

# GENETICS AND ECONOMICS: STUDIES AND PERSPECTIVES

by

Alfredo M. Navarro\*

*This paper analyzes the relationship between genetics and economics and the effects of both genetics and the environment on the phenotype of human beings. Among other issues, it describes how data from the human genome can be used to explain certain economic characteristics, such as the existence of entrepreneurship, attitude towards risk, income level, propensity to invest, and ease of receiving education. A series of works related to this issue are analyzed, and the various ways of drawing conclusions from existing data are described. The paper concludes by stating that this type of study is in its infancy, but that it helps to better understand, along with the evaluation of the environment, certain characteristics of human behavior and opens an interesting field of study for economics. (JEL A10, A11, A12, B41, C13, D31, D33, I14, I24, Z13)*

## I. INTRODUCTION.

Although we know that children share certain physical or behavioral characteristics with their parents, there has always been a question as to whether this similarity is due to the fact that they share the same environment, the same education, and the same social group or to the transfer of a certain genetic code, or if both factors influence a proportion that we do not know but that we can try to determine.

This question dates back to the beginnings of modern biology. Charles Darwin argued that evolution is possible because living beings transmit their characteristics to their descendants, incorporating increasingly complex mechanisms, which make different species to change towards more advanced forms.

In 1975 the work of Edward O. Wilson *Sociobiology* appeared in which he analyzes the social relations of all living beings. In the last chapter he studies the differences among humans, and unleashes a controversy, because while Wilson argues that genetic input.

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\* Full Member of the National Academy of Economic Sciences, Argentina.

plays a very important role in them, most social science scholars of that time attributed them to the environment and believed that they were due not to hereditary reasons, but to the fact that some individuals belonged to social groups that had the necessary economic means, better food, health and housing and, above all, education.

These scholars believed that culture had replaced genes as the cause of the evolutionary process, since these could not change so quickly to explain the dizzying transformation of the world operated in the last two centuries, while changes in cultural elements had operated drastically. This discussion, which was very heated, raises a very important question: what defines our phenotype, the factors that we inherit through the genes transmitted to us by our ancestors, or those we receive from the environment in which we operate?

It is worth pausing for a moment on this question, for which we must keep in mind the work of Steven Pinker (2002) *The Blank Slate*, in which he explains the nature of the at times violent discussion that caused the appearance of Wilson's work, and whose title refers to the concept of *clean slate*, as a characteristic of all human beings, whose differences do not come from nature, but from the society in which they have developed and from the education they have received. He points out that John Locke (1632-1704), who laid the foundations of empiricism, maintains that everything comes from experience and that therefore there is no innate difference between men, since all souls are equal in principle, so there is no support for class differences, monarchy, discrimination. of women or slavery. This idea, which had already been anticipated by René Descartes (1596-1650), is picked up by Jean-Jacques Rousseau (1712-1778), and also coincides

with Marxist thought. This explains the reaction against Wilson and the emergence of the debate that was called *nature vs. nurture*, which established the competition between the two to explain our characteristics: we have *inherited them from our ancestors, or we have acquired them from the society in which we live*. The greater the weight of the latter, the greater the result we can obtain by modifying the social system in which we are immersed. The appearance of genetics had a decisive impact on this question. Not *everything is nurture* anymore; we now have evidence that a part of our characteristics come from our ancestors, and we are no longer all the same in the event that we have a different genetic endowment. If we think that equality between human beings is something we want to move towards,<sup>1</sup> having a clearer vision of what human nature and society is will surely help us build a more egalitarian world to assist those who have inherited deficiencies. It is also necessary to make it clear that the effects of the environment cannot be ignored, and that, depending on the problem analyzed, this fact has more or less importance in the explanation of the different phenotypes.

In this paper we have proposed to review some of the works that we have considered most relevant and, based on this, to make an evaluation of the future prospects of this line of research.

## II. INITIAL EXPERIENCES.

During the last quarter of the last century, many studies were carried out which tried to explain the presence of a certain characteristic on the basis of hereditary and

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<sup>1</sup> Jan Tinbergen (1968) proposes, with general acceptance, five objectives for economic policy. One of them is to reduce inequality in society. The other four are: maintaining full employment, stable prices and balanced external accounts; eliminating poverty; maintaining peace and preserving all the individual freedom as it is compatible with the other ends.

environmental factors by examining the differences between own or adopted children and between monozygotic and dizygotic twins. The former share all of their DNA, but the latter share only one half.<sup>2</sup> Therefore, if we analyze certain characteristics of the offspring that correlate with those of their parents in greater proportion in the monozygotic than in the dizygotic twins, we can assume that this is due to hereditary factors, since the environment in which the twins develop is very similar because they are raised in the same family in most cases. Let's look at some examples of these preliminary studies.

