The Ontogenesis of Human Identity

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> "<<Es ist, als wären unsere Begriffe bedingt durch ein Gerüst von Tatsachen>>.
> Das hiess doch: Wenn du dir gewisse Tatsachen anders denkst,sie anders beschreibst, als sie sind, dann kannst du die Anwendung gewisser Begriffe dir nicht mehr vorstellen, weil die Regeln ihrer Anwendung kein Analogon unter den neuen Umständen haben."[1]
> (L. Wittgenstein, *Zettel*, § 350)

1. Introduction

Since the birth of western philosophy much time and energy have been spent on identity. Is it worth saying more on the subject? Such a question cannot have but a positive answer if philosophy is considered as a contextualised unending research into fundamental problems concerning human beings and their situation in history, in nature, and in society. If philosophy is really an unending research, its problems, its solutions, its arguments must depend on the historical and cultural context in which they have been formulated. *A fortiori*, this is true also for human identity.

In what follows, I will argue for a contextualised solution to the problem of human identity. This means that I will use results of contemporary disciplines that cannot be neglected if we want what we affirm has a value beyond the philosophical domain in which it has been formulated. In particular, I will resort to the biological sciences. Why should we, philosophers, forget biology, and therefore science, in dealing with human identity? Are we sure we are right in discussing the latter only from a purely philosophical point of view, and without considering, and sometimes also contradicting, what science teaches us? Are we sure that in this way we do not display philosophical *hybris*. Are we sure that, from this point of view, the scornful smile of the Thracian servant caused by the

[1] "<<It is as if our concepts involved a scaffolding of facts>>. That would presumably mean: If you imagine certain facts otherwise, describe them otherwise, than the way they are, then you can no longer imagine the application of certain concepts, because the rules for their application have no analogue in the new circumstances".

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proto-philosopher's tumble, magisterially discussed by Blumenberg (1987), must be really stigmatised?

If we glance at the history of modern discussions on human identity, we realise that Descartes, Locke, Hume, Leibniz, Kant were well-aware of the coeval science to which they sometimes actively contributed. Nevertheless if we run through the enormous contemporary philosophical literature on the same topic, we discover that science is totally on the sidelines, and its place is occupied by unreal and fictional thought-experiments, which are much used.

Let us think, for example, about the brain transplantation thought-experiment. Its first use may be dated back to Locke, and, given his scientific background, he was right in discussing it. But are we right in discussing it nowadays, more or less in the same terms and after almost four centuries of biological discoveries, in particular neurobiological ones?

If A's brain, or brain cortex, is transplanted into B, who is A? If half A's brain cortex is transplanted into B and half into C, who is A? If A's brain lay on a table in an anatomical theatre and if it is kept alive by artificial supports, where is A? If there is a tele-transporter ray that dissolves A here and now to reconstruct it in another place and in another time, what about A? If A's mental contents are totally transferred by means of a strange machine to B, who is A? Are we right in continuing discussing these cases?[2] Everyone is free to intellectually play as he prefers. I confess I prefer a different way of tackling human identity. I would rather ground my analyses in science than in science fiction.

Nevertheless, beyond a pure subjective preference, there are reasons that spur me to such a choice. The first concerns my belief that philosophy has to be useful also in non-philosophical domains, as has nearly always happened in its history. Nevertheless if we introduce fictional transplantations, fictional rays, fictional machinery, and so on, I do not believe that our conclusions are really interesting for others apart from ourselves.[3]

[2] On this discussion, cf. for example, Puccetti, 1969; Perry, 1972; Lewis, 1976; Parfit, 1984; Shoemaker, 1984; Robinson, 1985; Johnston, 1987; Wilkes, 1988; Noonan, 1989; Snowdon, 1991; Olson, 1997.

[3] For example, there is a lively debate inside biomedical sciences on the definition and criteria of death. This is an extremely important topic since, of course, it is preferable to perform organ transplantation when the donor is dead. But when is it dead? (cf. Boniolo, 2006). One of the seminal paper on this topic was written by Bernat *et al*., 1981. In it the authors, explicitly quoting some philosophers, write that they are totally disinterested in the "attempt to answer the speculation of science fiction, such as if the

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Some philosophers might reply they are doing metaphysics, not philosophy of science, or science. This is true. Metaphysics is a possibility, and personally I have nothing against it. It is on metaphysics that the castle of western thought was built a couple of thousand years ago. However, allow me to claim that to discuss certain topics physics and biology are more promising.

