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**MUTUAL BIOLOGICAL SOCIAL  
EVOLUTION, GENETIC  
DIVERSITY AND SOCIAL  
CHANGE: THE CASE OF  
ALCOHOL AND EUROPEAN  
COLONIZATION**

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## MUTUAL BIOLOGICAL SOCIAL EVOLUTION, GENETIC DIVERSITY AND SOCIAL CHANGE: THE CASE OF ALCOHOL AND EUROPEAN COLONIZATION<sup>2</sup>

The research project aims to find link between genetic diversity and social change. Although some studies associate certain genes with prosocial behavior, it is hardly to say that any genetic polymorphisms are responsible for social change. We assume that some existing differences in particular genotypes could be explained by extent of ancient urbanization, change in population density and historic pathogen prevalence. The pathogen load might have led to some genetic mutations that in their turn might have caused difference in some allele frequency among regions and populations.

Our case study is the use of strong alcohol as factor of European colonization in America, Africa and Eurasia. Historically, alcohol was one of the major trade items between Europeans and indigenous populations. I argue that there is a positive correlation between probability of being colonized by Europeans and particular allele frequency responsible for metabolism of alcohol. The risk of colonization by European powers is higher for indigenous populations which had genotype with lower allele frequencies which could 'protect' them against alcohol abuse. I test this hypothesis using binary logistic regression. The dependent variable is the binomial variable which is coded *colonization1900* of a given native population by Europeans from the 1500s to 1900. The unit of analysis is not a state, but a population. Independent variable is allele frequencies of Arg48His polymorphism among 56 populations from Africa, Asia and America. The suggested causal mechanism is uneven trade: the exchange of alcohol for local goods was unfavorable for indigenous populations. Economic dependence was followed by colonization.

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## *Introduction*

How can non-social factors affect social change? Without any intention to reject the importance of social factors on social developments, I try to put emphasis on the relative importance of non-social factors. Among the studied factors are natural disasters (Lazarev et al., 2012), geography and climate (Diamond, 1997), and infectious diseases (Murray, Schaller, 2010). These factors may affect voting behavior, cultural values and even the success of modernization. It is a broad theoretical framework that includes various explanatory mechanisms. In particular, I am looking at genetic diversity as a factor of social change. Can genetic diversity affect social change?

The development of mankind has two components - biological and cultural/social evolution. Biological evolution is a long, slow process, based on natural selection of species and adaptation to environment change. Social/cultural evolution is a relatively faster process which includes all forms of social and cultural developments. These processes are not mutually exclusive; there is interdependence between these cycles (Dobzhansky, 1973). I assume that there is interaction between biological and cultural evolution.

Genes affect the individual level. As we know, many diseases have a genetic basis, and many of these are explained by the effects of a single gene. Also many individual-level differences in personality traits are strongly influenced by genetic differences. The main question addressed in this paper is whether and how genetic diversity affects the societal level?

I suggest a new theory of gene-environment interaction that puts emphasis on the importance of historical pathogens prevalence and urbanization. It is argued that urbanization may lead to genetic change due to increased pathogens load, and later variation in certain genes frequency may affect further social change. I test this assumption using data on genotype frequency (Arg48His) associated with alcohol metabolism and probability of colonization of non-European populations by European powers by 1900. Using a set of binomial regressions, I find that higher Arg48His frequency is associated with a lower probability of being colonized by European powers. The suggested causal mechanism is alcohol trade.

This paper attempts to make an important contribution to the fast-growing field of culture-gene research. I take one step further comparing with many gene-environment or culture-gene studies (e.g., Barnes, 2010; Chiao, Blizhinsky, 2010; Cook, 2013; Dawes, Fowler, 2009; Fowler, Dawes, 2008; Peng et al., 2010) by incorporating a three element theory: social change – genetic change – social change. I define it as a mutual social biological evolution theory.

This article consists of six sections. The first part presents the theoretical framework of mutual social biological evolution. The second section includes an empirical analysis of the relationship between ancient urbanization and historical pathogens load based on data from 17 historical geopolitical regions. The third part contains a historical overview of the relationship between alcohol consumption and European colonization, and links genetic factors with alcohol consumption. The fourth part presents data and methods for empirical testing of our hypotheses. The fifth section presents the results of our binary logistic regression models that confirm our theoretical arguments. The final part concludes.

### ***Mutual biological social evolution cycle***

Various studies show that the relationship between genetic diversity and social change is complicated. It is not a one-direction effect: it is a mutual co-evolutionary cycle – social change may affect genes and, vice versa, genes may affect social change.

*Social change may affect genes.* The speed of biological and social evolution does not coincide. Rapid developments on the social level may not be followed by adaptation on a biological level. Under certain conditions, social change has an impact on biological evolution. The literature presents some evidence that social and cultural evolution may affect genetic selection.

The ‘thrifty genotype hypothesis’ argues that the human genotype is historically designed for hunters-gatherers. The set of diseases is caused by progress and civilization: obesity, diabetes, and hypertension. One possible explanation of this phenomenon is that social and cultural progress makes some genes ‘irrelevant’. For thousands years, hunter-gatherer populations were living in the ‘feast – famine’ cycle. To ensure survival during periods of famine, certain genes evolved to regulate efficient intake and utilization of fuel stores. Now, the combination of continuous and permanent food abundance and physical inactivity eliminates the evolutionarily programmed biochemical cycles emanating from feast-famine and physical activity-rest cycles. This, in turn, abrogates the cycling of certain metabolic processes, ultimately resulting in metabolic derangements such as obesity and Type 2 diabetes (Chakravanty, Booth, 2004; Brooks, 2011).

For many thousands of years human populations adapted to low-carbohydrates, low-cholesterol, low-calories and low-salt diet; and a thrifty genotype reflected it. At the present times, change in lifestyle, population size, economy, climate change and diet are among factors affecting genetic selection. One can make the conclusion that every stage of social evolution or social progress requires a specific or relevant genotype. Social evolution leads to radical shifts in human survival strategies. Hunter-gatherers require one set of genes and settlers another set, while modern people with a sedentary lifestyle may not need some of these genes.