1. Following this methodology, one of the first attempts to explain the differences exhibited by individuals in the labor market, considering the cultural conditions of the environment and the transmission of conditions through genes, was carried out by Behrman and Taubman (1976), who conducted a study based on a sample of approximately 2,000 twins, some of whom were identical, and some non-identical. They were able to observe that identical twins had more similar results in their educational performance and income level than those who were not identical. They came to the provisional conclusion that both cultural context and genetic transmission influence the years of education, socioeconomic status and income level of the individuals in the group studied.

2. Ebstein et al. (2010) report the results of a study that also analyzes the different correlations between the phenotypes of the two types of twins and their parents, in which they conclude that issues such as prosocial behavior, stress, infidelity, empathy, political attitudes, leadership, aggression, parental care and risk behavior are influenced by hereditary factors.

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<sup>2</sup> When the egg and sperm select half of the genome to join with their counterpart, they do so randomly, which is why this is the average value of a probability distribution.

3. Another example of these preliminary studies was the debate that occurred in the United States regarding the effect of cigarette smoking on health<sup>3</sup>. Several studies demonstrated the association between smoking and lung cancer, circulatory problems and other diseases. American tobacco companies turned to a highly regarded geneticist, Clarence Little, president of the University of Michigan, who claimed that lung cancer was acquired by genetic inheritance, and not necessarily through smoking. But while it has been shown that a particular gene is associated with lung cancer, it has also been statistically proven that smoking contributes to making it more likely that those with that genetic characteristic will get the disease.

This error is a consequence of taking into account only genetic factors and leaving aside those related to the environment in which each person lives, beyond suspicions of the existence of economic interests.

### III. WORK USING THE HUMAN GENOME

The general framework of analysis of the relationship between genotype and phenotype is exposed in the work of Benjamin et al. (2012), which is complemented by Cesarini and Visscher (2017), where they present the following equation:

$$Y_i = \sum_{j=1}^J X_{ij}\beta_j + U \quad [1]$$

where the explained variable,  $Y$ , is a certain characteristic of the phenotype (e.g., diabetes, learning difficulty, or obesity);  $j$  are the different locations of the SNPs;  $X$ , is the value of the allele, which can take values of 0, 1 or 2;  $\beta_j$  is the impact that this allele has on the explained variable,  $i$ , the different observations of the sample and  $U$ , a variable, or a set

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<sup>3</sup> See Akerlof and Shiller (2015).

of them, that express environmental conditions. Initially, the first term on the right side of the equation [1] was replaced by the relationship between relatives, which is also a manifestation of inheritance, as we have seen in the works analyzed above.

Genetics has been studied for a long time, but the work of Crick and Watson (1953), in which the famous spiral is described, produced an explosion in the amount of related research.<sup>4</sup>

We now know that the human genome, whose complete sequence has been available since the year 2000, has 23 pairs of chromosomes, inherited one from the father and the other one from the mother, and they form two strands that have the shape of a double helix, composed of basic elements called *nucleotides*, which are the *steps* that form the classic spiral. They contain one of four bases: adenine (A), cytosine (C), thymine (T) or guanine (G), so there are four different classes of nucleotides that are always grouped in pairs, with adenine being associated with thymine (AT) and cytosine with guanine (CG).<sup>5</sup> The human genome has 3,000 million of these base pairs, which make up about 22,000 genes, which are grouped into the 23 chromosomes<sup>6</sup>. Humans share our genetic basis in about 99%, but we differ in what is called *genetic polymorphism*, (of which the simplest are the so-called SNPs) which are the parts<sup>7</sup> of DNA in which there are differences. between

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<sup>4</sup> Kauffman (2018) states that the first indications about genetic studies can be traced back to Hippocrates and Aristotle, and that was Mendel who in 1868 discovers that genetic variations are discrete, and lays the foundations of modern genetics, being his work recognized recently, at the beginning of the last century.

<sup>5</sup> The readers interested on in deepening this question should consult the work of Carey (2003) if they want to see an accessible text, oriented to scholars of the social sciences, or to Albert et al. (2002) if they prefers a more in-depth work. The complete human genome can be seen on the John Hopkins website University (<https://www.ncbi.nlm.nih.gov/O.mim>), while it is medically oriented, it gives us an idea of the operation of this type of database.