It is precisely from science that other reasons to discuss human identity without fictional machineries rise. What do you mean by brain transplantation? The entire brain transplantation, brain cortex transplantation, brain-stem transplantation, cerebellum transplantation, hippocampus transplantation? Are all of them technically possible? Just for the sake of discussion, let us suppose that even if they are not technically possible now, they could be technically possible in the future. Nevertheless there are still two problems.

The first one concerns our knowledge of the brain. Even if we know something about neurophysiology, we know almost nothing about how neurophysiology gives rise to concepts, judgments and reasonings, that is, the higher mental functions. It follows that any time we are discussing about them in correlation with human identity and brain (or a part of it) transplantation, we are discussing something we do not know enough about. That is, we are philosophing in a way that has been negatively stigmatised by many philosophers, for example by Locke: "I think not only, that it becomes the Modesty of Philosophy, not to pronounce Magisterially, where we want Evidence that can produce Knowledge; but also, that it is of use to us, to discern how far our Knowledge does reach" (Locke, 1690, Book IV, Ch. III, § 7, pp. 541–2).

The second problem springs from the fact that to discuss brain (or a part of it) transplantation forgetting the genetic or immunologic implications is rather bizarre and naive. Not only, those who discuss it usually forget the scholars who took these implications into consideration. Already since the first decades of the 20th century, in the biomedical field it has been extremely clear that when we examine human identity and organ transplantation we could not neglect the immunologic aspects of the matter. For example, in 1937 Loeb published his 'The Biological Basis of Individuality', where he wrote that rejection in the case of organ transplantation

brain continues to function independently of the rest of the organism" (p. 390). That is, most of the philosophical discussions on human identity grounded in fictional brain transplantation, or on some other fiction machineries, are simply put aside as non-interesting.

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reveals the "differential of individuality", that is, what distinguishes an individual from another (cf. also Loeb, 1945). This means that to correctly face the problem of human identity we should discuss the problem of immunologic identity before talking of brain transplantation.

Moreover the mental contents we have, that is, what, according to some philosophers, is the main feature of personhood, are also due (even if we do not know how) to the brain we have. Yet we have the brain we have also because we have the genes we have. Therefore, before discussing identity from the point of view of the brain and its mental contents, we should face it from the genetic point of view, as some biologists already did in the 1930s (cf. Jennings, 1930).

2. The plan

After making the apologia of the discussion of human identity into a strong biological frame, it is time to unfold the plan oriented towards an argued solution to the question on stage.

First, I will survey some moments of the human ontogenesis particularly relevant to my purpose.[4] In specific, the endowment of genetic identity, the ontogenesis of both immunologic identity and neural identity. In this manner I will show that human identity is what results from an ontogenetic process during which genetically, epigenetically, and environmentally governed properties reveal themselves. By *genetically governed properties* I mean those phenotypic properties which appear only thanks to the genetic expression. It should be noted, with reference to this point, that a genetically governed property is totally different from the *property of having a particular genome*. The former concerns a possible phenotypic result of the latter. By *epigenetically governed properties* I mean those phenotypic properties which appear thanks to the expression of certain genes belonging to certain cells activated by their interactions with surrounding cell populations and with their peptidic products.[5] By *environmentally governed properties* I mean

[4] With reference to a more phylogenetic approach, even if it does not contain a philosophical analysis but a popular account, cf. Buss, 1987.

[5] There is a discussion on the definition of 'epigenetic process' and 'epigenetic property', cf. the seminal Waddington, 1957; also Jablonka and Lamb, 2002, pp. 310–11; West-Eberhard, 2003, p. 112.

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those phenotypic properties which appear thanks to the interactions between cell populations, or even the entire human being, and the environment in which the latter lives.