*Genetic diversity may affect social change.* In general, genes are not likely to carry any cultural information like norms, values, customs, etc. Cultural values and social behavior is likely to be transmitted through social mechanisms – learning, education, etc. However, some research claims that some genes are associated with prosocial behavior. The famous paper by Chiao and Blizinsky (2010) argued that 5-HTTLPR allele – a serotonin transporter gene – is associated with some cultural values: prevalence of collectivism and individualism. However, this paper was heavily criticized (Eisenberg, Hayes, 2010). It was argued that these findings were methodologically weak: the sample includes de facto non-independent cases; not only cross cultural correlation, but in-cultural-zone correlation is required. In addition, such analysis should incorporate the effects of neutral genetic processes (Mattews, Butler, 2011). The hypothesis about the purely geographic explanation of this gene variation cannot be refuted.

Various dopamine receptor polymorphisms are associated with prosocial behavior: low neuronal activity and increased exploratory behavior, novelty seeking, and risk-taking (Mattews, Butler, 2011). Eisenberg et al. tested dopamine receptor polymorphisms DRD4/7 and DRD2/A1 and found that certain polymorphisms had different effects among nomads and settled populations.

Based on analyses of nomadic and recently settled Ariaal men in northern Kenya, they found that DRD4 7R+ genotypes were associated with better nutritional indices among nomads, but worse indices among settled individuals. They have suggested that this allele provides additional benefits to nomads compared to settlers, thus, depending on social context (Eisenberg et al., 2008).

Another finding claimed a positive association between altruism and COMT Val158Met polymorphism, one of the dopaminergic gene variants (Reuter et al., 2011). Based on experimental study, this paper revealed that carriers of at least one Val allele were twice as altruistic as participants without a Val allele.

There is still a big problem. Even if research reveals positive association between certain polymorphisms and prosocial behavior, it might be difficult to put these results in a broader framework. Genetic mutations and natural selections are much slower than social change.

Assuming that biological and social evolutions are complimentary, I suggest a new theoretical perspective. Since the Neolithic Revolution, the population density of certain areas is rising faster than in others. This may be reflected in rapid urbanization and the emergence of ancient states. Historically, these areas were the Middle East, Egypt, Southern Europe, the Indian subcontinent, South Eastern Asia, and Central America. Due to many reasons, statehood is strongly associated with increased population density and urbanization (Diamond, 1997). Urbanization is often seen as proxy for complex social organization: the size of the largest city in a region is a function of the scale of political organization (Morris, 2013: 165). Many ancient cities even at 1000 BCE had a large population by contemporary standards – like Sian (China) with 100,000 residents, or Thebes (Egypt) with 120,000 (OpenHistory Project). Urban settlements with a population more than 1000 residents can be dated as early as 7000 BCE (Morris, 2013). Increased population density – via emergence of urban settlements – leads to growth in infectious diseases. These infections may lead to some genetic mutations both as the emergence of new alleles and as change in allele frequencies of particular genes. Therefore, ancient urbanization may be associated with pathogen history.

There is a vast literature on the relationship between ancient urbanization and the rise and spread of infections: ancient urbanization is strongly associated with the increase in pathogens load. Urban settlements show larger populations and much higher population density which facilitates the emergence and spread of particular infections, or ‘crowd diseases’ (Clark, 2010; Barnes, 2005; Cook, 2013a; Shug et al., 2013; Miksic, 1999). Some infections (e.g. childhood diseases) have emerged relatively recently – and particularly in the urbanized areas; before the emergence of large cities some infections had not existed yet. Viruses need some population threshold for emergence and ‘survival’. Ancient cities, even if they had some sanitation facilities, were characterized by low hygiene standards. For example, Clark (2010) argues western European cities achieved ancient Roman hygiene standards only in the 19<sup>th</sup> century. The rise of urban settlements is also proxy for a certain level of social development: a) a transition to sedentary agriculture – often involving the domestication of animals, the cohabitation of humans with disease vectors like rodents and insects, and a shift to poor monocereal diets, which also weaken the human immune system and expose people to infections, b) relatively high levels of population mobility within urbanized areas, and c) the existence of dense trade, cultural and military contacts (and the transmission of infections) between various communities.

It can be thus defined as a reciprocal effect: on the one hand, ancient urbanization is likely to lead to the explosion of infectious diseases that became the main mortality reason for the population. Clark (2010) claims that historically, urbanization had a negative effect on population growth in society – urban population growth occurred only due to migrations from rural areas. But on the other hand, earlier urbanization means the early start of selection of genes associated with natural resistance to infections.

Pathogens may cause genetic mutations. Infectious diseases affect natural selection: carriers of genes resistant to diseases are ‘winners’ of evolution. Genetic resistance to particular pathogens may be associated with other biological traits. Therefore, I argue that ancient urbanization may cause genetic mutations. At least one paper supports this argument (Barnes et al., 2010). Ancient urbanization is reported to be connected with the frequency of an allele (SLC11A1 1729 + 55del4) associated with natural resistance to intracellular pathogens such as tuberculosis and leprosy. They found a highly significant correlation with duration of urban settlement: populations with a long history of living in towns are better adapted to resisting these infections. I suggest a very similar causal mechanism in our case. Earlier urbanization means earlier epidemics in the ancient cities; populations with an early urban history start the process of selecting genes that are resistant to these infections earlier. Therefore, populations with a longer urban history may have a different frequency of particular alleles. I assume that to be a mechanism for social factors to affect genetic selection.

I argue that such mutations may affect social evolution in the long run. Variance in some frequencies of particular alleles may become important for social change (Table 1).

Table 1. Mutual social-biological evolution cycle

<i>Social change</i>	<i>Biological change</i>	<i>Social change</i>
States / Population density	Genetic mutations/ Change in allele frequencies	Social change
Ancient urbanization		

The next section provides preliminary empirical testing for our theory: finding an association between early ancient urban centers and the rise of historical pathogens prevalence.