<sup>6</sup> The number of genes that make up each of the chromosomes is variable. While chromosome 1 has 3000 genes, the 21 has only some 300. Each of the genes has a name, usually related to the protein it orders to produce, which is assigned by a committee called *Gen Nomenclature Committee* (HGNC). So do each of the SNPs that have been studied.

<sup>7</sup> SNP stands for the expression *Single Nucleotide Polymorphism*, which is a variation in a single nucleotide. Some people have an A where others have a G, which is a special type of allele, is somewhat broader, since it refers to a different version of a gene. Sites in the genome where individuals differ in a

one individual and another. A gene can have hundreds of SNPs, and these can also be in areas that are not part of the genes. It is assumed that there are hundreds of millions of SNPs, and <sup>8</sup>in each of them three possibilities: when we inherit one allele from our father and another from our mother, we can have no minor allele, one minor allele, or two minor alleles. This circumstance, for statistical purposes, is identified with the numbers 0, 1 and 2, values that are used in the regressions.<sup>9</sup>

In the beginning, the analysis of the human genome was used in medicine, because the mutation of certain genes was related to diseases and the relationship of a single gene with a phenotype was studied. An example is the case of Huntington's disease, a neurological disease that produces, among other symptoms, involuntary movements in the extremities. It was possible to determine which is the gene in which the mutation that produces it is found and then allow the diagnosis, predict the age at which the disease will appear and its severity, as well as allowing better treatment design.

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simple base, those called SNP. There are 140 million referenced to date, a figure that is growing progressively. The 1% of our genetic code that we don't share with other individuals in our species is what makes us not be identical.

<sup>8</sup> It can be found in the database *dbSNP*, which is freely accessible.

<sup>9</sup> The estimation of the parameters of models with a large number of regressors that are often correlated and present *overfitting* which makes traditional econometric methods not appropriate, so some alternatives have appeared to overcome these problems. The most commonly used is the method called LASSO (*least absolute shrinkage and selection operator*) which is known as *regression Penalized* which consists of introducing a constraint in the Ordinary Least Squares model, so the equation to be estimated is as follows:

$$J(\phi) = 1/2m \left[ \sum_{i=1}^m (h_{\phi} x^{(i)} - y^{(i)})^2 + \lambda \sum_{d=1}^m \theta_d^2 \right]$$

where J is the function to minimize; m is the number of observations;  $h_{\phi} x^{(i)}$  is the value that arises from the estimated model for each of the i observations;  $y^{(i)}$  sound the observed values of the dependent variable;  $\lambda$  is the value of the applied constraint; and  $\theta_d$  are the values of the J parameters to which this constraint applies. In this way, biased parameters are obtained, the variance of the model is reduced, and overestimation is avoided (*overfitting*), which optimizes the tradeoff between the two. An amount to be determined from the parameters they take zero value and arrive at a model that forecasts better outside the sample used for the estimation. To do this, the sample is divided into three parts. In the first, which is usually 60% of the available data, the estimate is made using the Artificial Intelligence procedure known as *machine learning*; in the second, composed of 20% of the available data, The values of the estimated parameters are validated, and the remaining part is used to test the model. (See Gunes, 2018).

These types of studies began to develop rapidly since 2005, when three researchers<sup>10</sup> from the *Broad Institute*, Harvard University and MIT, developed a fairly complex statistical analysis method, called GWAS (*Genetic Wide Association Studies*), which is a procedure that seeks to find associations between genetic variants and phenotypes in a population, in order to identify genes or SNPs that may contribute to the appearance of a disease or a certain characteristic. While it is used to study the relationship between genes and different diseases, it can also be used to analyze characteristics of people that have to do with economics, such as wages, income, risk behavior, entrepreneurship, learning and some other issues.<sup>11</sup> A population that has a certain characteristic is compared to one that does not, and then by performing regressions (sometimes up to hundreds of thousands of them), where the explained variable is a characteristic (e.g. income level) and explanatory statements of SNP's or combinations of them. The results are expressed by means of a graphical representation called "Manhattan graph", where on the abscissa axis the 23 chromosomes appear along with the genes and SNPs that compose them, and on the ordered axis the value of the probability assigned to each one, which allows to identify which are the SNPs that are related to a certain characteristic of the phenotype.<sup>12</sup> The GWAS works in interaction with various databases, of which *The Human Gene Mutation*

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<sup>10</sup> See Altshuler et al. (2005). A description of how GWAS operates can be found at Hardy and Singleton (2009), in <https://www.nejm.org/doi/full/10.1056/NEJMra0808700>.

