, 1945; Thornton, 1987). Crucial epidemics, warfare, enslavement and famines resulted in significant population declines among Native Americans during the 16th century. Additionally, the scale of the contraction suggests that the depopulation was not localized to particular regions or communities, but was instead likely to have been widespread or to have had an especially severe impact on the most populous regions (O'Fallon and Fehren-Schmitz, 2011). At the same time, the infections were not as dangerous for Europeans as for Native Americans. The populations of European conquistadors and colonists increased sharply over the same period. This is evidence that most Europeans had acquired genetic immunity against influenza long before the time of the great geographical discoveries.

These facts demonstrate that influenza infection had not been met by indigenous Australians and Americans before European colonization of these continents. In contrast, the Eurasian population of humans adopted the disease after the exodus out of Africa (75,000 - 62,000 ya) and after isolations of Australians (50.000 ya) but not earlier then 14.000 years before the present. Thus, current estimation of the first emergence of influenza among humans is between 50,000 ya and 15,000 ya.

3.4.3 Emergence of measles

Before widespread global use of measles vaccination, the disease killed 0.4% of the worldwide population in 1980 (Wolfson et al., 2007) and nearly 0.1% in 2000 (CDC, 2009). In contrast, epidemics of measles are currently a major cause of childhood mortality in West Africa. Thus, most but not all currently living people are inherently immune to measles infection. This evidences the performance among most ancient humankind of very strong natural selection for the traits of hereditary immunity against the disease. Meanwhile, infection with the same virus may be extremely disastrous for people the ancestors of whom did not confront this specific infectious agent.

This happened to the primary settlers of Australia (Rasmussen et al., 2012) and America when European newcomers penetrated their 50,000 year-long isolation and brought to indigenous Australians a set of viral infections including measles, which had devastating effects on their population (Webb, 1995). The same occurred with the primary settlers of America, whose isolation occurred nearly 14,000 years. The integration of these epidemiological, immunological and anthropological data and their sensing from the position of evolutionary ecology of infectious diseases allow for the estimation of the emergence of human measles in the territory of Eurasia not earlier than 15,000 ya.

3.4.4 Adoption of smallpox

Aboriginal settlers reached Australia ~50,000 years ago (Rasmussen et al., 2012). In 1788, Australia probably had between 750,000 and one million people. During the European colonization of Australia, the health of the aboriginal population declined rapidly in the face of highly infectious diseases, including smallpox, which occurred as early as 1789 at Sidney Cove. With the introduction of smallpox, the Sydney aboriginal population of 1500 was

reduced dramatically in the first years of European contact. The bodies of aborigines were reported floating in the Sydney harbor and found in foreshore rock shelters. Two years later, almost half of the entire indigenous population died in the smallpox epidemic of 1789 and it is said that only three people were left by 1791. Smallpox was thus a major catalyst for the decline of traditional aboriginal society during 19th century (Webb, 1995). The integration of this historical fact with the absence of hereditary immunity against smallpox among aboriginal Australians evidences that the adoption of smallpox by humankind could not have occurred before 50,000 years ago.

European colonization also induced widespread mortality among indigenous Americans (Stearn and Stearn, 1945; Thornton, 1987). For instance, according to the records of Franciscan friar Fray Toribio de Benavente, during the 16th century the Mexican territory was extremely full of people, but when the smallpox began to attack the Indians it became so great a pestilence among them that in most provinces, more than half the population died (Foster, 1973). Recent comparative researches of genome's ancient and contemporary mitochondrial sequences confirmed that Native American populations suffered a significant, although transient, contraction in population size some 500 years before the present (O'Fallon and Fehren-Schmitz, 2011).

Because the isolation of indigenous Americans from the Eurasian population occurred 14,000 years ago, the adoption of smallpox and the selection for hereditary immunity against the disease could not have occurred before this date. In the case that Eurasians first contracted smallpox from cattle, the adoption could be performed 10,000 ya after the domestication of cattle by inhabitants of the Middle East. The resulting estimation of the first emergence of smallpox among humans can be determined as occurring between 14,000 and 10,000 ya.