### *Ancient urbanization and pathogens prevalence*

I collected data on ancient urbanization at 650 BCE and correlated them on the Murray and Schaller index of historic pathogen prevalence (Murray and Schaller, 2010), (Table 2). I take data for ancient urbanization from the OpenHistory Project; for some areas and urban settlements, data were checked with original source of the above mentioned OpenHistory Project (Chandler, 1987). For 650 BCE there are records of urban settlements for 20 geopolitical areas in Africa, Asia, Europe and America. Next, I excluded three regions from the initial sample: Mexico (as a region from the New World), Yemen and Sudan (as standing apart from major ancient civilization centers). Based on this data I constructed two variables (see Table 2):

*Number of cities to 650 BCE* – the number of cities that existed in a particular geopolitical area at 650 BCE (not including those one that were destroyed or abandoned before this date).

*Urban population estimate* – an approximate estimate of urban population in all existing cities in a particular geopolitical area.

Table 2. The list of geopolitical areas at 650 BCE

<b>№</b>	<b>Geopolitical area</b>	<b>N of cities</b>	<b>Urban population estimate</b>
1	Mexico	2	13900
2	Spain	2	30000
3	Italy	2	46000
4	Greece	3	70000
5	Turkey	3	71000
6	Tunisia	1	50000
7	Egypt	6	143000
8	Syria	3	60000
9	Israel	5	115000
10	Georgia	1	4000
11	Armenia	2	36000
12	India	4	105000
13	Uzbek	1	20000
14	Iran	4	97000
15	Iraq	6	295000
16	Korea	1	30000
17	China	8	218000
18	Yemen	1	35000
19	Sudan	1	45000
20	Lybia	1	1500

Source: Chandler (1987); OpenHistory Project (<http://openhistory.net/>)

These correlations show that ancient urban population correlates with the index of historic pathogen prevalence, both 7 and 9 items (Table 3):

Table 3. Ancient urbanization (c. 650 BCE) and pathogen prevalence

	Pathogen history 7 items	Pathogen history 9 items
<b>N of cities to 650 BC</b>	<b>0.563*</b> (p=0.018)	<b>0.594*</b> (p=0.012)
<b>Urban population estimate</b>	0.480 (p=0.051)	<b>0.588*</b> (p=0.013)

N=17; \* - significance on 0.05 level, \*\* - significance on 0.01 level

Source: Murray, Schaller, 2010; Chandler, 1987; OpenHistory Project (<http://openhistory.net/>)

Urban population correlates with the 9-item index of historical diseases (0.583, p=0.013), and it is almost significant for the 7-item index (0.480, p=0.051). I also found significant correlations between the number of ancient cities and both 9-item (0.594, p=0.012) and 7-item indices (0.563, p=0.018) of historical diseases.

Murray and Schaller composed their historical pathogen prevalence indices based on two sources – Rodenwaldt’s and Bader’s, *World-atlas of epidemic diseases* (Rodenwald and Bader, 1952, 1961) and Simmons et al.’s, *Global epidemiology*. (Simmons et al., 1944). I referred to these editions to obtain data on separate infections: leishmaniasis, schistosomiasis, trypanosomiasis, leprosy, malaria, typhus, filariasis, dengue, and tuberculosis.

On the level of separate infections in this sample, both *Urban estimate* and *Number of cities* are correlated with leishmaniasis - 0.749 (p=0.001) and 0.746 (p=0,001), dengue – 0.745 (p=0,001) and 0.699 (p=0.002), schistosomiasis - 0.545 (p=0.024) and 0.585 (p=0.014), and leprosy - 0.554 (p=0.026) and 0.524 (p=0.037).

Based on this preliminary finding, our main argument is as follows. Possible genetic mutations, accumulated due to the rise of population density in certain areas may not only lead to ‘pure’ biological change like variation in frequency of some allele/genes, but also to social change. Variation in frequency of particular genes may become a factor of social change.

This paper aims to provide an empirical illustration for one case: the use of strong alcohol as a factor of European colonization. I argue that variation in the frequency of Arg48His allele (responsible for metabolism of alcohol) among populations was one of the factors that influenced the probability of European colonization. I collected data on ancient urbanization for the 56 populations mentioned below and correlated the history of urbanization (defined as ‘2000 minus year of foundation of the first urban settlement’) with frequency of Arg48His allele among those populations. Correlation was marginally significant:  $r = 0.270$  (p=0.044). The chosen research strategy is to test our basic assumption about genetic diversity and social change in retrospective comparative research.

### ***Alcohol dependence and European colonization***

Our case study for testing the hypothesis about the existence of the above mentioned *mutual cycle of biological social evolution* is the use of strong alcohol as a factor of European colonization in America, Africa and Eurasia. The onset of European colonization can be dated as the late 15<sup>th</sup> century – the Columbus expedition to America. Within 300-400 years, Europeans colonized America, Australia, the largest parts of Africa, and many parts of Eurasia.

Alcoholic beverages were widespread thousands of years ago, but the invention and diffusion of distilled alcohol is a relatively recent phenomenon. The word “alcohol” is of Arabic origin; usually the invention of distillation in the Arab world is dated from the 9<sup>th</sup> century. However, the re-invention of distillation and mass spread of strong alcoholic beverages in Europe and then around the world can be dated from the 14-15<sup>th</sup> century. This coincides with the start of European colonization.

I have numerous pieces of historical evidence from America, Asia, and Africa indicating that native populations suffered not only with ‘guns, germs and steel’ (Diamond, 1997), but liquor as well. Alcohol was among the major trade items with native populations across Asia, Africa and the Americas. The alcohol trade significantly influenced the growth of the world economic system in the early modern world. Alcohol was sold to the New World in exchange for everything from timber to slaves. All major colonial powers – Britain, France, Spain, Portugal, the Netherlands and Russia – relied on alcohol in their trade, at least to some extent. In Western Africa alcohol, especially rum, became an important part of the ‘Golden triangle trade’: slaves from Africa – sugarcane in the West Indies – rum in America. As early as the fifteenth-century, Europeans began purchasing slaves from Western Africa, trading among other things, distilled spirits such as rum, gin and brandy. Alcohol became so important to the slave trade that by the late 18<sup>th</sup> century Western Africa was purchasing almost three mln liters of alcohol per year, and by the mid-19<sup>th</sup> century almost 24 mln liters per year. Overall, in the 300 year history of the Atlantic slave trade, perhaps 5-10% of all slaves were purchased with European alcohol (Hames, 2012: 48-49). The Portuguese especially relied on their trade for slaves in Angola. Records indicate that 25% of the almost 1.2 mln slaves sold out of Angola in the 18<sup>th</sup> century were bought with Brazilian cachasa (Hames, 2012: 49).