<sup>11</sup> The genetic basis of about three thousand diseases, but only about a dozen of the characteristics of the phenotype that have to do with economic issues.

<sup>12</sup> It has been used to analyze the genetic components of thousands of diseases, but its use in social sciences it much less. It is not exempt from criticism, of which the most common is related to type I errors (obtaining false positives) and type II errors (by applying a too strict probability criterion, such as the so-called *Criterion of Bonferroni*, herself discard valid relationships). On the other part it does not consider environmental factors, since it analyzes only the genetic causes of the characteristics of the phenotype analyzed.



*Database (HGMD)* stands out<sup>13</sup>, whose function is to compare the relationship between alterations in genes and certain types of alterations of the phenotype related to diseases.<sup>14</sup>

Soon after it was realized that this new knowledge could also be useful to the social sciences, so they began to study certain characteristics of individuals related to the subjects they study.<sup>15</sup>

Thus, was born the discipline known as *genoeconomics* (expression that is due to Benjamin et al. (2010)), which studies the use of genetic information to analyze economic problems. It represents an important advance over the methodology that was previously used, when the knowledge of the human genome that we have today was not yet available. Economics, according to these authors, can explain how market forces respond to genetic factors, while genetics can help economists to identify and measure important causal relationships, and alternative suggestions to economic policy could emerge.

During the last ten years a number of works have appeared in which the relationship between the genetic code of individuals and their economic behavior is studied, through two alternative forms of work: the first is to establish *a priori* which molecular markers can produce certain characteristics and then, if people with that marker really have them. For example, as we know that oxytocin contributes to a behavior tending to solidarity, we can analyze the relationship between this behavior and the presence of a certain molecular marker. The other way is to take a set of people of whom we know certain characteristics and their genetic code, and look for correlations between them, which requires the management of an enormous amount of data, since we work with samples from several

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<sup>13</sup> We thank *The QIAGEN Digital Insights Team* by that have provided me with access to their database.

<sup>14</sup> The procedure known as *meta-analysis*, which consists of creating a *pool* with other works that analyze the same question, in order to increase the sample size.

<sup>15</sup> See Benjamin et al. (2012).

thousand individuals of which several hundred thousand molecular markers are analyzed <sup>16</sup>. Let's analyze some of the works that we have considered most representative.

1. An example of the first way to study the relationship between genes and phenotype is the experiment described by Benjamin et al. (2006), carried out on data from a work dedicated to analyzing cardiological diseases in Iceland and which began in 1967. Were analyzed 30,795 men and women born between 1900 and 1935, living in Reykjavík. In 2002 was studied the genome of 2,300 survivors, who were questioned about some economic issues, such as their years of education and income, their attitude to risk and their intertemporal preference. Then they made a list of SNPs that were suspected candidates to explain these characteristics, where they analyze markers related to the production of dopamine, serotonin, cognitive ability, intelligence level (IQ), memory, and other issues and seek their correlation with the economic phenotypes. There are certain characteristics in economic behavior that are relatively easy to associate with genes, such as impulsivity, risk aversion, and solidarity, and others that while they may be influenced by genes, that are more distant, such as education level and wealth or income, for which sets of genetic characteristics must be analyzed.

2. Ding et al. (2006) use a database generated to study the effect of cigarette smoking on adolescent behavior, composed of 893 students who were surveyed to determine whether they suffered from obesity, hyperactivity, inattention, drug and tobacco use. In addition, saliva was extracted to analyze their genetic code, with the idea that certain genes had to do with addictions, and these in turn with the state of health that influenced their academic behavior, which in turn did so on their occupation, their income and their family life.

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<sup>16</sup> That is to say that the study is carried out without any previous theoretical basis, so this procedure resembles what the econometricians call "*data mining*", which consists of looking for correlations and then explaining them, like someone digging wells for oil without any plan, expecting to find it accidentally. This is what GWAS does.

