From Genes for Intelligence to Our Understanding of Genes

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From its very beginnings, this century has been under the sign of genetics. Indeed, it was in 1900 that the laws established by Mendel in the mid-nineteenth century were rediscovered. In that same year, Landsteiner identified the first human blood typing, the ABO system. At that time, agronomists, eugenicists, and physicians were the principal agents of the development of genetics. The chromosome theory of heredity was asserted beginning in 1911; it was followed in the 1940s by the understanding of the role of the gene in cellular metabolism, and in 1954 by the explanation of the structure of the DNA double helix. The pre-eminence of genetics was to increase through the second half of the century, particularly in the 1970s with the advent of genetic engineering.

In our day, the science of genetics reigns triumphant. It has extended its domain over vast sectors of economic and social activity, and it monopolizes the attention of the medical community. It stirs up political debates and it transforms and fuels many a philosophical viewpoint. If it has made possible advances in food production and the eradication or containment of several rare diseases, it has also guided several genocides and contributed to imposing a determinist and biologically oriented vision of intellectual activities and behaviors, to the point of modifying the nature of human relations at times.[1] Like many human enterprises, genetics thus exhibits the double face of Doctor Jekyll and Mr. Hyde.

Scientists in the second half of the 1970s were already issuing warnings against the "rush to treat social and political questions biologically" and against "the ascendancy of biological representation in the practice and apparatus of social control."[2] Recent

progress in genetics only increases this need to reflect on the place of science in our society, in particular by fundamentally questioning its neutrality and objectivity. In our day, these reflections most often come down to ethical questions, including quite radical ones such as the following: is "progress" inexorable?[3] Are some areas of research off limits?

In order to put the current triumphalism of genetics (particularly human genetics) in its place, it is useful to cast a look backwards on some lessons that history has taught us. One of these examples is supplied by Provine, who analyzed the way in which supposedly "objective and neutral" scientific knowledge on the subject of "race crossing" or "interracial" mixing, has in reality varied with the historical evolution of various ideological and cultural positions on the matter.[4] Beginning with Sir Francis Galton at the end of the nineteenth century, and through the beginning of the twentieth century, numerous observations and experiments, performed principally but not exclusively on animals, led geneticists and anthropologists to the more or less categorical conclusion that an interracial mix could only produce undesirable results, coyly labeled "disharmonious." Certain agnostic scientists, such as Pearson around 1930, and later Huxley and Haldane, criticized such positions, but they confined themselves to pointing out the methodological or statistical insufficiency of the studies that led to such conclusions. At the dawn of World War II, there was no record of decisive progress that might effectively cast doubt upon the previous conclusions by consensus.

However, between 1938 and 1946, the scientific community underwent a veritable ideological awakening. This electroshock was clearly provoked by the excesses of the Nazi regime, which legitimized its repression on the basis of this old scientific consensus on the "disharmony" of interracial mixing. This belated awareness of a political reality that posed a challenge to their responsibilities as scientists radically altered their views of the earlier observations and experiments, leading in turn to a global and fundamental revision of the scientific interpretations of facts that until then had been considered well established. This change found concrete expression in Dobzhansky's 1946 declaration that "the widespread belief that hybrids of human races are inferior ...

must be classified as a superstition."[5] Similar pronouncements were issued by institutions such as UNESCO.

Thus, once the mirage of neutrality was dispelled, research that had been considered "objective and neutral" turned out to be deceptive. Provine, who analyzed this process in detail, concluded that "The real danger is not that biology changes with society, but that the public expects biology to provide objective truth, apart from any social influence. Geneticists and the public should realize that the science of genetics is often intertwined with social attitudes and political considerations."[6]

This message, voiced twenty-five years ago, remains entirely a propos today, and it explains the caution with which questions on the heredity of cognitive aptitudes and behaviors must be approached.

These last themes are obviously far from unexplored; they were already the particular subject of intellectual disputes in the nineteenth century. Let us recall the debates on the role of social or biological heredity in the expression of criminality, of mental retardation, or of "genius." Since the work of Francis Galton, this matter of the heredity of intelligence and of behavior has continually resurfaced in the scientific literature and in the media, demonstrating that, after more than a century of research, the case is neither definitively nor clearly closed. As hackneyed as it is, this old theme continues nevertheless to provoke numerous polemics, both scientific and ideological alike.

