COVID-19 and Population Genetics: Correlation, Causation and Likelihood
(Third and last delivery)

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To my daughter, Carmen Gómez Jiménez, R1 physician

May 31, 2020

At the beginning of April, after verifying that no one noticed a detail regarding the spread of SARS-CoV-2, or coronavirus, I decided to write a report that was published in a couple of articles. The organizers of *Mirabilia*, the scholarly journal where these academic essays are included proved to be diligent, flexible and efficient all to the same extent. For this answer to my request, I feel indebted to Profs. Antonio Cortijo, Vicent Martines, and Armando Alexandre dos Santos. Furthermore, for these and other reasons I would like to congratulate the entire Editorial Committee and also the members of the Advisory Council. These three articles respond to the same concern and claim that Population Genetics is a priceless tool in the fight against the coronavirus epidemic.

My achievements, if any, are due to the generous help and advice of some colleagues and friends, headed by Dr. Charles B. Faulhaber, director emeritus of the Bancroft Library. He was the organizer of a virtual debate at the University of California, attended by some referees on Molecular Biology, Genetics, Epidemiology or Virology; and also by some researchers from organisms such as the NIH (National Institute of Health), the CDC (Center for Disease Control) and several laboratories. Besides that, I have been in contact with experts of different countries and fields of research from the very beginning of this venture.

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If I leave these and other names aside, is with the only purpose of keeping them from any controversy that may arise, whether pointless quarrels or brainy debates since all are equally annoying and unbearable. In only one case, the post-crossing has been followed by an extensive and complimentary appointment of my work. This happened in a lecture delivered on May 25 by my colleague Juan Ramón Lacadena, Professor of Genetics at the University Complutense of Madrid and member of the Royal Academy of Pharmacy, whom I thank for his generous comments on my work.

I must highlight the interest, which at the end is support to my work, of three new acquaintances: Dr. Frank Bentrem, expert in Computational Statistics (currently affiliated to Insight Data Science, Virginia), who made his diagrams available to me, helpful materials for clarifying the relationship between COVID-19 and haplogroup R1b. Dr. Chris Golightly, a geological engineering consultant trained in Great Britain and residing in Belgium, has been informing me about all initiatives of potential interest, as well as his suspicions regarding hemochromatosis. Lastly, Dr. Attilio Cavezzi, a cardiovascular surgeon, contacted me on May 17 to inform me that, according to his data, COVID-19 might be a hematological disease. I would like to express my admiration for his unmatched enthusiasm. (The link to his new article on the subjet).

The generous comments of Prof. Dr. John G. Anderson, a prestigious orthopedic surgeon, are the authentic proof of a friendship fueled by the memory of a mutual friend, Steven G. Symmes, who united us to both applying the transitive property. It should be noted that before asking them for anything, Professors Elena del Río Parra, Vicent Martines Peres, Hernán Sánchez Martínez de Pinillos, Rebeca Sanmartín Bastida, Jaime Olmedo Ramos, Demetrio Castro Alfín and Antonio del Valle had already started to help me. With them have been Arsenio Escolar and David Arranz, journalists, philologists and friends.

I take this opportunity to greet Dr. Santiago Cousido Martínez-Conde, a retired neurologist from Cádiz. Dr. Cousido refers to haplogroup J2, a shield that Phoenicians left to their descendants in that part of Spain. And last but not least, I wish to express my gratitude to Antonio Marcos Tomaz Correia, a young Portuguese businessperson who, from the beginning of May, entrusted me with his research on R1b and other haplogroups, in Europe, Brazil and East Asia.

Once again, I stand by Population Genetics, whose applied dimension —unthinkable, more than merely unknown— I will try to bring to light by means of this short article. Although it is said that an abyss separates Letters from Sciences, I cannot see it or live

it that way. In essence, philologists and geneticists use the same heuristic procedure, which starts from the observation of a phenomenon, continues with the collection of all kinds of both data and materials for comparison and analysis, to culminate in a deduction or conclusion that, whenever possible, should be abridged in a formula. In the case of this research, it is as follows: *The higher the frequency of R1b, the greater incidence, proven or probable (real or potential), of COVID-19*.