4. CONCLUSION

The present research was based on integration and consequent sensing of appropriate recent achievements of evolutionary sections of epidemiology, immunology and anthropology. The results of the investigation demonstrate that the origins of today's HIV, influenza, measles and smallpox epidemic processes have roots in the far more distant past than have been thought before. These infections, the existence of which depends on regular transmission of infections from one organism to another, have not been adopted by early Homo sapiens in the African Savannah, the place of human descent but over the wanderings of early humans outside of Africa.

The wanderings began 75,000–62,000 ya along different geographical directions and ecologically various parts of the world and thus substantially expanded both the quantity and quality of infectious agents they encountered on their way. In addition, over the dispersion of humankind, most infectious agents and principal protective genes flowed between different human populations by mixing together. This was most likely to have been realized mainly on the Eurasian territory.

Some populations appeared to be geographically isolated from this process at different times of their history. The African sub-Saharan population was excluded 60,000 ya, the Australian population 50,000 ya, and the American population 14,000 ya. Integration of these anthropological data with recent achievements of evolutionary immunology and epidemiology allows us to estimate that the emergence of considered human viral infections

among humans occurred non-simultaneously on the Eurasian territories between 14,000 and 10,000 ya.

After emergence, the epidemics and the traits of hereditary immunity against them has continued to exist among humankind until now, supported by mitigated forms of relevant infections. The strategy and tactics of infectious prophylactic should be oriented toward the identification and defense of both homozygous and heterozygous susceptible individuals.

COMPETING INTERESTS

Author has declared that no competing interests exist.

REFERENCES

Abdool Karim, S.S. (2012). An AIDS-Free Generation? Science, 337, 133.
Balter, M. (2011). Genes confirm Europeans' blow to native American. Science, 334, 1335.
Black, F.L. (1975). Infectious diseases in primitive societies. Science, 187, 515-518.
Black, F.L. (1966). Measles endemicity in insular populations: critical community size and its evolutionary implication. J Theor Biol., 11, 207-211.
Bowden, F.J. (2005). Controlling HIV in Indigenous Australians. Med J Aust, 183, 116-117.
Burgasov, P.N., Rumyantsev, S.N. (1974). Evolution of Clostridiosis [in Russian]. Moscow: Meditsine.
CDC. (2008). HIV/AIDS among American Indians and Alaska Natives, HIV/AIDS Fact Sheet ed CDCCDC.
CDC. (2009). Global measles mortality, 2000–2008. MMWR, 58, 1326.
Centers for Disease Control and Prevention. (2007). AIDS Surveillance-General Epidemiology: Estimated Number of Persons Living with AIDS by Race/Ethnicity, 1993-2003.
Cockburn, T.A. (1971). Infectious diseases in ancient populations. Curr Anthropol, 12, 45-62.
Foster, E.A. (1973). Motolinia's History of the Indians of New Spain. Westport, CT: Greenwood Press.
Gaggiotti, O.E. (2006). Evolutionary population genetics: Were the Vikings immune to HIV? Heredity, 96, 280-281.
Gao, F., et al. (1999). Origin of HIV-1 in the Chimpanzee Pan troglodytes troglodytes. Nature, 397, 436-441.
Guthrie, J.A., et al. (2000). HIV and AIDS in Aboriginal and Torres Strait Islander Australians: 1992-1998. Med J Aust, 172, 266-269.
Haldane, J.B.S. (1949). Disease and evolution. La Riserca Sci, 19S, 68-76.
Khor, C.C., Hibberd, M.L. (2011). Revealing the molecular signatures of host-pathogen interactions. Genome Biol., 12, 229.
Laurent, C., et al. (2012). Viral Epidemiology and Severity of Respiratory Infections in Infants in 2009: A Prospective Study. Pediatr Infect Dis J.
Lipsitch, M., et al. (2009). Managing and reducing uncertainty in an emerging influenza pandemic. New Engl J Med, 361, 112-115.
Lucotte, G., Dieterlen, F. (2003). More about the Viking hypothesis of origin of the delta32 mutation in the CCR5 gene conferring resistance to HIV-1 infection. Infect Genet Evol., 3, 293-295.
Novembre, J., Stephens, M. (2008). Interpreting principal component analyses of spatial population genetic variation. Nat Genet, 40, 646-649.

O'Brien, S.J., Nelson, G.W. (2004). Human genes that limit AIDS. Nature Genet, 36, 565-574.

O'Brien, S.J., Dean, M. (1997). In search of AIDS-resistance genes. Scientific American, 277, 44-51.