However, research methods have been perfected, and models have been developed with greater formality and complexity, giving rise to the hope that simplistic arguments that oppose genes to environment, innate to acquired, or, to use Galton's terms, nature to nurture, might become utterly obsolete.[7] Similarly, these developments gave reason to hope that reductive or mechanistic approaches would be abandoned. Yet such is not the case. Many errors of interpretation stem from a poor understanding of genetic models and from a certain blind propensity to wilfully misunderstand the hypotheses they underlie.

In what follows, I shall try to understand and analyze how some of the deviations of human genetics have come into being and persisted, when genetics enters into territory that is no longer confined to the strictly biological.

Pan-Mendelism

The rediscovery of Mendel's laws was a major event in the development of the biological sciences. These laws made it possible to understand why some of the biological traits that are observed in parents can also be found in their children, and why brothers and sisters may share these traits. Since Weismann (1892), it has been known that these resemblances are due to entities known as "genes," which are present in every individual in pairs, one of which is transmitted by the mother, chosen at random from the two genes she possesses, and the other transmitted by the father, also chosen at random from the two that he possesses. A child and his father or mother therefore share half of their genes, and two brothers or two sisters have, on average, a quarter of their genes in common.

Let us recall that these laws of transmission apply to discrete biological traits such as the color of flowers or the shape of seeds. They apply straightforwardly to blood type (ABO, Rhesus factor), to tissue and immunity groups (HLA, Gm), and many other traits. Mendel's laws are so effective in resolving questions that had baffled many a great mind before him that there was a powerful temptation to use these same laws to explain any resemblance whatsoever between related persons, including those resemblances that were not considered a priori to have any obvious biological foundation. Thus, in 1911, Davenport proposed to explain criminality or nomadism by genetic heredity. Other authors, as we have seen, used similar arguments to explain that racial mixing was genetically harmful because it caused a genetic "disharmony" in offspring, thus fostering criminality, mental retardation, and laziness.

These attempts to apply Mendel's laws in such varied domains as anthropometry, physiology, psychology, or sociology received generous support and political backing at the highest level. Thus, it was thanks to Rockefeller and Harriman that Davenport founded the Eugenics Record Office, whose mission was to carry out such studies by gathering genealogical records for thousands of families. The influence of such studies was felt well beyond the middle of this century. Scholarly books in biology still bear wit-

ness to it. The genealogies of great families of mathematicians, musicians, and painters served to illustrate the heredity of aptitudes, in keeping with the views developed by Galton in his book *Hereditary Genius*.[8]

But the most salient offshoot of pan-Mendelism came to the fore in the 1970s, in the excesses of sociobiology. Indeed, many of the theoretical developments of sociobiology were based on the concept of the gene as applied to behaviors such as altruism, selfishness, aggressivity, sociability, the capacity for innovation, and so forth. Such was the case with Lumsden and Wilson's notion of *"culturgens"* (1981)[9] or Dawkins' *"memes"* (1976)[10]. While the notion of the gene is often expanded in such approaches, two characteristics of the "true" genes of genetics are retained: the notion of the simple, discrete entity, to which sociobiologists attempt to reduce the complexity of a behavior, and that of transmission, which is sometimes generalized in order to escape Mendel's overly rigid rules. In fact, no observation can *directly* support the hypothesis of a gene for such behaviors, since no one has been able to prove their universal transmission according to Mendel's laws from generation to generation. But sociobiological models exempt themselves from the requirement of proof, maintaining that the validity of the genetic hypothesis is a given as long as it furnishes an accurate explanation for certain sociological, cultural, or ethnological observations. In this reductive universe, the fitting of a model to the data is sufficient to validate the model's hypotheses.

Another illustration of the pan-Mendelian trend is manifested today in medicine and in the behavioral sciences. Research on genes is becoming an all-consuming activity, as we shall see below.