For example, the Cameroonian experience tells us that German commercial trade houses were already exporting approximately 45 million liters of spirits to sub-Saharan Africa in the mid-1800s. Between 1874 and 1888, spirits were at least half of the exports to Africa from Hamburg, then the second largest port in Europe. After 1884, spirits made up two-thirds of Hamburg's exports (Diduk, 1993: 1-2). In the late 19th century, alcohol became a currency in some African states: Nigeria (Diduk, 1993: 2) and South Africa (Hames, 2012: 84).

In North America, alcohol was one of the major trade items with Native Americans. As a result of Native American enthusiasm for alcohol, the Europeans traded it to them in exchange for their land and other goods as furs, and sometimes even sexual access to their wives (Hames, 2012: 51). The biggest profits for traders came when trading alcohol, sometimes as much as 400% (Hames, 2012: 51). Native Americans had barely consumed alcohol before the advent of Europeans. Native Americans drank in quantities to get very drunk; they believed that alcohol

contained magical powers and the ability to help them communicate with the spirits (Hames, 2012: 52)<sup>3</sup>.

As a result of European alcohol trade, in all parts of the world, indigenous drinking patterns radically changed. Although native populations often consumed alcohol beverages in the pre-colonial periods, it was available mostly during festivals and rituals. With the advent of Europeans, alcohol became available all the time; local populations were introduced to European habits of drinking daily. The consumption of alcohol skyrocketed.

All these factors, including alcohol, led to the loss of lands and independence by many native populations. Historians and scientists claim that alcohol abuse and alcohol dependence were widespread among native populations in America, Africa and Eurasia. Strong alcohol and epidemic disease were the major reasons for high mortality rates of the Indian populations in North America. Strong alcohol quickly became a major trade item. The Indians did not have any experience with alcohol and this led to alcohol abuse and alcohol dependence. Besides that, the Indians were dependent on the Europeans for their supplies. It is also true for Africa, Australia, and some parts of Eurasia (Russians in Siberia).

In one case, there is a strong evidence of the role of alcohol for European colonization. Indeed, liquor was so sought after that it led chiefs along Cameroon's coast to sign a treaty placing themselves "under the protection" of the Germans rather than the British. The chiefs, having received no correspondence from the British for a lengthy period, and being in need of rum and tobacco, took their requests to the Germans instead (Diduk, 1993: 7-8).

I found a lot of evidence that alcohol trade was one of instruments of colonial expansion in Central, Western and Southern Africa (Korieh, 2003; Blocker, Fahey, Tyrell, 2003; Eltis, Jennings, 1988; Diduk, 1993), in Siberia and Russian Far East (Foust, 1961; Gibson, 1970; Etkind, 2011), and in North America (Frank, Moore, Ames, 2000). Alcohol is also mentioned as a trade item in European imports in India (Sharma, Tripathi, Pelt, 2010), China (Cochrane et al., 2003), Japan (Higuchi, 2007), Malaysia (Jernigan, Indran, 1999), Papua New Guinea (Marshall, 1999), Iran (Yukht, 1956), and North Caucasus (Narochnitskii, 1988). I found only weak evidence (or even no strong evidence) that alcohol was a trade item in the Volga region (Russia), Central Asia, North Africa, Korea, Mexico and the Ottoman Empire. Some of these areas were populated mostly by Muslims; perhaps this explains the low demand for alcohol from these populations. At least, I found no data to prove alcohol to be a trade item.

Alcohol was surely not the only factor of European colonization – as well as technological superiority, more advanced social organization, immunity to infectious diseases, etc., but I argue that it was among the most significant factors.

If the invention of distillation is a product of social evolution, alcohol consumption is an outcome of biological evolution. The process of alcohol metabolism is associated with certain

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<sup>3</sup> Jina Hames provides an apocryphal story from 1609 about Hudson's expedition that came upon Native Americans fishing in the bay of New York. Meeting them on the island, an Englishman offered alcohol (after having drunk the first cup himself), but the chief would not drink it. Instead, a warrior came forward, drank the alcohol, and then passed out. After he woke up, he declared it was the best experience he had ever had. The rest of the group then drank the alcohol and they all became drunk. As a result, the Delaware Indians called the place Manahachtaniek, or "the island where we all got drunk". The island was later named Manhattan (Hames, 2012: 52).

genetic polymorphisms. The process is best understood in terms of the simple two-step pathway which is responsible for the bulk of alcohol metabolism. Alcohol is first oxidized by alcohol dehydrogenase (ADH) to acetaldehyde, which is then oxidized to acetate by acetaldehyde dehydrogenase (ALDH). Both proteins occur in several isozyme forms encoded by multigene families. Enzymes encoded by two gene families, alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH), mediate alcohol metabolism in humans. Allelic variants have been identified that alter metabolic rates and influence the risk of alcoholism. Specifically, Arg48His has been shown to confer protection against alcoholism, presumably through the accumulation of acetaldehyde in the blood, resulting in “flushing syndrome” – elevated blood flow, dizziness, accelerated heart rate, sweating and nausea (Mulligan et al., 2003). This allele is significantly frequent among some Asian and Jewish populations. Relatively low frequencies are detected among European, North African, and Middle Eastern populations (Mulligan et al., 2003; Borinskaya et al., 2009; Borinskaya et al., 2011). Among African populations, allele Arg370Cys is relatively frequent (Borinskaya et al., 2011). Many findings support that this allele can greatly lower the risk of alcohol dependence and alcohol abuse as well as alcohol-induced medical diseases (Li D. et al., 2011; Li H., 2011; Toth et al., 2010). Research shows that Arg48His allele frequencies are high in South Eastern Asia (China, Korea, Vietnam, Taiwan) (Chen et al., 1999), but they are low among northern Asians (Chukchi, Khanty, Nentsy etc.) and other non-Asian populations with few exceptions in the Middle East. The northern Asians do not have significant differences comparing to European populations (Borinskaya et al., 2011).