In the previous articles, I focused in two maps so similar that may be reduced to just one. While the first shows the spread and rates of the coronavirus, the second expresses the levels of R1b haplogroup. As I say, these two facts are connected to the same milieu: the European territory. For more precision, their limits correspond to those of Western Europe. At the same time, we have to pay attention to other Y-DNA haplogroups present in the human communities that surround the Mediterranean basin. One of them, E1b1b, of North African origin, reached Europe much earlier than has been thought (later, there would be successive waves of carriers of this haplogroup). On the other hand, three more haplogroups whose origin is Eurasian (J2, G and I2) witness the early contacts of Europe and Asia Minor. To strengthen my proposal, I base my enquiries on graphs, maps, and conceptual diagrams, which show the dispersion and frequency of haplogroups at global, national, and regional scales.

Besides satisfying scientific curiosity or interweaving genetic and historical data, this article considers Population Genetics as an essential tool to make decisions and set priorities in case of sanitary alarm. Specifically, it can act as a barrier for new infections without interfering with the freedom of movement of all citizens. The failure to determine the etiology and to evaluate the spread of such disease ought to be forgiven by all community members, since not even the experts have so far solved some of the main dilemmas. On the contrary, slowness and lack of coordination are to blame for the loss of human lives and the spending of huge amounts of money, and all for nothing. Radical disagreements lead to major setbacks and, in extreme circumstances, inexorably end in disaster.

The difficulties for defining a common policy in Spain, when a joint action at the international level is required, should suffice to warn the parts in conflict (imaginary or real) and to convince them for leaving aside their ideological differences. It does not matter if they are leftists or rightists, or whether they identify themselves as Spanish or not. There is no possible excuse. The permanent disagreement threatens the feeling of cohesion of all structured societies. In the case I deal with, the feeling increases when

we compare the success of other nations, insistently labeled as "miracles", with the lack of ability of Spaniards to deal with the crisis.

This, however, not only happens in Spain: the European countries most criticized by local or foreign observers are those with the worst rates recorded in relation to COVID-19, which not by chance also have the highest percentages of the R1b haplogroup. Here I label them as "R1b countries", although there are also "R1b regions", as we see in the following relationship, which lists, from highest to lowest, those that are above 30%.

R1b countries and regions in Europe

Ireland 81%
Spain 69% (Top: Basque Country 85%)
Great Britain 67% (Top: Wales 74% & Scotland 72%)
Belgium 61%
France 58'5% (Top: Normandy 76%)
Portugal 56%
Switzerland 50%
The Netherlands 49%
Germany 44% (Bavaria 50%)
Iceland 42%
Italy 39% (Alpes 70%)
Denmark (33%)
Malta 32'5%
Norway 32%
Austria 32%

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Among all these countries, Belgium is going through its worst moment. What is striking is that nobody has ever pointed out that its rates, far from being out of place, are in accord with the percentage of haplogroup R1b (61%). Neither the hospital staff who turned their backs on Prime Minister Sophie Wilmès, nor the journalists, nor even the experts who analyzed the facts, data and figures of the COVID-19 crisis in Belgium are aware of these genetic findings. Belgian, Irish, Spanish, British, French, and North American people should take into consideration their special vulnerability to the coronavirus.

In my previous articles, I gathered a number of samples to prove my hypothesis, but to submit it to the standards of the scientific method requires a research group with at least a geneticist, a molecular biologist, an immunologist, and a chemist or someone else of some related areas, as well as the assistance of a statistician. The aim of this team ought to be to demonstrate that the correspondence between haplogroup R1b and COVID-19 is more than a simple correlation, as some skeptical readers of my previous deliveries have pointed out. The samples reviewed in this three-articles series, if not indisputable proofs, may at least provide valuables clues.