Instead of directly analyzing the relationship between the selected genetic markers and the academic result obtained, they build a model where five conveniently chosen genes constitute the explanatory variables. The brain's reward system is linked to addictions, and certain neurotransmitters make us susceptible to contracting them. They describe how an activation of the area of the brain called the *ventral area* releases dopamine, and neurotransmitters carry the signal to the *nucleus accumbens* (limbic part of the brain) to be transmitted from there to the cerebral cortex, which is where decisions are made. These neurotransmitters explain smoking addiction, depression, hyperactivity and other issues, and their abundance is determined by certain genetic markers,<sup>17</sup> which are included in the model used to explain the academic behavior of the adolescents studied. This model is composed of three equations,<sup>18</sup> in two of which the genetic endowment appears as an explanatory variable and is significant in the estimates made. In this way, the genetic factor is incorporated not directly but as one more variable of those that make up the model,<sup>19</sup> which makes the work an interesting methodological contribution. If both the genetic code and the environmental conditions are significant variables in the explanation of certain economic circumstances, we can consider that the debate referred to above has

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<sup>17</sup> The genes are DRD2 and SLC6A3, related to dopamine and CYP2B6 related to another neurotransmitter called tryptophan hydroxylase.

<sup>18</sup> The three equations are as follows:

$$\begin{aligned}
 (1) \quad A_{ijT} &= \beta_0 + \beta_1 X_{iT} + \beta_2 H_{iT} + \beta_3 Q_{jT} + \varepsilon_{iT} \\
 (2) \quad H_{iT} &= \gamma_0 + \gamma_1 X_{iT} + \gamma_2 k_{iT} + \gamma_3 G^H_i + \varepsilon_{iT} \\
 (3) \quad k_{iT} &= \delta_0 + \delta_1 X_{iT} + \delta_2 H_{iT} + \delta_3 G^k_i + \varepsilon_{iT}
 \end{aligned}$$

where A is academic achievement; X, features of the individual and the family; H, the state of health of that person; Q, the categorization of the school; k, cigarette smoking; G, the genetic component; i, the number of the individual making up the sample; and T represents the course attended by the adolescent considered. As the error terms were correlated with the endogenous variables and the covariance between the three error terms was assumed not zero, they estimated the model applying, in addition to Least Squares Ordinary, Minimal Squares in two and three stages. The results of the estimates can be seen in the appendix of the cited work.

<sup>19</sup> The inclusion of variable G avoids the existence of bias due to the omission of explanatory variables in the model, while highlighting that the genetic markers chosen to have explanatory power over the variable we are trying to analyze, which is academic behavior.

been overcome, since the empirical evidence seems to confirm that both conditions, heredity and environment, influence our characteristics to be determined in each case.

3. Benjamin et al. (2013) study the relationship between the genetic code, permanent income and wealth,<sup>20</sup> and find results that allow us to assume that permanent income is influenced by the genetic code, given the high correlation found between the income of parents and that of children when they are monozygotic twins, higher than that found in dizygotic twins. At the same time, they analyze the methodological problems facing genoeconomics and consider that there is evidence indicating that certain behavioral characteristics are heritable, that the context in which an individual develops is less important to explain their behavior than genetic endowment, and that there is a very important part that is not explained by either of the two factors. Finally, they warn that there is a long way to go, when it comes to risk aversion, the intertemporal discount rate and altruism, among many other issues where genetics can help economists.

4. The second way of working, referred to above, is the one used by Beauchamps et al. (2011), which present, by way of example, a study<sup>21</sup> on the influence of the environment and genetic transmission. In a first stage they estimate the following equation:

$$\text{Edu} = \beta_0 + \beta_1 \text{SNP}_k + \beta_2 \text{PC} + \beta_3 \text{X} + \varepsilon$$

where Edu are the years of education;  $\text{SNP}_k$  is the number of copies of the smallest allele (0, 1 or 2) that an individual has in  $\text{SNP}_k$ ; PC is a vector of principal components;<sup>22</sup> X, a vector with control variables;  $\varepsilon$  is a random error term with zero mean and constant

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<sup>20</sup> This work, as can be seen in the references, has 22 authors, those who belong to different universities or research institutes located in different countries and research centers, dedicated to economic, biological or medical studies, which gives an idea that it is an interdisciplinary task, which brings together scholars located in places very distant from each other.

<sup>21</sup> Framingham Heart Study, started in 1948 and extended over three generations.

<sup>22</sup> In the statistical appendix of the work that we are discussing, in <http://e-jpe.org>, are defined both variables precisely.

variance and the  $\beta$ , parameters to be estimated. They worked with a sample of 8,496 individuals and 363,776 SNPs, for which they ran this last number of regressions, which gives an idea of the volume and complexity of the statistical work involved in this type of studies. They selected the twenty SNPs that were most significant, taking into account that, given the enormous number of regressions, false positives would appear. The results obtained were satisfactory, but then, in a second stage in which they applied the way of working referred to above as the first of the methodologies in use, they tried to verify the results obtained and analyzed the relationship of those twenty SNPs with the years of education in another sample and the results were quite discouraging, since of the twenty SNPs analyzed, in only nine cases the signs of coefficient coincided. This tells us that we must be attentive to false positives, and that all statistical studies must be very well confronted and verified.