Among Native Americans, these alleles are almost absent; some studies suggested that other variations of these genes are responsible for alcohol dependence among native populations, e.g. ADH1C\*349Ile (Mulligan, 2003). Numerous studies tested various candidate polymorphisms to be predictors of alcohol dependence among Native Americans, suggesting that some variants of these genes may be protective against alcohol dependence: ALDH1A1 and ADH4 (Liu et al., 2011), ADH1B and ADH4 (Gizer et al., 2011). There is no hard evidence that there is a genetic predisposition to alcohol dependence on population level.

I tested correlation between Arg48His (Arg370Cys) allele frequency and modern alcohol consumption for 64 populations, based on the WHO data (WHO 2011). The correlation is significant:  $r=0.318$  ( $p=0.010$ ); if Koreans excluded<sup>4</sup>  $r=0.430$  ( $p=0.000$ ). That means that higher allele frequency is associated with less consumption of alcohol.

I argue that there is a positive relationship between probability of being colonized by Europeans and allele frequencies responsible for metabolism of alcohol.

## ***Data and methods***

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<sup>4</sup> Koreans are an outlier: they are reported to have very high frequency of Arg48His allele and high alcohol consumption.

## Variables

**The dependent variable** is the binomial variable which is coded *colonization1900* of a given native population by Europeans from the 1500s to 1900. The unit of analysis is not a state, but a population. The list of populations coincides with list for frequencies of Arg48His and Arg370Cys polymorphisms from the ALFRED database. I code “1” – for colonization,” 0” – for independence or non-European colonization. The great European powers (“colonizers”) in our sample are Britain, Russia, Spain, Portugal, France, Sweden and USA. Colonization is understood as colonial expansion to Eurasia/Asia, Americas, Africa and Oceania. Territorial conquests in Europe are not defined as colonization; only one is defined as a case of colonization – the Sami in Sweden.

The areas covered are Asia, Africa, the Americas and Oceania. I have 56 cases in my sample – from Asia, Africa, North America and Europe. I code “1” for only colonization by Europeans; if these populations were colonized/conquered by non-European powers (like China, Japan or Turkey), I code it as “0”. If a population was not colonized, or was colonized later than 1900, I code it as “0”. Data are checked with the State Antiquity database (State Antiquity Index, 2012). These populations are: Xhosa, Yoruba, Chagga, Ibo, Moroccans, Saharawi, Druze, Palestinians, Iranians, Darghinian, Abkhaz, Adygei, Avar, Ingush, Kalmyk, Mari, Sami, Udmurt, Kazakh, Khanty, Komi-zyrian, Altaian, Buryat, Chukchi, Tuva, Yakut, Kyrgyz, Mongolians, Tajik, Uzbek, Turks, Indians (mixed), Yanadi, Yerukula, Bunun, Dong, Ewenki, Han, Hmong, Japanese, Koreans, Tibetan, Li, Uygur, Yao, Filipino, Thai, Malaysians, Maori, Papuan New Guinea, Cheyenne, Huichol, Otomi, Pima, South-West Amerindians, and Maya.

The following three figures show sample distribution by regions (Fig.1); sample distribution by colonized and non-colonized populations (only by Europeans) (Fig. 2); and sample distribution by colonizing great powers (Fig. 3).