If that correlation were fortuitous and repeated not tens but hundreds of times (as many as these are possible), we would have crossed the limits of plausibility, which are also the limits of probability theory. That no common factor, whatever it may be, underlies all cases considered in my study is less likely than winning the United States lottery grand prize ten times in a row. Nevertheless, the work of the research team to which I refer will be essential in order to know how the coronavirus infection occurs. The main task of this will be to demonstrate that this is a basic principle to understand how coronavirus infects people. One last remark: the attention that geneticists devote to haplotypes should be extended to haplogroups (what they call "ancestry"), so that the knowledge of skilled specialists replaces laymen such as myself, who can only do so much by calling attention on the matter.

According to what corresponds to high percentages of haplogroup R1b, the disease is inflicting a harsh punishment in two American countries: the United States and Brazil. Nobody should blame President Donald Trump for being responsible of the current COVID-19 figures in the United States. These complaints are the result of despair, as the European experience taught us first. Actually, those who search for an explanation to this tragedy should take into account the genetic composition of modern USA society, in which the descendants of the British, Scottish and Irish settlers amount to 87 million people, most of whom carry haplogroup R1b. Other well-represented community, the German, adds a further amount of R1b, since it is present in 44% of males.

In addition, there are two communities that are growing rapidly and steadily, which reinforces —nothing odd, as I will explain— the hegemony of the R1b haplogroup: Hispanic and African-American. In the first, it is common for the mitochondrial haplogroup to be Amerindian and for the Y-DNA European. The frequency of certain branches or subclades of R1b in America, such as P-312 and DF-27, is a proof of Spanish ancestry. I remind that, in 1503, the Catholic Monarchs promulgated a law that

encouraged mixed marriages, which soon filled America with *mestizos*. To be added are many of the 12 million African-Americans who identify themselves as *mestizos*, carriers in many cases of R1b (*link*).

Brazil is a similar case. The majority of its immense population (212 million) is comprised by whites and *mestizos*, with several branches and subclades of R1b coinciding (M-269 is among the most frequent, as we see in this *link*). The scope is different in Oceania, perhaps because Australia and New Zealand share a series of peculiarities, such as their immigration policy, rigid as no other in the world, and an absolute control over their borders. That its customs, in ports or airports, prevent access to those who do not meet a series of requirements is something that is widely known.

In the narrative of the coronavirus, invectives meet their counterpoint in praises to the most successful health systems. Japan is one of the nations that has received the largest number of compliments since the beginning of the crisis. With respect to this nation, Mario Noya, a Spanish journalist who pays special attention to COVID-19, concluded that the key to managing the pandemic lies in the forward-looking nature and the effectiveness of Japanese people. Actually, the title faithfully describes the content: "Coronavirus: Japan vs. Spain. Do not compare what is worse". (*Libertad Digital*, May 18, 2020). It is another ear tug, one more, to the Spanish people; or, if you prefer, it is a new reason, one more, to whip ourselves. In my opinion this punishment is unfair since the differences of both nations yield a very inaccurate comparison.

First, let us remember that Japan has 126.5 million inhabitants and Spain 47.5 million. Due to the systematic rejection of immigrants by Japan and the massive immigration to Spain during that same period, the Spanish population today amounts to just over a third of the Japanese (the current ratio is 2,663) when at the end of the 20th century was well under a third. Only now can we get an idea of what the 16,395 infected from Japan and the 232,037 from Spain entail. If we stick to the size of their respective populations, the 16,395 cases in Japan would have to be compared to 617,914 in Spain, all without resorting to the unofficial number of infected in Spain, totaling around two and a half million. Without forgetting the indicated ratio, the death toll in Japan (773) and Spain (27,778) can only be understood when considering that R1b haplogroup is missing in the Far East.

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Antonio CORTIJO, Vicent MARTINES, Armando Alexandre dos SANTOS (orgs.). *Mirabilia 30 (2020/1)*
War and Disease in Antiquity and the Middle Ages
Guerra y enfermedad en la Antiguedad y la Edad Media
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ON May 15, 2020, the feast of San Isidro in Madrid was frustrated by COVID-19. On that eve, sad omens hovered over Spain, when the result of the serological analysis of the Spanish population was made public by the Ministry of Health. A report on the immune system and the development of antibodies against the coronavirus was also included. Attention was paid not only to regular patients who showed the symptoms of this ailment, but also to those asymptomatic. Most important is that, at least hypothetically, they would have developed defenses against a new contagion. According to the report, only 5% of the population (just over two million and three hundred thousand people) would have been immunized against COVID-19. In just a few months we will see whether they are really protected and to what extent.