Secondly, I will come back to philosophy to discuss more profoundly what developmental biology, immunology and neurobiology have taught. Therefore, I will try to offer a possible solution to the following problem:

> *"Given two human beings, B and B′, which is the set of the sufficient properties* $<P^{B}_{i}>$ *of B and* $<P^{B'}_{i}>$ *of B′, such that, at a certain time t, if* $<P^{B}_{i}>=<P^{B'}_{i}>$ *then B=B′?"*

While tackling this question, I will also face another one:

> *"Given a human being B, which is the set of the sufficient properties* $<P^{B}_{i}>_{t}$ *and* $<P^{B}_{i}>_{t'}$*, where t′>t, such that if* $<P^{B}_{i}>_{t}=<P^{B}_{i}>_{t'}$ *then* $B_{t}=B_{t'}$*?"*

This way of proceeding will clearly show that human identity has to do with something dynamic, something that concerns the genetic, epigenetic, and environmental appearance of necessary properties during the human being's development and life. Moreover, it allows me to implicitly present a different side of the problem of the correlation between human animal and person. This is a much discussed topic nowadays, also for its ethical implications, but almost always without resorting to real science.[6]

A last note to conclude these preliminaries. In the following, I will discuss an abstract (but not a fictional) living being from the time t_f, identifiable with the fusion between the pro-nucleus of its mother's ovule and the pro-nucleus of its father's spermatozoon, to a time $t<t_n$, where t_n is the time of the beginning of the necrotic processes.[7]

[6] Cf. Williams, 1970; van Inwagen, 1980; Parfit, 1984; Schoemaker, 1984; Lockwood, 1985; Johnston, 1987; Noonan, 1989; Unger, 1990; Lowe, 1991; Singer, 1995; Olson, 1997; Wiggins, 2001.

[7] The necrotic processes lead towards the entire organism's death, which is *a posteriori* recognisable by some associated changes such as *algor mortis*, *livor mortis*, *rigor mortis*, and *postmortem autolysis*. *Algor mortis* is the post-mortem decrease in body temperature; *livor mortis* is the purplish discoloration from settled blood in given body regions; *rigor mortis* is the muscle stiffening; and *postmortem autolysis* concerns the putrefactive changes. On the estimation of death time, cf. Hensshe *et al*., 2002.

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3. Spacetime properties

In modern times, Locke (1690, Book II, Ch. XXVII, § 4) advanced the proposal that given two non-compound bodies, if they occupy the same spacetime region they are the same. As is well-known, this thesis was contested by Leibniz (1765, Book II, Ch. XXVII, §§ 1–2) who pointed out that two different light rays or two different shadows may occupy the same spacetime region without being the same ray or the same shadow.[8]

The Lockean idea was reformulated in more formal terms by Lewin (1922) who took from Special and General Relativity the notion of *worldline*, that is, the set of the spacetime points occupied by whatever being during its existence. It must be remarked that, in this way, all the 3-dimensional morphological spatial modifications of a living being that occur in time since its birth are considered.

Thus Lewin proposed the so-called *criterion of genidentity*, then reconsidered by Reichenbach (1927^{1}–1958^{2}): let **B** be the set of the living beings and **W** the set of the correlated worldlines,

$$\forall B, B' \in \mathbf{B} \text{ and } \forall W, W' \in \mathbf{W}, [B =_G B' \leftrightarrow (W \equiv W')],$$

where "$=_G$" is to be read as "... is genidentical to ...", and a) W and W´ do not admit a solution of continuity; b) closed time-like curves in W and W' are not allowed. The continuity requirement (a) prohibits the "resurrection" of a living being.[9] Instead, the elimination of closed time-like curves (b) avoids strange meetings between a young and an old myself, i.e., the well-known "Grandfather paradox" (cf. Boniolo, 1999).[10]

Unfortunately Lewin's criterion has some problems, linked to the fact that it might be necessary but surely not sufficient. First, every living being loses some parts over its lifetime. Trivially, we lose some hair every day; more specifically there is the issue of the apoptosis, that is, the genetically programmed cellular death. Moreover, many times living beings split their worldlines into two

[8] It should be noted that if we wanted to tackle Locke's proposal and Leibniz' objections from the point of view of contemporary science, in particular Quantum Mechanics, the matter would profoundly change. In that case light should be considered also in its corpuscular nature. Moreover at a quantum level speaking of spatial regions for microscopic bodies means bumping into serious problems correlated with the quantum description (entanglement, uncertainty principle, etc.).

[9] For a debate about "resurrection", cf. Hughes, 2002.

[10] At this macroscopic level the problem of the striation of spacetime is not relevant.