The Breakdown of Genetics

A second important advance in the field of genetics was achieved in 1981 by the statistician and geneticist Fisher. Indeed, Mendel's laws provided no explanation for resemblances among related individuals for continuous traits such as height, head dimensions, or ... mental faculties. Galton had devoted significant time to this

problem, without being able to propose any satisfying explanations. Fisher developed a genetic model capable of accounting for familial correlations. He conceived of continuous traits as being determined by the existence of numerous genes that are mutually independent, each one making a small contribution to their expression. Other non- genetic causes could also contribute to the ultimate expression. This dissection of a trait into different components is clearly artificial. As an example, let us consider the intelligence quotient (IQ). How can we say that with an IQ of 110, the first 90 points could be explained by genes and the remaining 20 points by environment? This breakdown also presents the major disadvantage of ignoring a term of interaction that would express the fact that the influence of genes on the trait being studied is not necessarily independent of the environment. It is particularly reductive to disregard this interaction, as in the case of IQ, since doing so is tantamount to seeing the genetic differences between the IQ values of two individuals as being of the same importance, whether these individuals are raised in a favorable environment or a disadvantaged one.

This non-interactive or "additive" model is the essential foundation of what is called quantitative genetics as applied to IQ. Its objective is to identify the causes of IQ *disparities* in a population or in a group of people. From a statistical point of view, this disparity or difference is expressed by a statistical parameter called "variance," which grows smaller as a function of smaller IQ differences among the individuals. The method thus proposes to break down this variance and to quantify the part that is thought to be due to genes and the part thought to be due to other causes, termed environmental as a simplification. The relation between the genetic variance and the total variance of the trait is referred to as heritability.

To estimate this heritability, it must generally be supposed that there is no correlation between the effects of the genes and those of the environment. This hypothesis amounts to the assumption that all the particular forms of a gene (called *alleles*) can be found with the same probabilities in any environment. There is therefore an assumption that the same alleles are found as frequently in a favorable environment as in a disadvantaged one. The estimation of her-

itability then relies on calculations and comparison of correlations between different types of family members, such as monozygotic or dizygotic twins, brothers and sisters, half-siblings sharing the same mother or the same father, parents and their children. Added to this are studies of adopted children and of family members living together or separated at various stages of childhood.

All of these successive hypotheses – the additive model, the absence of correlation between genes and environment – have the advantage of setting up conditions that promote the testing of simplified (some would say simplistic) models. The number or parameters that define this model, as for example the various components of variance, then become reasonable. They can therefore be estimated with great precision – even if the model is false. These simplifications have the major drawback of removing any degree of universality from the conclusions. As a result, this breakdown of variance has only a very limited significance: it is valid only for a given group of persons, in a certain narrow range of environmental variation, and for a precise time period. In other words, if a different population, environment, or generation is examined, this breakdown cannot produce the same results, and comparisons of "heritability" lose all usefulness.

The objection that is often raised to such reservations about quantitative genetic models comes from the fact that these models give good results in experiments with animals and plants, as for example in efforts to improve feed grain or dairy production. But what relation is there between an experimental science on the one hand, in which crossings of animals or plants can be controlled for uncontestably biological criteria (such as the quantity of lipids contained in milk or the ratio of lean matter to fatty matter), and on the other hand, what can be cobbled together from observations of the effects of imperfectly controlled situations (such as adoption situations or socio-economic criteria) on a trait, a cognitive aptitude, or a behavior, which are particularly difficult to measure and whose biological nature is by no means evident?

The utter inanity of research on the heritability of IQ or other behavioral traits was largely demonstrated in the latter 1970s,[11] but not without spirited resistance. In our day, even the most ardent defenders of the genetics of behavior have come to admit

this vacuousness,[12] without however fully accepting the implications of this admission.