5. Regarding education and genetics, the work of Lee et al. (2018) stands out, which was carried out by a large number of authors (77) and laboratories and consortia (3), who analyze a sample composed of 1.1 million people, and is one of the most complex to which we have had access. As education is related to health, in addition to problems studied by the social sciences in general and economics in particular, the databases created to analyze problems related to diseases have data on education, which made it possible in that case, to study a very large number of observations. They applied the GWAS method and found numerous SNPs related to the years of education received and discovered that most of the genes involved are those that are related to communication between neurons and that genome characteristics explain 11% of the variance of results in the education received.

6. In a similar approach to the question, van der Loos et al. (2010) analyze whether the conditions for someone to be a successful entrepreneur have something to do with their

genetic code. They consider as an entrepreneur one who is self-employed, (without considering the size of his enterprise), in several different alternatives: never employed, only sometimes or always, and explain the way in which, joining several studies come to gather a sample composed of about 70,000 individuals, whose genotype is known and 500,000 SNP can be analyzed. They use the GWAS procedure and apply the meta-analysis technique, obtaining satisfactory results but still unreliable given the small sample size.

7. More recently, Krammer and Gören (2021) analyzed the relationship between entrepreneurship, which they express with an indicator called *Total-Early Stage Entrepreneurial Activity (TEA)*, which measures entrepreneurial activity in a society and is elaborated for various countries, by quantifying the adult population that is involved in the creation of a new business or one that is in gestation, which is explained by several independent variables. On the one hand, a variable showing the effect of the genetic code, which is expressed by DRD4 gene,<sup>23</sup> related to the production of dopamine, which has effects on personality and competitiveness, and is linked to entrepreneurial temperament.<sup>24</sup> On the other hand, it uses other independent variables, among which are the GDP, the capital stock, the degree of openness of the economy, the urbanization rate, corruption, legal certainty, unemployment, the age of the population and its growth rate, immigration, some *dummy* variables to capture the geographical location and some others. They make a cross-sectional estimate using data from 97 countries. They estimate the equation by Ordinary Least Squares<sup>25</sup> and obtain satisfactory results because almost

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<sup>23</sup> The DRD4 gene has four exons, of which the alleles of the third are of interest (which are segments of the ADN that contain information to produce a certain protein and make up the mRNA).

<sup>24</sup> The way they construct the values of this variable is explained in detail in Seeing (2016).

<sup>25</sup> He also does some tests with the Instrumental Variables method. It is necessary to point out that they do not use GWAS, because they start from a certain gene and analyze if it is statistically significant, while this method starts from the characteristics of the phenotype and looks for which genes they are related.

all the variables are significant, which indicates that the entrepreneurial spirit is linked to the genetic code (*nature*) and also to the environment (*nurture*), which indicates that both elements influence the explanation of the dependent variable, thus contributing to the clarification of the debate to which we referred above.

8. A novel way to study these issues is the one used by Molins et al. (2022), who apply an alternative way of investigating the current state of studies of the relationship between certain genetic characteristics and risk aversion. For this, they identify 23 works through *Pubmed* and *Science Direct*,<sup>26</sup> following the method PRISMA (*Preferred Reporting Items for Systematic Reviews and Meta-Analysis*), a procedure used to analyze the quality of papers. They begin by pointing out that both risk and loss aversion involve behavior that might not be rational. Most of the studies they have analyzed support the hypothesis that risk aversion is associated with the pathway followed by serotonin and dopamine, although not its totality. However, they do not find agreement as to the genetic cause of loss aversion, although there are indications that the two are related<sup>27</sup> and claim that there is a long way to go. The progress made so far is insufficient as to have a definitive idea on this issue.

9. The relationship between genetics and income is studied by Hill et al. (2019),<sup>28</sup> who analyze a sample composed of 286,301 people and identify 30 *loci* (of which 29 were previously unknown). They conduct a complex study, as they study genes that present *in*

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<sup>26</sup> They are databases that allow you to find works in scientific journals, which are among the best known and used by scholars of all disciplines.