Fig. 1. Populations in sample by region

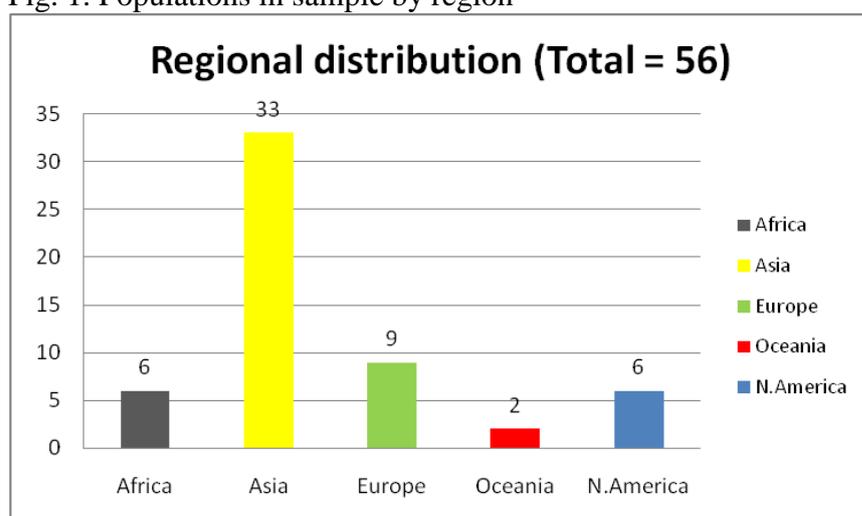


Fig. 2. Populations in sample: colonized and not colonized by Europeans

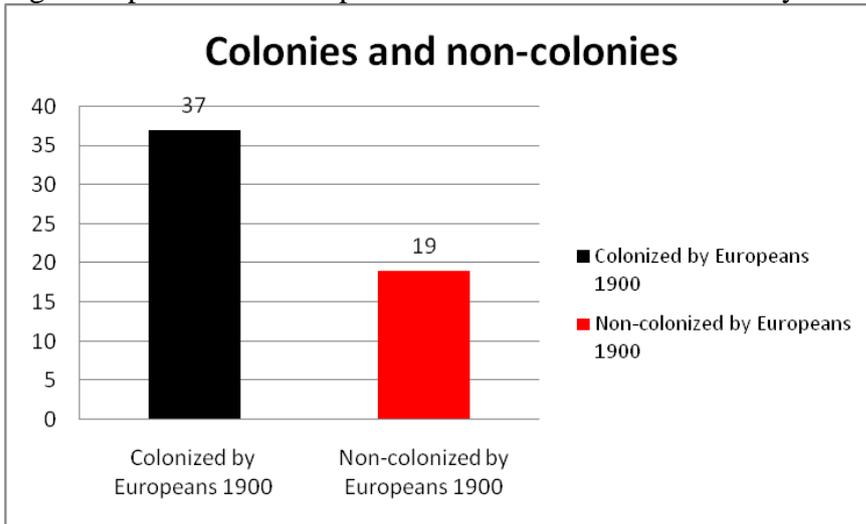
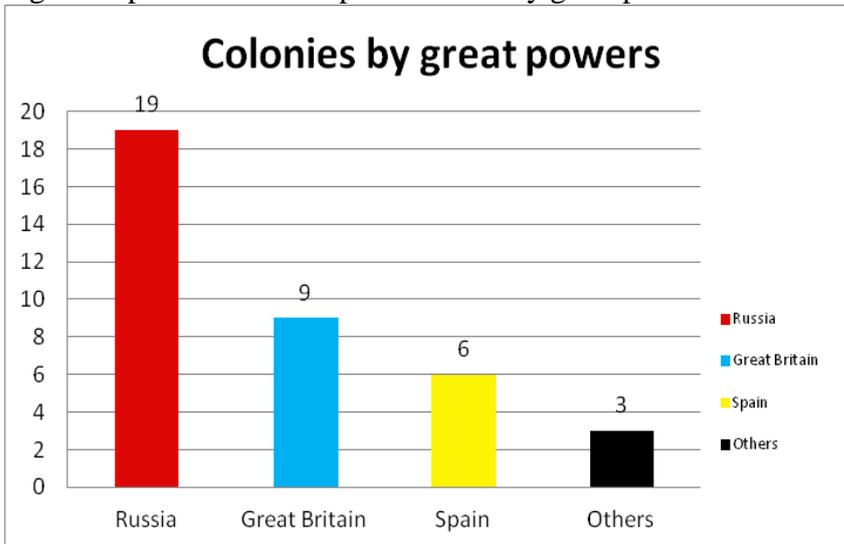


Fig. 3. Populations in sample: colonies by great powers



*Independent variables*

**Allele frequency Arg48His** - allele frequencies of Arg48His polymorphism among selected populations from the ALFRED database. The ALFRED database is in one of the largest datasets on population genetics; it provides data on hundreds of populations on thousands of alleles. For some African populations (Ibo, Yoruba and Chagga), data for Arg48His was replaced with Arg370Cys polymorphisms; it has a similar effect. For some Asian populations, data is adjusted based on recent findings (Borinskaya et al., 2011). Considering that biological evolution is a very slow process, I assume that allele frequencies do not differ from the ones 300-400 years ago. I take data for these alleles from the ALFRED database. I take the populations where the sample exceeds 90 individuals; some cases were omitted due to inability to collect data on pathogen prevalence and urbanization. Asian populations are overrepresented in the dataset; therefore, they dominate in the sample. All allele frequencies are reported in Appendix 1.

## *Control variables*

**Index of technologic development:** I suggest that technological superiority is one of the strongest factors predicting success of European colonization around the world. As Diamond argued, ‘guns and steel’ were crucial for European colonization. I measure technological development as a mean value of three indicators (“0”/“1”): *existence of writing* before colonization, *existence of firearms* (even purchased/imported), *existence of metals* before colonization (“0”/“1”). Populations with lower index of technological development are more likely to be colonized by European powers.

**Pathogen history:** To measure historical prevalence of infectious diseases, I take the index of historic pathogen prevalence for seven diseases (leishmaniasis, schistosomiasis, trypanosomiasis, malaria, filariasis, dengue, and typhus) (Murray and Schaller 2010). The higher values for this indicator have protective effect: diseases are likely to stop or slow down European colonization. They also may affect the type of colonization: a settlement or extractive one (Acemoglu, Johnson, Robinson, 2001; Acemoglu, Robinson, Johnson, 2003). I expect that the higher the index of historic pathogen prevalence is, the lower the probability of European colonization. *This data is country-based, not population-based.*

**History of statehood:** “1” – state organization exists prior to colonization; “0.5” – protostate, tribal union, statehood experience in the past; “0” – no statehood. I expect that populations having states, or at least statehood history have better chances to resist European influence, conquests and colonization.

**Economy type:** “1” – sedentary agriculture; “0.5” – nomads; “0” – hunters-gatherers. I expect that economically developed populations may have better chances of resisting European colonization.

**Population density** – rough estimations of population density in 1900. I take estimates for the population in 1900 (country-based) and refer them to contemporary country areas. I expect that smaller populations have fewer chances to resist European colonization.

**Alcohol trade** – rough estimates of alcohol trade volume between the population and European powers. I could not find reliable and precise statistical data between European powers and populations; nevertheless, I found historical sources that allow us compose this variable as a rough estimate, although not for all populations. “1” – alcohol is mentioned as a major trade item; “0.5” – alcohol is mentioned as a trade item; “0.25” – alcohol is mentioned as a minor/potential trade item, “0” – no trade evidence found. I have these data for 52 populations of 56.

**Religion** – this variable indicates whether the population belonged to a religion that contained religious permission to consume alcohol. This is true for most religions except Islam. “1” – religious permission for majority; “0.5” – Muslims are significant share; “0” – no religious permission for absolute majority/Muslims. I assume that Islam could have a negative impact on alcohol consumption and probability of colonization.

Descriptive statistics for all variables are presented in Table 4.