Indeed, works using this model continue to flourish in the scientific literature, as witnessed by an article recently published under the title "The Heritability of IQ."[13] The originality, so to speak, of this work lies in the way it combines together in one model the results of some 212 studies already published on the subject since the beginning of the century. It consists then of a compilation of necessarily heterogenous studies, each of which takes a different approach to evaluating IQ patterns among family members (monozygotic and dizygotic twins, siblings raised together or separately, parents and children, adoption studies, and so on). The perfectly classic model used is that of the breakdown of variance whose limits we have just seen; the existence of interactions, of correlations between genes and environment, of genetic and environmental differences among the samples, are all completely disregarded. We may wonder about the innovation of such a "meta-analysis." In fact, the authors attempt to integrate into their model an additional effect, the maternal influence: the IQ's of children of the same mother tend to show greater similarity because these children shared the same prenatal conditions. The conclusions of this work therefore moderate the role of genes in determining IQ differences, according greater importance to maternal influences. Perhaps it would have been less deceptive if the article were entitled "The Implications of Maternal Effects on Similarities in IQ." The deliberate choice of another title seems to prove that the goal of this work is not so much to assert these maternal influences as it is to attempt to take them into account with the sole aim of "refining" the measure of heritability in order to give it greater weight; the trade-off requires only a minor concession as to the value of the model. In another article in the same issue of this journal, "The Democracy of the Genes," McGue points out this contradiction, but only in order to reassert the classical biology-driven refrain: "Research on the nature and nurture of IQ is converging on the view that human intellectual ability has a strong, but malleable, biological basis."[14] Everything thus boils down to biology, but in a magnanimously democratic gesture – *démocratie oblige* – biology allows the little people a voice in

numerous causes. According to this interpretation, genes remain the all-powerful *deus ex machina*, even in their very moments of abdication. We can only wonder when the democracy gene will be discovered.

Genes Exist: We Can Find Them

A virtually irrefutable argument in favor of a genetic foundation for cognitive aptitudes, behaviors, and personality traits would be the ability to localize and materialize on chromosomes one or more genes that act directly upon them. Three methods are generally used in an attempt to achieve this end.

The first consists in looking to see whether people who show the trait possess a particular form of a gene, an allele, that is not present, or that is possessed with less frequency, by persons who do not show this trait. This association between the trait and the genetic marker would thus be the first argument in favor of a relation between the two. However, it does not constitute a proof, for such an association can have other causes, which are well documented by population genetics (selection, admixture of populations, random variations, and so forth). Nevertheless, it is possible to get closer to a proof by invoking a second method, which endeavors to investigate whether the trait and the marker are jointly transmitted from parents to their offspring, according to Mendel's laws. If the trait and the marker are linked in this way, it means they obey the same genetic cause, or that the causes are topologically close together on the same chromosome. In order to determine which of these possibilities is operative, recourse must be had to a third method, which will investigate directly the relation between the product of the gene (a protein, an enzyme, etc.) and the expression of the trait. This last approach takes us outside the realm of genetics to the field of molecular and cellular biology or to that of pharmacology.

A) Associations

The study of associations came into existence with the discovery of the first genetic markers in human beings. The ABO system of

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blood types was a natural candidate for testing this sort of association. Carried to an extreme, some such studies have reached the point of caricature. Thus Léone Bourdel devoted an entire book the relations between blood types and "temperaments."[15] According to this author, blood type A governs "intimacy, the domain of affective harmony, reaching beyond the self," along with the capacity for contemplation and passion; group B, in contrast, characterizes rational, deliberate and authoritarian tendencies ... These conclusions, drawn from observations at the level of individuals, were extended to entire peoples. Those peoples with predominantly type-A blood, for example, are said to wage war only in "self-defense of their affective intimacy," whereas the peoples with predominantly type-B blood are considered to be innately "the most spontaneously belligerent" peoples, for whom war is a "natural function."

These wild imaginings obviously have no statistical foundation, but they are reported in a highly "scientific" language in the series "Bilan de la science," directed by a scientific guru of the period (Leprince-Ringuet). The resulting mirage has played an active role in attempts to explain human nature in biological terms.

This theme was still alive and well as of 1973. For example, Gibson *et al* [16] reported that IQ is higher in individuals of blood types O and A2 (a variant of type A) than in individuals of other types. The difference is significant, although particularly small (3 percent).

The insistence on finding an association between a genetic marker and a behavior always ends up exacting a toll. Dumont-Damien and Duyme[17] report that between 1956 and 1991, the more than 140 studies of association that were carried out in connection with alcoholism explored close to 50 different markers (ABO blood types, rhesus factor, HLA, various enzymes playing a role in the metabolism of alcohol, and others), some of which were revealed to be significantly associated with certain forms of alcoholism.

Recently, an association has also been reported between a marker located in the gene for the dopamine (a neurotransmitter) receptor and the personality trait of novelty seeking. In the same vein, a number of works demonstrate associations with homosexuality, criminal tendencies, and so on.