<sup>27</sup> They analyze in detail diverse types of genes that are presumed to be related to risk aversion and loss, and their respective alleles. Among them are the SLC6A4, DRD4, and ANKK1 among others. The first is the gene that produces the serotonin transporter protein.

<sup>28</sup> In this work appear 17 co-authors, who they say, have collaborated equally. It is noteworthy that all are geneticists, and none is an economist.

*pleiotropy*,<sup>29</sup> and find relationships between environment, genes, nerve transmitters, brain structure, intelligence, education and income. They start from a large number of genes, which they discard until they are left with only 24, of which 18 are linked to intelligence, which in turn they consider as one of the determining factors of income and consider that their work shows that genetics has to do with the existing inequalities in Great Britain.

#### IV. SELFISHNESS, COOPERATION AND ALTRUISM.

Wilson (1975) opened an interesting debate about the presence of altruism in human societies, which questioned the basic assumption of social sciences and also of biology. How can it be compatible for human beings to pursue other goals than their own well-being? What room is left for altruism? Wilson's answer is that, for human groups to survive, there must be individuals willing to sacrifice for others, so groups with more altruistic animals would have a better chance of survival.<sup>30</sup>

Becker (1976) argues that a society composed of individuals who maximize their individual benefits should generate a better situation for the whole, because although at first, we might think that if the altruists transmit part of their income to the egoists the total income would be the same, but the selfish will be encouraged to perform or omit actions that improve the situation of altruists, since that will benefit them, and so the total situation of society should improve. In this way he argues that economic theory has found an explanation for the benefits of altruism.<sup>31</sup>

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<sup>29</sup> It is called this form the ability of some genes or genetic variants to affect the phenotype in different ways, i.e., they influence more than one characteristic observed in the phenotype.

<sup>30</sup> See Navarro (2018).

<sup>31</sup> See also Colollard (1978).



It would be an important step forward to be able to determine whether certain genes are related to cooperation and altruism, meaning reciprocal help in the first case and unilateral aid in the second. Let's look at some work on this topic.

1. Garretón and Salinas (2007) argue that there are numerous cases in biology where it has been established that behavior of this type, in which some individuals sacrifice themselves for the survival of the entire species, is linked to the existence of a certain characteristic of the genome,<sup>32</sup> but that there are so far no studies that allow such a statement to be made in humans.

2. Ebstein et al. (2010) start from the premise that the size of the brain of human beings is more related to the interaction between them than to the challenges of the environment and consider that the growth of brain size is due to this situation. They analyze the genetic mechanism that makes us more or less prone to interact with our peers, and for this purpose they study the relationship between certain genetic markers<sup>33</sup> and social behavior. They refer to various research related to oxytocin and arginine, which are known to be related to social behavior, including some pathological behaviors, such as autism. Likewise, it has been possible to link its presence with certain SNPs. Among them we can highlight the tendency to social behavior, aggression, popularity, altruism in decision-making, affection for children, sexual behavior, life planning, infidelity, romanticism, leadership capacity, political attitudes and appreciation for music and dance.

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<sup>32</sup> There are many examples of this behavior, one of which are described in Mirsky (2009), where it is explained how if one group of bacteria is sacrificed for the others, these can survive adverse conditions that would otherwise cause their demise.

<sup>33</sup> Although they also use GWAS, they refer to the results obtained by analyzing twin monkeys and dizygotic, which are consistent with those.

3. Finally, it would be convenient to continue with the line of research proposed by Dawkins (2006) in his work *The Selfish Gene*, whose first edition was published in 1976 and which he has been improving up to the present. He argues that living beings are only a support for genes to survive and endure through their transmission, which leads him to question whether humans are selfish, altruistic or both simultaneously. Genetics, with its recent advances, could identify genes that explain some of these behaviors.

## V. EPIGENETICS.

On the other hand, a discipline has recently appeared, derived from epigenetics, which is known as<sup>34</sup> *behavioral epigenetics*, which analyzes how the environment, social context and behavior can influence the *silencing* of certain genes.<sup>35</sup> So far it has been used in medical matters, but it is interesting to note that studies have been carried out on alcohol and drug addiction. Their consumption produces the silencing of the genes that protect us from them, so it generates more consumption of these substances. Perhaps in the future these techniques can be used to analyze harmful economic behaviors. On the other hand, the silencing of some genes (*methylation*) has the characteristic of being transmitted by inheritance. This would give new life to the theory of evolution of Lamarck (1744-1829), proposed half a century before Darwin, but while the latter believed that the engine of evolution was natural selection, the former argued that it was adaptation to the

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<sup>34</sup> Epigenetics can be defined as the set of modifications that our genetic material undergoes that changes the way genes are activated or deactivated, but without altering them themselves. Same. "Epi" in The word Epigenetics derives from Greek "envelope", that is, above genetics (Carey,

2011). A good description of this discipline and its medical applications can be seen in Horvath and Raj (2018).