Table 4. Descriptive statistics for all variables

	<i>N</i>	<i>Min.</i>	<i>Max.</i>	<i>Mean</i>	<i>Std. Deviation</i>
<b>Colonization by Europeans by 1900</b>	56	.0	1,0	.661	.4778
<b>Frequency of allele Arg48His</b>	56	.000	.840	.24125	.242239
<b>History of statehood</b>	56	.00	1.00	.4107	.47775
<b>Type of economy</b>	56	.00	1.00	.8214	.29277
<b>Pathogen history</b>	56	-.91	1.19	.0564	.69080
<b>Religion</b>	56	.00	1.00	.6964	.45406
<b>Alcohol trade</b>	52	.00	1.00	.3894	.35498
<b>Technology development index</b>	56	.00	1.00	.6195	.35133
<b>Population density</b>	55	.39	122.49	21.9027	32.15176
<b>Urban history</b>	56	51	6000	1291.43	1423.401

### *Methods*

Our main argument is as follows: higher Arg48His allele frequencies followed by advanced social organization leads to lower probability of colonization by Europeans. The higher Arg48His allele frequency is associated with protective affect from alcohol abuse and alcohol dependence.

*H1:* A lower level of Arg48His allele frequencies leads to a higher probability of being colonized by a European power.

As my dependent variable is a dichotomous variable, the method of binary logistic regression is implied. To test my hypothesis, I ran three sets of regressions with *colonization1900* as DV. Firstly, I ran two models that included variables predicting the probability of European colonization by 1900, but excluded genetic factors. In the second set of models genetic variable was added. Additional specifications added religion or excluded such populations as Chinese, Japanese and Koreans as potential outliers. Finally, I ran the third set of models to test the effect of alcohol trade as a causal mechanism between probability of European colonization and the Arg48His allele frequency.

## Results

The first set of models predicts colonization without the inclusion of a genetic factor. I tested the factors most likely to affect colonization, suggesting that successful colonization depends on technologic superiority, economy type, pathogen history and population density (Table 5).

Table 5. Factors of European colonization

	<i>Unstandardized Beta – coefficients</i>	
	<i>Model 1</i>	<i>Model 2</i>
	<i>DV – Colonization 1900</i>	<i>DV – Colonization 1900</i>
Technology development index	<b>-5.429***</b> <b>(1.973)</b>	<b>-4.485***</b> <b>(1.597)</b>
Type of economy	-1.071 (1.449)	-
History of statehood	.945 (1.020)	-
Population density	<b>-.038**</b> <b>(0.016)</b>	-.020 (0.14)
Pathogen history	-	<b>-1.439**</b> <b>(0.723)</b>
<i>Cox and Snell R-square</i>	0.398	0.427
<i>-2 Log Likelihood</i>	42.980	40.241
<i>Observations</i>	55	55

\*- significance on 0.1 level, \*\* - significance on 0.05 level, \*\*\*- significance on 0.01 level

Model 1 includes technology index, type of economy, history of statehood, and population density. Technological development and population density are significant – economy type and history of statehood are not. This means that for Europeans it was easier to colonize technologically inferior and scarcely populated areas; economic development and social organization were not so important. Model 2 excludes insignificant predictors and includes the pathogen history variable. Two of three indicators are significant with predicted sign: European colonization was more successful in technologically inferior areas with lower prevalence of infectious diseases. By 1900, success of European colonization was explained by technological superiority in areas with lower prevalence of infectious diseases.

Table 6. Factors of European colonization (including genetic factors)

	<i>Unstandardized Beta – coefficients</i>		
	<i>Model 3 DV – Colonization 1900</i>	<i>Model 4 DV – Colonization 1900 China, Japan, Korea excluded</i>	<i>Model 5 DV – Colonization 1900</i>
Technology development index	<b>-5.451*** (2.098)</b>	<b>-5.233** (2.104)</b>	<b>-6.561*** (2.485)</b>
Frequency of allele Arg48His	<b>-5.188** (2.155)</b>	<b>-4.828** (2.269)</b>	<b>-4.728** (2.230)</b>
Pathogen history	<b>-1.596** (0.710)</b>	<b>-1.615** (0.711)</b>	<b>-1.569** (0.732)</b>
Religion	-	-	-1.148 (1.128)
<i>Cox and Snell R-square</i>	<i>0.478</i>	<i>0.431</i>	<i>0.488</i>
<i>-2 Log Likelihood Observations</i>	<i>35.345 56</i>	<i>35.076 53</i>	<i>34.241 56</i>

\*- significance on 0.1 level, \*\* - significance on 0.05 level, \*\*\*- significance on 0.01 level

The next set of models includes the genetic variable, Frequency of allele Arg48His (Table 6). Model 3 tests the impact of technologic development, pathogen history and genetic variable on probability of European colonization. All variables are significant, including genetic one. It means that lower Arg48His allele frequency affected probability of colonization: populations with lower Arg48His allele frequency had lower protective capacities against alcohol abuse and alcohol dependence.

Model 4 presents a robustness test. South-eastern Asian populations show very high Arg48His allele frequencies; therefore, I decided to exclude three populations as potential outliers – China, Japan and Korea – from our sample. All variables, including genetic variable, are significant. R-square became a little bit lower, but still relatively high.

Model 5 includes religion as another control variable. Results show that the existence of religious permission to consume alcohol has no significant impact on the probability of colonization. All other variables including frequency of allele Arg48His are significant and have signs as predicted.

### *Causal mechanism*

Our proposed causality mechanism is based on the ‘*unequal trade hypothesis*’. Alcohol was one of the major items in trade with indigenous populations. Moreover, it was one of the most profitable goods; the exchange of alcohol for local goods was unfavorable for indigenous populations. They adopted European drinking patterns with an emphasis on strong alcohol and daily consumption. European merchants benefited from the increased demand, earning sometimes from 400% to 900% profits. Such unequal exchange, given the increasing demand for spirits from non-Europeans, was likely to lead to economic dependence of native populations in Africa, America and Asia. Economic dependence led to political dependence and colonization.

To test the unequal trade hypothesis, I include the alcohol trade variable (Table 7). Correlation between the frequency Arg48His allele and alcohol trade is  $r = -0.370$  ( $p = 0.007$ ).