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or more branches, and it should be noted that any worldline of a human being continues in time and persists in space also after its death.[11]

However, for macroscopic bodies such as the human beings, that usually do not resurrect and do not bump into spacetime wormholes, Lewin's proposal can be turned into the first necessary property which makes up the set of the sufficient properties characterising human identity. That is, I can state the *spacetime necessary condition for human identity*:

if $\boldsymbol{B}$ *is the set of human beings and* $\boldsymbol{W}$ *is the set of the correlated worldlines, then*
$\forall B, B' \in \boldsymbol{B}$ *and* $\forall W, W' \in \boldsymbol{W}$, $[B \equiv B' \rightarrow W \equiv W']$.

4. Life properties

I have just said that **B** is the set of human beings. I have also stated I consider the time interval from the fusion of the gametes to the beginning of the necrosis; that is, the time interval in which a human being is alive. How can we formulate the property of being alive?

We may start again with Locke, who in his 1690 masterpiece (Book II, Ch. XXVII, § 6) noticed that both the living beings and the non-living beings are organised beings, but with a really important difference: the organisation of a living being is such as to permit its life. More or less the same idea, even if with a totally different jargon and in a totally different philosophical context, is shared by Kant (1790) when in his *Kritik der Urteilskraft* he characterises living beings by their possessing a *Naturzweck* (or *Zweck der Natur*). That is, any living being has a *bildende Kraft*, which is correlated with what he calls *innere Zweckmässigkeit*. In other words, also Kant characterises living beings by means of a specific kind of organisation: the one which permits their life.

We may translate the particular kind of organisation that allows the human being to live, glimpsed at by Locke and Kant, into what nowadays we call metabolism. Therefore we may state that being alive means having functioning integrated metabolic processes.[12]

[11] On the biological body identity, cf. Boniolo and Carrara, 2004.

[12] Here metabolism has to be understood in a particular way: "[…It] refers to the use, and budgeting, of energy for bodily construction and maintenance, as well as for behaviour. Metabolism, in other words, is more than mere material self-organisation […] Metabolism in this […] sense,

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At this point we have another property that necessarily characterizes human identity: to be alive, that is, to have functioning integrated metabolic processes. Let us call L this property. Thus I may state the *metabolic necessary condition for the human identity*:

> *if* $\boldsymbol{B}$ *is the set of human beings and* $\boldsymbol{L}$ *is the set of properties permitting their being alive, that is, the set of certain functioning integrated metabolic processes, then*
>
> $\forall B, B' \in \boldsymbol{B}$ *and* $\forall L, L' \in \boldsymbol{L}, [B \equiv B' \rightarrow L \equiv L']$.

5. Genetic properties and the beginning of the ontogenesis

Around $3 \cdot 10^6$ spermatozoa are ejaculated and almost 200 of them arrive at the oviduct. Only one binds to the zona pellucida of the ovum. After about 24 hours the fertilisation is over: the two pronuclei of the human parents have concluded the fusion process; a zygote is formed, and the ontogenesis begins. This means that something new has appeared; something which has 23 couples of homologous chromosomes in its nucleus; something which has in it around $6 \cdot 10^9$ base pairs of nucleotides making up its DNA double helix. Here, in the DNA, there is most of what a human being was, most of what a human being is, and most of what a human being will be.

Given any two unrelated human beings' genomes, they differ for one base per thousand. This means that of the $6 \cdot 10^9$ base pairs, two human beings share the great part. In this part the phylogenetic story of each human being is contained. Instead in the remaining part, that is, in the $6 \cdot 10^6$ base pairs, its genetic individuality lies.[13]

[13] Of course, note that if two genomes differ at some 1/1000 of their bases, these are not necessarily the same ones for all the genomes. Moreover, beyond the differences in the nucleotide sequences, there are also differences in the number of nucleotides. Another remark is worth making here. Genetic human individuality can be expressed differently. We know that the coding part of the human DNA is around 3÷4% of the

both generates and maintains the distinction between the physical matter of the individual organism and that of other things, whether living or not. Metabolism, in this third sense, necessarily involves closely interlocking biochemical processes" (Boden, 1999, pp. 236-237). Boden (1999) offers a good and sharable argument to sustain that metabolism (in the sense just mentioned) is sufficient, at least *prima facie*, to consider a human being as a living one.