<sup>35</sup> For example, see Francis (2011).

environment that made different species change.<sup>36</sup> But if the silencing of genes were hereditary, in such a way that it would influence the phenotype of the offspring, it would produce changes in the theory of evolution (some people already talk about *evolution of the theory of evolution*), which would have implications for the social sciences, among other things because it would give new arguments to those who, in the debate *nature vs. nurture* to which we have referred above, take sides with the second, since it would recognize the effect of the environment on our genotype, but it would also constitute a novel and relevant topic of study for economists. If poor nutrition, in addition to causing problems for those who suffer from it, generates conditions that are transmitted to the offspring, the economic cost, as well as the human cost, would lead us to a reevaluation of the need to eliminate extreme poverty in our society, as well as to strengthen the fight against drugs and alcoholism.

## VI. DISCUSSION AND FINAL REFLECTIONS.

Everything we have seen leads us to the conclusion that the *nature vs. nurture* debate has been overcome, since we now know that both hereditary factors and the environment contribute to define the characteristics of the phenotype, in a proportion that varies from one circumstance to another, but that fortunately we can measure empirically with the methodology that is available.

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<sup>36</sup> Lamarck [1809], states “No it is the organs, that is, the nature and shape of the parts of an animal's body, which have given rise to its habits and its faculties. What on the contrary, their habits, their way of living and the circumstances in which the individuals from whom they come have found themselves, are those that over time have constituted the form of their body, the number and state of an organ, and the faculties, in short, that they enjoy. [...] It is known that this animal [the giraffe], the tallest of mammals, lives in the interior of Africa, where the arid and grassless region forces it to browse the trees. From this habit, sustained after a long time, in all individuals of his race, it turned out that his front legs have become longer than those of the back, and that his neck has been elongated in such a way, that the animal, without rising on the hind legs, raises its head and reaches with it six meters in height.”

On the other hand, progress has been made in the analysis of the relationship between the genetic code and economic behavior, and as we have seen, genetics influences to a variable extent characteristics of the genotype such as entrepreneurship, risk aversion, addictions, and education, among others.

However, these procedures are still in their early stages of development and deserve important questions, the most significant of which, in our opinion, is related to the choice of probability for accepting or rejecting the hypotheses considered. If we choose a very low value, we run the risk of committing a type I error, and otherwise of falling into a type II error. This choice cannot be made on a single criterion but depends on the nature of the model we want to estimate, so it has a high degree of subjectivity, and sometimes of arbitrariness. On the positive side, these techniques are evolving rapidly and maybe we can have more confidence in their results soon. We do not know what will happen with this line of studies, but we believe it is worth continuing to work on it to better understand the reality in which we are immersed, and although there is a risk that it may be a dead end, we understand that it is worth traveling the path.

If we could have more conclusive results, perhaps we could design economic and social policies on clearer and more consistent bases, considering the different starting conditions of each human being, precisely to help those who are less gifted.

The consequence of the above is that, in our opinion, the results we have today should not be used for policy design.

Of course, all of this has an ethical side that must be taken into account. Is it legitimate for us to intrude into the deepest part of a human being, such as their genetic code, and then treat them based on what we have interpreted? But on the other hand, if we can help those who have a different conformation and we do not do so, are we not missing an opportunity for them to be in a better situation? These are questions for which we will

have answers when our knowledge of these issues is much more precise, and we have advanced much further along this line of knowledge. Auffray (2004) wonders, "Doesn't exploring the genome run the risk of promoting a reducing view of man and justifying new forms of inequality? Will the ethical procedure allow for balanced cooperation among all social actors, respectful of human dignity, a factor of well-being and peace for future generations?" But there is something we are sure of, and that is that if we want to reduce inequality, we should first fully understand the mechanisms that produce it, and genetics can be useful in this task.

Finally, we wish to point out that economics should be more aware that human beings are fundamentally living organisms and have a closer relationship with biology, from which it has distanced itself over time. This shows that the sciences are interdependent and that, while it is necessary for specialization to be increasing, a certain degree of generality is also needed, which would lead economics to adopt a different methodological perspective.

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