Table 7. Genes, alcohol trade and European colonization

	<i>Unstandardized Beta – coefficients</i>	
	<i>Model 6 DV – Colonization 1900</i>	<i>Model 7 DV – Colonization 1900</i>
Technology development index	<b>-4.348**</b> (2.156)	-
Frequency of allele Arg48His	<b>-4.043*</b> (2.272)	<b>-3.605*</b> (1.931)
Pathogen history	<b>-1.955**</b> (0.792)	<b>-1.996***</b> (0.709)
Alcohol trade	2.661 (1.788)	<b>3.829**</b> (1.612)
<i>Cox and Snell R-square</i>	0.502	0.418
<i>-2 Log likelihood</i>	32.057	37.409
<i>Observations</i>	52	52

\*- significance on 0,1 level, \*\* - significance on 0,05 level, \*\*\*- significance on 0,01 level

Model 6 includes the alcohol trade variable as a potential causal mechanism for genetic factors and European colonization. This model does not support our initial hypothesis: Alcohol trade and Technology development are not significant, and Frequency of allele Arg48His is significant only on 10% level. Perhaps, it can be explained by multicollinearity: correlation between Alcohol trade and Technology development is  $r = -0.616$  ( $p = 0.000$ ). These variables are likely to indicate technical development: only relatively developed populations have the capacity to produce liquor or sell in industrial volumes. Model 7 test excludes the technological development variable. All remaining variables are significant and have signs as predicted, although frequency of allele Arg48His is again significant only on a 10% level. Alcohol trade now is one of the factors that explains the probability of European colonization.

These models present some cautious evidence that alcohol trade may be regarded as one of the potential causal mechanisms between genetic factors and the probability of European colonization. Populations with lower Arg48His allele frequency were less involved in unequal alcohol trade and they probably consumed less strong alcohol. Thus, they had less of a chance to lose their political independence.

Models 3-7 show that the frequency of Arg48His allele is significant with sign as predicted. I suggest that it provides evidence of importance of genetic diversity as one of the potential factors of European colonization.

## *Conclusion*

In our case study, I tested the hypothesis about the impact of frequency of the Arg48His allele, which is responsible for the metabolism of alcohol and the probability of European colonization. I found that a higher frequency of that allele is associated with a lower probability of colonization by Europeans. The proposed mechanism is unequal trade: in many areas, distilled alcohol was one of the major trade items with native populations. Uneven trade resulted in economic dependence, and later, political dependence and loss of sovereignty.

Another implication from this study is the importance of sudden changes in traditional diets for social development. Many societies had diets which, in fact, were outcomes of adaptation to the environment. Europeans brought with them ‘a European diet’ that was associated with carbohydrate-rich foods, dairy products and alcohol. Such innovations as distilled alcohol and the habit to consume alcoholic beverages daily resulted in significant changes in social and political development. Readiness to overpay for alcohol led to the loss of lands and sovereignty.

This paper presents an empirical illustration of our main hypothesis about the potential relationship between genetic diversity and social change, a mutual biological social cycle that includes population density and genetic mutations. The suggested theoretical framework contains three elements in chronological order: ancient urbanization is associated with rising pathogen load, infectious diseases may lead to genetic changes, and the resulting variation in certain genotype frequencies may affect social development. It is one step forward compared to previous studies. The Arg48His allele frequency is one of the well-studied polymorphisms; it was thus one of the most convenient candidates for testing this theory. Among other candidate genes could be the ones associated either with food intolerances (as a genetic adaptation to environment), or with prosocial behavior like trust, individualism/collectivism or altruism.

I admit that this study has some limitations. Firstly, the sample is rather modest and some geographic areas are underrepresented. An alternative empirical strategy could be the approach suggested by Cook (2013): to convert population-level data into country-level data using ethnolinguistic approximation. Secondly, one may argue that colonization is not a binary variable, but a categorical variable with many values possible. Indeed, Europeans used to establish not only colonies, but also “zones of influence”. Thirdly, the list of tested predictors of colonization definitely may be extended with additional geographic, demographic, military and other factors. Nevertheless, this study reveals that genetic diversity – and I take it as an endogenous factor to social development, taking into account the relationship between ancient urbanization and the rise of pathogen load – affects social change.

Another contribution that this paper attempts to make is changing the focus in culture-gene studies from contemporary social and political developments to historic issues. Bridging history, political science, sociology and natural sciences (biology, genetics and geography) is a novel interdisciplinary approach in this field.

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## Appendix

### *Frequencies of the Arg48His allele from the ALFRED database*

<b>Population</b>	<b>Region</b>	<b>Arg48His</b>
Xhosa	Africa	0.005
<i>Yoruba</i> ( <i>Arg370Cys</i> )	Africa	0.290
<i>Chagga</i> ( <i>Arg370Cys</i> )	Africa	0.116
<i>Ibo</i> ( <i>Arg370Cys</i> )	Africa	0.360
Moroccans	Africa	0.080
Saharawi	Africa	0.100
Darghinian	Europe	0.133
Abkhaz	Europe	0.100
Adygei	Europe	0.048
Avar	Europe	0.190
Ingush	Europe	0.160
Kalmyks	Europe	0.263
Mari	Europe	0.112
sami	Europe	0.005
udmurt	Europe	0.117
Druze	Asia	0.133
Palestinian	Asia	0.064
Iranians	Asia	0.240
Turks	Asia	0.125
Indian (mixed)	Asia	0.099
Tajik	Asia	0.350
Uzbek	Asia	0.286
Yanadi	Asia	0.000
Yerukula	Asia	0.000
Bunun	Asia	0.840
Dong	Asia	0.521
Ewenki	Asia	0.090

Han	Asia	0.775
Hmong	Asia	0.696
Japanese	Asia	0.780
Koreans	Asia	0.777
Li	Asia	0.593
Tibetan	Asia	0.131
Uygur	Asia	0.362
Yao	Asia	0.647
Filipino	Asia	0.605
Thai	Asia	0.373
Malaysians	Asia	0.592
Altaiian	Asia	0.200
Buryat	Asia	0.267
Chukchi	Asia	0.020
Tuva	Asia	0.255
Yakut	Asia	0.094
Cheyenne	N.America	0.000
Huichol	N.America	0.000
Otomi	N.America	0.068
Pima	N.America	0.000
Sw amerindians	N.America	0.000
Maya	N.America	0.000
Maori	Oceania	0.450
Papuan New Guinea	Oceania	0.069

Source: The ALFRED database

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