The Minister of Health and the official spokesperson added, with undisguised frustration, that we are far from 60%, a percentage where some experts would mark the "return to normality". It is striking, however, that, according to other opinions, this percentage must be reduced to 40% and even to an immediate 10% (a figure reached yet in various places in Spain, including Madrid). Specifically, these percentages have been released by José María Olmo: "Two studies conclude that group immunity is reached before 60% of infections" (*El Confidencial*, May 7). I guess that at least some of the samples alluded by him in this article belong to a specific phenomenon, innate immunity, which I would look for not only in haplotypes, but also in haplogroups, since it seems that some haplogroups are resistant to the coronavirus and others seem to immunize those who carry them. Advancing from hypothesis to scientific truth would only be possible with further DNA tests.

If so, even in the worst scenario, the distance to the target would have been shortened, and the news shifted from a feeling of sadness to reasonable optimism. Such a positive perception was the one that resulted from the decrease in the number of infections and deaths and the ascent in hospital discharges. However, in the evening of the same May 15, a new warning from the WHO ruined the whole day: "The coronavirus is here to stay." For any *madrileño*, either witness or (much worse) protagonist in the tragedy caused by the coronavirus there is no comparable prediction. Nevertheless, we should take into account that the sound of this warning differs in Spain (or in other R1b countries) or Africa, where nobody cares about COVID-19. How dare they compare an apparently mild disease with malaria, which in Africa reaps 600,000 lives a year?

Moreover, due to genetic peculiarities, COVID-19 hardly affects African people, as verified after a search of those data into the Johns Hopkins University Website, which gives the figures of contagions and deaths worldwide. Those who have not yet done so

will be surprised by the extremely low numbers of both infections and deaths from Maghreb to Middle East, all along the Southern Mediterranean shore, and thereafter proceeding down towards the heart of Black Africa. There is not a single African nation that comes close to Europe or the United States; in fact, even South Africa, where figures closer to those of western Europe would be expected, informs us of only 524 deaths (in a country with more than 59 million inhabitants). Unless we pay attention to haplogroups in our research, this fact lacks any logic and stands as a nonsense. Contrary to the dictates of logic, while Europe falls defeated by COVID-19, Africa succeeds. It is the same foe, but the struggle ends in a most unexpected manner, as if we were in the Upside-Down world.

We should not forget that we are dealing with countries with the lowest rates in per capita income, life expectancy and other indicators, but if we turn to coronavirus the numbers are just astonishing. Two references and some few numbers prove the truth of my assert: at one hand, we see Mozambique, with only 244 cases and 2 deaths; or Ethiopia (110 million inhabitants), with only 845 cases and 8 deaths. At the other, the casualties in the United Kingdom, 38,161 deaths after 271,222 contagions, speak by themselves. What strikes me is that some supporters of such a viewpoint are completely skeptical when they have to deal with my proposal on haplogroups; and, in order to ward off danger, they have a phrase (a sort of amulet) at hand: "Correlation does not imply causation".

COVID-19 showed its destructive power as soon as it found a large reservoir of haplogroup R1b: that corresponding to Italians who live between Tuscany and the Alps. With memory still alive, since we know that the coronavirus, somehow and somewhere, is still there, though its activity has decreased, I wonder what could be done to stop the horror. What was the use of the messages from the WHO, which called for swift action without warning of the extreme virulence of the new virus? In fact, someone as insightful as Salvador Sostres, prestigious *ABC* journalist, had to turn 180 degrees in his chronicles dealing with the disease: if he did not depart from the "official version" of the WHO (and the statement "COVID-19 runs like a mild flu") Madrid's particular drama would forever remain incomprehensible.