Finally, IQ itself has not escaped association studies. Plomin's team[18] thus selected three groups of persons with IQs averaging 130, 105, and 82. Then 100 genetic markers were tested for possible associations. Three of these turned out to be significant, the strongest association being with mitochondrial DNA. This is an important point, since this DNA is transmitted only by the mother. Therefore, if the relation between mitochondrial DNA and IQ is grounded in fact, we would expect IQ to be transmitted by the mother and not by the father, something for which we have no evidence as of yet.

In all of these investigations, the essential difficulties – apart from the methodological questions already discussed above[19] – lie in the definition of the trait and the classificatory distinction between those who carry it and others. This problem is particularly flagrant for alcoholism, which is the expression of multiple risk factors: neurophysiological, psychological, familial, professional, and perhaps genetic. The same holds true for a personality trait or for IQ, which are only statistical composites of performance on multiple tests.

B) Linkage

Methods of investigating links between a genetic marker (whose precise location on a chromosome may be known) and IQ, a cognitive aptitude, or other behavioral traits are clearly more conclusive than studies of association. Research on links attempts to determine whether the "candidate" gene for a trait is transmitted at the same time as a genetic marker in family genealogies. Once again, the definition of the trait and the to some degree arbitrary decision to assign its presence or absence in an individual are rife with ambiguities. Another handicap is our ignorance with regard to specifying a priori the mode of transmission and expression of the candidate gene, which remains hypothetical and which we are attempting to concretize. Is it recessive or dominant – that is, is it expressed only when it is present in two copies, or is one sufficient? Is the expressivity or penetrance of the hypothetical gene total or partial – in other words, is it always expressed whatever the circumstances, or only under certain conditions, controlled or not controlled, such as those of age or environment? What is the

gene's frequency in the population? What is the probability of its appearing in sporadic non-genetic cases, or the frequency of mutation *de novo*? The choice of a model is therefore particularly delicate, for upon it will depend the ultimate conclusions. It might therefore be said that the systematic search for a gene always has a strong probability of succeeding without undue strain. For even if one does not find *the* gene for the trait, there is still a chance of proving the existence of a gene with variable penetrance. And even when the latter fails, there is still the possibility of finding one or several genes for "susceptibility," that is, genes whose presence indicates fertile ground for developing the trait, always under certain conditions. Moreover, if the physical localization of the gene on the chromosomes is not possible, it can always have a "statistical" existence." And as a last resort, there is always the possibility of saying that the gene is so rare that it has been found in only one family, even in a single individual; or even that it has heretofore eluded detection but that nothing is lost by waiting ... As we can see, the notion of the gene is so elastic on this level, between physical reality and virtual existence, that it is virtually impossible to escape it.

Finally, it is possible that there is not just one "candidate" gene to explain a trait, but rather several that can come into play alternatively, synergistically, or independently, depending on the individual.

This mad hunt for the slightest effect of a gene is one of the faces of contemporary pan-Mendelism. Obviously, it has only a minor impact in the area of public health, though it absorbs a major expenditure of energy.

The Gene That Does the Most Does the Least?

These studies of linkage have thus far never shown the existence of links between cognitive aptitude, personality traits or behaviors, with the exception of specific pathological situations. Indeed, positive results have been obtained in some cases of mental retardation, such as those provoked by phenylketonurea or the weak X chromosome. In the case of altered intellectual functions, such as in Alzheimer's disease, studies of linkage show that the genetic

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forms with dominant mode of inheritance involve only about 4 out of every 1000 cases, but they represent from 10 to 20 per cent of early-onset cases (before the age of sixty). Several different mutations of the genome appear to be responsible for this neurodegenerative disease.[20] Likewise, a certain Dutch family appears to demonstrate a mutation of monoamine oxydase which might be linked to a type of impulsive violent behavior.

The encouraging results in the search for genes implicated in different forms of mental retardation, mild or severe, are often used as arguments in favor of the determinant role of genes on behaviors or cognitive aptitudes. The reasoning is as follows: since there exist genes that account for mental retardation, therefore there must also, necessarily, be genes for intelligence. This deduction amounts to viewing mental deficiencies, which are incontestably of genetic origin, as being of the same order as "intelligence." But there is no reason that genes implicated in mental deficiency, if they do exist, should be the same as those that dictate normal or exceptional intelligence. The causes that lie behind dysfunction are not necessarily the same as the causes of normal functioning.