O'Fallon, B.D., Fehren-Schmitz, L. (2011). Native Americans experienced a strong population bottleneck coincident with European contact. PNAS, 108, 20444-20448.

Paxton, W.A., et al. (1996). Relative resistance to HIV-1 infection of CD4 lymphocytes from persons who remain uninfected despite multiple high-risk sexual exposures. Nat. Med., 2, 412-417.

Polunin, I.V. (1953). Diseases in contemporary ad ancient hunter-gatherers. Med. J. Malaya, 8, 55.

Powe, N.R. (2003). To have and have not: Health and health care disparities in chronic kidney disease. Kidney Int., 64, 763-772.

Rasmussen, M., et al. (2012). An Aboriginal Australian genome reveals separate human dispersals into Asia. Science, 334, 94-98.

Roberts, L. (2012). HIV/AIDS in America. Introduction. Science, 337, 167.

Rumyantsev, S. (2010a). Selection for Hereditary immunity to infections during the evolution of humankind. WebmedCentral HUMAN BIOLOGY 2010; 1(10):WMC001033. WebmedCentral HUMAN BIOLOGY WMC001033 1, 1-36.

Rumyantsev, S.N. (2002). Structural predestination of individual diversity in the course and severity of infectious diseases. The Scientific World Journal, 2, 205-216.

Rumyantsev, S.N. (1977). Immunity and molecular constitution. Zh Obtsch Biol, 38, 500-511.

Rumyantsev, S.N. (2006). Genetic immunity and influenza pandemics. FEMS Immunology and Medical Microbiology, 48, 1-10.

Rumyantsev, S.N. (1992). AIDS: Fears are exaggerated. Moscow News, 1992, August 30.

Rumyantsev, S.N. (1993a). AIDS: Is it a big problem for Eurasia? IX International Conference on AIDS, 139. Berlin.

Rumyantsev, S.N. (1993b). Diagnosis of inherent predisposition to AIDS, IX International Conference on AIDS, I, 157. Berlin.

Rumyantsev, S.N. (2008). Hereditary Immunity: Fundamental Principles and Exploitation in Life Study and Health Care. New York: Nova Biomedical Books.

Rumyantsev, S.N. (2010b). Bioecology of pleistocenic spurt in anthropogenesis. Int. J. Integ. Biol., 10, 14-21.

Rumyantsev, S.N. (2012). Dating of first emergence of human epidemics. J. Med. Med. Sci., 3, 423-433.

Rumyantsev, S.N. (2004a). Toward Molecular Level of the "*Salmonella*-Victim" Ecology, Genetics and Evolution. The Scientific World Journal, 1, 193-199.

Rumyantsev, S.N. (2004b). The best defence against bioweapons has already been invented by evolution. Infection, Genetics and Evolution, 4, 159-166.

Rumyantsev, S.N. (1997). Chemical ecology and biomolecular evolution. Acta. Biotheor., 45, 65-80.

Stearn, E.W., Stearn, A.E. (1945). The effect of smallpox on the destiny of the Amerindian. Boston: B. Humphries, Inc.

Stohr, K. (2002). Influenza - WHO cares. Lancet Infectious Diseases, 2, 517.

Thornton, R. (1987). American Indian holocaust and survival. Norman: University of Oklahoma Press.

UNAIDS. (2006). AIDS Epidemic Update: December 2006. Geneva, Switzerland: Joint United Nations Programme on HIV/AIDS (UNAIDS) and World Health Organization (WHO).

Ungchusak, K., et al. (2005). Probable person-to-person transmission of avian influenza A (H5N1). Engl J Med., 352, 333-340.

Webb, S.G. (1995). Paleopathology of Aboriginal Australians, Cambridge University Press.
White, T.D., et al. (2003). Pleistocene Homo sapiens from Middle Awash, Ethiopia. Nature, 423, 747.
Wolfson, L.J., et al., Measles Initiative (2007). Has the 2005 measles mortality reduction goal been achieved? A natural history modelling study. Lancet, 369, 191-200.
Worobey, M., et al. (2008). Direct evidence of extensive diversity of HIV-1 in Kinshasa by 1960. Nature, 455, 661-664.
Wright, M.R., et al. (2005). Fulfilling prophecy? Sexually transmitted infections and HIV in Indigenous people in Western Australia. Med J Aust., 183, 124-128.