Although the virus is only one, its behavior depends a lot on the genetic characteristics of the inhabitants of every affected country or region. In my view, the demonstrations on March 8 would have had a very different effect —insignificant or null— had they not taken place in Spain but in any town of Finland. In addition, I am not saying this because of its lower population density or the contained effusiveness of its natives,

which means a greater distance between two or more people. These are not the reasons why the coronavirus does not take root in Finland, which on May 30 totaled only 320 deaths caused by the pandemic. The main thing is its low percentage in R1b: a negligible 3.5%.

The Irish were in for a surprise when the COVID-19 data began the galloping climb some had anticipated. For a few journalists, the magnitude of the disease in Ireland made no sense, "when compared to similar nations." However, for those who carried out the comparison, the resemblance of Ireland to other nations (Norway, Denmark, Finland, Croatia and Slovakia) was limited to the number of inhabitants and in no case to its percentage of R1b: an impressive 81% which places it at the head of all R1b countries. And, as Ireland approaches five million inhabitants and Finland exceeds them by half a million, and since the difference in the number of deaths is so great (when I write this, the figure for Ireland, 1,651, is five times that of Finland) The surprise and irritation of the Irish was enormous. I hope that at some point they become aware of all this, so they know that they are not as unable as they think.

Boris Johnson, the famous British statesman, was much more reckless in this regard. Moreover, as we know, he paid dearly for his mistake, since the coronavirus took him to the ICU and put his life at risk. Fortunately, it was not long before he got rid of his first idea, which was to promote contact with the sick so that a large part of the population would be immunized against the coronavirus as soon as possible. In my opinion, the British political leader was deceived by those people repeating the phrase coined by the WHO: that COVID-19 did not pose a greater risk to health. To be added is a risk factor they are still unaware of: Great Britain occupies one of the top places as a R1b country, with a percentage of 67%, which places it just behind Spain, with 69%.

Between May 17 and 19, the mass media released the news that the coronavirus had granted us no more than a mere truce. Some, moreover, have begun to classify it as deceptive, because nothing has changed or will change until we succeed protecting the most sensitive part of the population, which becomes hopelessly ill when being exposed to such a silent, insidious and ruthless enemy. We need to know who the members of this extreme fragile group are before it is too late, that is, before their names appear in the list of deceased on the previous day by action of COVID-19. This is a race against the clock, but also a test of endurance. How many casualties are we willing to accept? Moreover, how many months of war can we stand? I am sure that neither the industrious Germany nor the powerful United States would succeed confronting an enemy of everlasting stamina.

Everlasting or long lasting? The first adjective seems to be most adequate to the current perception of the menace. Actually, for many the Western civilization is in extreme danger. It is for this reason that we have no choice: we must defeat the coronavirus finding out a remedy, a goal that ought to be attended as soon as possible. The healing virtue of that medicine should allow us to either cope with the disease (as it happens with AIDS), or to eradicate it (as with smallpox). Today there is only one means to stop it: cutting the chain of contagions. With this purpose in mind, it is necessary to observe the behavior of this virus, individual to individual (that is, haplotype to haplotype), but also haplogroup to haplogroup, and —once again, I have to point out this loss of information— I cannot understand why so many geneticists do not consider haplogroups of interest in their research.

I would like to prove that the acquaintance with haplogroups becomes a valuable source of information to deal with COVID-19. Not only R1b is a risk factor, but the main risk to become infected by COVID-19. I suspect that genetics determine the evolution of the disease and counts as much or even more than the individual's age, health, morbid obesity, chronic ailments (among them, diabetes, heart failure, chronic obstructive pulmonary disease, etc.), or treatment with immunosuppressants to prevent allograft rejection after organ transplant and to prevent or treat disease flares in autoimmune diseases. On their part, haplogroups R1a and I2 seem to hinder the action of the virus and mitigate its lethality; on the other hand, E1b1b, J2 or G could be perceived as a protective shield against COVID-19 (in the case of haplogroup G, this idea has been advanced by Gigi Tevzadze in a note dated April 3, "Haplogeography of COVID-19: A hypothesis", which also alludes to a feeble R1b). These hypotheses are very impressionistic and will not be of any use as long as accurate analysis are not carried out and studied by statisticians.