This reasoning, however, has been put forth recently, not without some rather amusing – if unwitting – implications.[21] We know that the "candidate" genes implicated in these mental deficiencies are quite numerous, numbering somewhere between 300 and 400, and for the most part they are located on the X chromosome. This situation is not without consequences. For this chromosome is found in two copies (XX) in women, whereas in men one X chromosome is associated with one Y chromosome (XY). Since many of the genes that are responsible for mental retardation are located on the X chromosome, that, according to the author, is where the intelligence genes are also to be found. This generalization, a rather hasty one, leads to various far-fetched deductions, however logical they may appear here, as well as to some marital advice for those who wish to guarantee "superior" offspring.

Indeed, if "intelligence" is transmitted with the X chromosome, then the father no longer has any influence on the intelligence of his son, since in that case he transmits only his Y chromosome. If he wants to maximize his son's intelligence, he must therefore

select his parents-in-law with great care. Likewise, whomever a woman may marry, the intelligence of her son will depend only on that of her own parents (since she gives her son the X chromosome inherited from her father or her mother). On the other hand, if she wished to insure her daughter's superior intelligence, there is no point in choosing her father-in-law carefully, since he plays no role in her daughter's brilliance (he gives her no X chromosome). But she will have to pay attention to the "intellectual quality" of her mother-in-law ...

The genealogies of famous families are given by the authors as confirmation of this simplistic theory. For example, that of Sir Charles Darwin and his cousin Sir Francis Galton, undeniably two great scholars of the nineteenth century. There again, we are sorely deceived, for the genealogy proposed by these authors is carefully purged of all the weaker minds, the unstable or suicidal individuals, those suffering from depression, dyslexics, and the like.[22] As for the women, they are never counted as "brilliant" – probably because no one ever dreamed they might be ... But do they not possess an X chromosome, just as men do?

This example shows how the obsession with the gene at any cost, and the extension of a possible pathological reality to the realm of normality, can result in aberrations, even leading some authors to advocate certain types of behavioral strategies.

All of this pseudo-science, which appears in prestigious reviews indiscriminately mixed together with true science, furnishes solid proof – by counter-example and by hyperbole – that the social and moral perception of intelligence is more determinant than a genetic pseudo-definition based on multiple series of biases, errors, and falsifications, which persist despite having been disproven already a thousand times over.

* * *

From this rapid survey of the relations between genetics, cognitive aptitudes, behaviors, and personality, one is tempted to admit the relative failure, temporary powerlessness, or absence of universality of the conclusions. The methods of genetics, both classical and modern approaches alike, could no doubt explain a *tiny* fraction of *certain* behaviors linked to *certain* forms of deviance in a *limited*

number of families, as the above examples have shown. It is not impossible that this knowledge might shed some light on certain molecular or neurophysiological mechanisms, and this information would enrich the current pharmaceutical repertoire. But that no longer pertains to the field of genetics.

The limits of genetics quickly become clear in the domain of cognitive sciences and social sciences. For example, a form of gene therapy that would make it possible to correct some deviations with respect to "standards" at the particularly fluctuating boundaries of these disciplines is not within the realm of the possible. Would it even fall within the realm of the desirable? Likewise, a reproductive selection based on genetic criteria (if their reality were indeed demonstrated) would raise the issue of justifying the distinction between negative selection (interrupting the transmission of unfavorable genes) and positive selection (promoting the transmission of favorable genes). This issue opens up the Pandora's box of a related problem – that is, eugenics.

Must we consign the radiant future of the gene to the rubric of science fiction? Up until now, as we have tried to show, the biologically correct discourse has placed DNA at the center of a rather totalitarian machinery. Thus, Plomin remarks that "the current enthusiasm for genetics should not obscure the important contribution of non-heritable factors, though these are difficult to investigate."[23] But this profession of faith regarding the importance of non-genetic factors is nullified a few lines later, with a reversion to positions that are directly in line with exclusively genetic thought: "The ostensible measures of environment appear to assess genetically influenced characteristics of individuals. In a certain way, individuals create their own experiences for genetic reasons." This statement coincides with McGue's interpretation of the pre-eminence of biological, despite its high degree of malleability.

Now, it is not inconceivable that all of these discourses are merely an ideological falsification of biological reality. Let us recall the question of racial mixing. For the gene must no longer be seen as a program whose inexorable execution governs all the details of our lives – cellular, hormonal, or social. At the level of populations, at the level of the individual, and at the molecular level, the mechanisms of the expression of genes do not obey rigid determinisms.[24]

Rather, they are subject to the play of probabilities, which are affected by all the structures and molecules that surround genes, all the stimulations of the physical, chemical, and human environment, and all the interactions with other "gene carriers."

Upon this complex tapestry, time also plays its role as the creator of unique histories, those that each of us is free to live out.

Translated from the French by Jennifer Curtiss Gage

Notes

1. Reference is made here to the growing importance accorded to biological kinship and to the desire to preserve one's genes for posterity through one's descendants, whereas kinship was first defined by affective bonds within an ethno-sociological context, without particular reference to biology.
2. Lévy-Leblond *et al.*, in *Le Monde*, 15 June 1977.
3. The lack of interest on the part of certain biologists and the ignorance of many technocrats with respect to these questions often stem from an unshakable faith in the inevitability of "progress." "One of the inevitabilities of scientific progress is that if something can be done then it will be done," according to Daniels *et al.*, in *Journal of Biosocial Sciences* 28 (1996): 491-507. Moreover, some ways of accentuating and encouraging the hope that may arise out of any scientific advance is in fact only a clever tactic for leaving one's hands free to cover up research whose consequences will ultimately escape control (on this subject, see the analysis by Axel Kahn, *Futuribles* 223 (1997): 5-27.
4. Provine, "Geneticists and the Biology of Race Crossing," *Science* 4414 (1973): 790-796. The word "race" is to be understood in its historical context.
5. Dunn and Dobzhansky, *Heredity, Race and Society* (New York, 1946), p. 114.
6. Provine, "Geneticists and the Biology of Race Crossing," p. 796.
7. Galton himself participated actively in this effort by developing the statistical concept of correlation.
8. Sir Francis Galton, *Hereditary Genius : An Inquiry into Its Laws and Consequences* (London, 1892; 1st ed. 1865).
9. Charles J. Lumsden and Edward O. Wilson, *Genes, Mind and Culture: The Coevolutionary Process* (Cambridge, Massachusetts, 1981).
10. Richard Dawkins, *The Selfish Gene* (Oxford, New York, 1976).
11. In particular by Feldman and Lewontin, and in France by Jacquard in *Eloge de la différence* (Paris, 1978).
12. "Estimates of heritability apply only to the population studied at that particular time, and under environmental conditions that prevail at that point," in

the words of M. Rutter and R. Plomin, "Opportunities for Psychiatry from Genetic Findings," *British Journal of Psychiatry* (1997), pp. 209-219.
13. Devlin et al., *Nature* 388 (1977), pp. 468-471.
14. McGue, "The Democracy of the Genes," *Nature* 388 (1977), pp. 417-418.
15. L. Bourdel, *Sangs et tempérament* (Paris, 1962).
16. Gibson *ect al.*, "Ig and ABO Blood Groups," *Nature* 246 (1973), pp. 496-499.
17. Dumont-Damien and Duyme, *Genetics and Alcoholism* (Les Editions INSERM, 1993).
18. Plomin *et al.*, *Behavior Genetics* 21 (1995): pp. 31-48.
19. And a few other questions whose technical aspects exceed the scope of the present article.
20. Campion *et al.*, "Les facteurs génétiques dans l'étiologie de la maladie d'Alzheimer," *Médecine/Sciences* 12 (1996): pp. 723-731.
21. G. Turner, "Intelligence and the X Chromosome," *The Lancet* 347 (1996): pp. 1814-1815.
22. Resta, "Whispered Hints?", *American Journal of Medical Genetics* 59 (1995): pp. 131-133.
23. Plomin, Owen, and McGuffin, "The Genetic Basis of Complex Human Behaviors," *Science* 264 (1994): pp. 1733-1739.
24. Kupiec and Sonigo, "Du génotype au phénotype: instruction ou selection?," in *Pour Darwin*, ed. Patrick Tort (Paris, 1997), pp. 1025-1034.