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Close to the end of this trilogy, I noticed a number of unanswered questions, and doubts that should be solved. For instance, considering quarantining all workers of haplogroup R1b, should the disease resurge, is categorically impossible, regardless of whether we deem the economy irrelevant. On the other extreme, trusting everything to luck would be like committing suicide, since the virus, lacking a partial or definitive remedy to controls or kill it, will wreak havoc. Fear is spreading wide and deep: I wish this were the first step towards a reaction for a new assault this fall. What will happen? Well, it is not hard to imagine: it may be the debacle, the closest thing to the biblical Apocalypse.

Should it befall upon us and having had no time to recover, discouragement will spread and take over even the most combative spirits. It will be a run for the hills. In the midst of chaos, urban centers will be disbanded to take refuge in second or third residences, anywhere and at any price. Unfortunately, we have already seen what awaits those who want to escape from the city to take refuge where infections and casualties have been either few or just none. The stable population will prevent them from entering, and the same will happen traveling down the road to the next population, and the next, and the next ... This exercise does not require much imagination: the transformations induced by COVID-19 are profound.

It is not a time to harbor fatalistic feelings, but to anticipate what may come. As I said, we are enjoying an extension that is likely to end with a sudden spike in numbers after a lockdown of more than two months. In spite of it, there are reasons for optimism, since we know that, along with individuals and groups sensitive to coronavirus, there are resistant ones. In reality, a good part of the world population is partially or totally safe from its action. The main consideration is that these are not isolated individuals, but rather communities settled in specific areas. Without this factor, it is impossible to understand that the lowest contagion rate in the world occurs in Syria, where the destruction of a war of all against all reigns; and in Zambia, which marks two extremes: the one with the highest number of those infected with AIDS and one of the lowest in those infected by the coronavirus.

That coronavirus has a special fondness for Western Europe is an obvious fact: for the people, and in no case for the environment, the quality of the waters, or other factors. The number of infections and deaths, which skyrocket as soon as we pass from the Slavic World to Occident, show this: from Poland to Germany (the defenders of the so-called "German miracle" do not have a point), or from a Slavic city as Zagreb to a Western city as Milan. Let us recall that these two cities are separated by 520 km, a distance less than that which takes us from Madrid to Seville; however, the COVID-19 figures range from 550 infections per million in Slavic Croatia (whose flag displays the colors red, white and blue of Pan-Slavism) to 8,562 in Lombardy; and from the 99 dead in Croatia to the 15,786 dead in Lombardy, the latter having 10 million inhabitants and the former 4 million.

As I have explained before, the figures change following the patterns of Population Genetics, which in the chosen examples lead from the Slavic haplogroup R1a, for which the coronavirus seems less interested, than the quintessential western haplogroup: the R1b. In the reaction of coronavirus, when it contacts the different haplogroups, may lie

the key for the aim pointed out by Dr. Pablo Daniel Lapunzina, geneticist of the Hospital La Paz (Madrid), in an interview for *iSanidad* (May 14): "a "selective confinement". There, too, he refers to group zero, made up of those who have been very close to the virus and have not been infected. And, without going into detail, he adds: "we do not have samples of them, but it is possible that there are people in whom, due to variables in the receptors, the virus has not been able to enter". As is the norm among geneticists, any allusion to haplogroups is missing.

In addition, the expectations of this lucid geneticist will be met by someone with differences in the alleles or nucleotides. He has the best possible partner in this enterprise: Prof. Ángel Carracedo and his team from the University of Santiago. In the search for a remedy against coronavirus, the name-list of experts includes personalities such Luis Enjuanes, Anna Planas or Mariano Esteban, who made some generous remarks on my research on haplogroups. (Thanks for your kind words, master!) If I may insist, I would like to remind that, from time to time, it would be convenient to go from haplotypes to haplogroups such as E1b1b or J2, which seem to provide a unique resistance to coronavirus infection.

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Checking how the coronavirus interrelates with the haplogroups may help to control COVID-19 outbreaks and subsequent de-escalations. For this, it is essential to have a database that collects genetic information of the entire population or of a stablished segment of special interest. Those who are most reluctant to invest resources on this concept must bear in mind that they are leading the present and, at the same time, anticipating a future in which genetics will mark — it already does mark— the paths followed by most specialties.

Sebastiano Schillaci shares my opinion ("Possible Correlation between COVID-19 Susceptibility and Haplogroup R1b", *OSF Preprints*, May 26, DOI). In my articles, he says, he has found a much wider frame for his own research ("For a more detailed but complementary account see also [9] and [10]", where the numbers correspond to my two works), since he limits his enquiries to Italy. We both agree that massive genetic analysis would provide the most valuable information we may ever imagine:

> Most likely, one or more genes that heighten the susceptibility to the virus are also correlated with the haplogroup R1b. In order to validate this hypothesis, a study of the genetic profile of the population in the most affected areas would be needed, such as the study in Vo' (Veneto) [4]. Anyway, this hypothesis, if correct, could speed up the discovery

of a treatment, help make more reliable quantitative forecasting models or, at least, help better tune the social distancing measures.

At no time, however, should we ignore the risks derived from the use of Genetics without the appropriate ethical code. In such a case, there is a real danger, since it may modify the research at different points: defining the project and identifying the goals, collecting data of interest, processing the information or arriving to conclusions and listing the results. Held by certain private companies or a State that tries to control the lives of its citizens, it may be the cause of aberrations such as discrimination due to genetic causes. Similar to this is the fact that more and more individuals, urged by a curious spirit, order genetic analysis to know their predisposition to develop certain ailments and anticipate the appearance of some diseases.

As for the interference of the State in the lives of citizens, it should be noted that this is one of the paramount issues of our day. Whether in parliament, on the street or even at home, there is talk of the growing control of political-social structures over the individual, whose existence is regulated, from the cradle to the grave, in an Orwellian manner. The fear of a world controlled by a few people, thanks to the advances in technology, has been accompanied by a reaction that takes all forms that can be imagined and is linked to the appearance of the coronavirus, whose origin and expansion involve experts of all the disciplines, from the most diverse viewpoints and ideological approaches.

If we follow this path, we will meet again —eventually, but inexorably— with the coronavirus. I invite you to discuss the most controversial points of my proposal. Consider this paper a tribute to all those brave people who gave their best of them — in some cases, accepting a sort of martyrdom— to stop the advance of coronavirus. I myself am motivated by one single purpose in my essay: I wish to help the community of which I am a part, especially if, as can be expected, a feeling of discouragement due to a flare-up begins to spread. If this circumstance were to occur, economic activity (especially that corresponding to strategic sectors) should not stop under any circumstances, since we know that a large part of the population has innate or acquired immunity against the coronavirus.

In the group to which I refer, there are not only Spaniards, but also residents in Spain and contracted foreigners who temporarily come to work in the Spanish agriculture or join our fishing fleet. That all of them, without discrimination, have been forced to cease their activity is as illogical as the fact of applying the same prophylactic measures

to all people, when not all need them. In order to know how to proceed in each case, we need to gather information about haplotypes (i. e., data on individual genetics, since haplotypes are the genes that we inherited from our ancestors), without forgetting haplogroups (groups of haplotypes with the same characteristics, in which the coexistence for centuries, if not millennia, of a human community is reflected).

With these data, updated information provided by the National Institute of Statistics, and paying special attention to the strategic sectors (food comes first), we may overcome a new threat by the coronavirus in the two fronts where it does the most damage: health, and economy. For a more precise picture, I invite all of you to attend the forthcoming virtual debate on this issue arranged by Prof. Vicent Martines, coordinator of the activity, at the University of Alicante.

In advance, I inform you that I will concentrate on some strategies for bypassing the coronavirus in case of strong comeback. Prof. José Miguel Sempere, renowned expert on Biotechnology and head of the Dept. of Immunology at the Alacant-Hospital General, has promised to bring a *status quaestionis* for this occasion. Since Prof. Sempere is always in the frontline, in his intervention we will see the state of the art in the treatment of the disease. This promising event, to be held on June 22, will put an end to an odd academic course. If you have the possibility, please do not hesitate in joining us.