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If our phylogenetic history is contained in ($6{\cdot}10^9$–$6{\cdot}10^6$) base pairs, it means that there, there are also the DNA sequences that characterize us as *Homo sapiens*. That is, there is what marks our species-specificity.[14] Therefore, I may enunciate the *species-specific necessary condition for human identity*:

if $\boldsymbol{B}$ *is the set of human beings and* $\boldsymbol{S}$ *is the set of the species-specific DNA sequences, then*

$\forall B, B' \in \boldsymbol{B}$ *and* $\forall S, S' \in \boldsymbol{S}$, $[B \equiv B' \rightarrow S \equiv S']$.

Once the condition of our belonging to the species *Homo sapiens* has been stated, that is, once our species-specific identity has been defined, we may turn to the genetic identity of each member of such a species.

In the long history of humankind more than one pattern has been proposed to grasp the differences among human beings. We started simply by estimating the more manifest phenotypic features (eyes, hair, skin, height, size, etc.). However with the rise of molecular genetics extremely more sophisticated patterns entered the field: from the differences in the protein sequences, to the differences in the

[14] It is well known that human genome has been sequenced (cf. Lander *et al*., 2001; Craig Venter *et al*., 2001). To actually identify the DNA sequences that are typically human, we should compare the human DNA with the DNA of the closest species, that seem to be *Pan troglodytes* (chimpanzee). Until the chimpanzee's genome has been sequenced, we cannot have the exact amount of the differences. However, it seems that *Homo sapiens* and *Pan troglodytes* share 94÷97% of the genome; cfr. Olson, 2000; Chen and Li, 2001; Marks, 2002. It should be noted that the forensic scientists have extremely sophisticated methods to identify a DNA as a human DNA; cf. Crouse and Schumm, 1995. Here above, I have implicitly talked about nuclear DNA, actually it is also possible to identify a living being as a human living being by resorting to the mitochondrial DNA, cf. Parson *et al*., 2000.

total amount and that there are around $4{\cdot}10^4$ genes. It is supposed that the human average heterozygosity (the two correspondent loci in the two homologous chromosomes have two different alleles of the same gene) is around 6,7%. It means that a human being could be heterozygous in about 2680 loci. It follows that, in principle, it could produce 2^{2680} different germinal cells. If we take into account all of this and the fact that the zygote is given by two different germinal cells, we can say that two different zygotes have an extremely low (but nevertheless different from zero) possibility to possess the same alleles.

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Restriction Fragments Length Polymorphisms (RFLPs),[15] and to the differences of the so-called microsatellite DNA sequences.[16] Now a new pattern appears to be the best one to genetically grasp human individuality: the Single Nucleotide Polymorphism (SNP) based pattern.

Take a chromosome and a site containing a given nucleotide sequence, a SNP is a single nucleotide change in that sequence due to allelic polymorphisms. For example, if the sequence were TTA**G**GCTC, a SNP would be TTA**A**GCTC, where the fourth base G is changed into A.

In other words, given A and B, they have in common at least 99,9% of their genomes and their genetic individuality is given by the remaining 0,1%. Such individuality can be grasped by their SNPs. With some limitation and from the genetic point of view, A is A just because it has its own SNPs. And the same goes for B.

Is it, therefore, sufficient that two human beings have the same SNPs to conclude that they are the same human being? No, for at least two reasons.

The first concerns identical twins: they have the same SNPs, since they have the same genome, but, of course, they are not the same human being. The second has to do with the fact that the supposed number of different SNPs is rather great (approximately $10 \div 30 \cdot 10^6$) but not infinite. It follows that there should be an extremely low, but different from zero, probability that two different human beings have the same SNPs.[17]

[15] In the 1970s, enzymes called *restriction nucleases* were discovered. Each of them has the capacity to cut the DNA sequence at a specific site. Different restriction nucleases cut the DNA at different sites. In this manner DNA fragments can be obtained. Given the same DNA sequence of different individuals, each sequence, as a consequence of the diversity of the nucleotides making it up, will be fragmented in a different way by restriction nucleases of the same kind. On the RFLP, cf. Nathans and Smith, 1975; Danna, 1980; Kessler and Manta, 1990.

[16] Microsatellites are short repeated nucleotide sequences. Suitable chosen microsatellites on the same DNA sequence of two different individuals differ, and therefore the former permits us to distinguish the latter. Microsatellites can be used for the DNA fingerprint in forensic science, as well as the RFLP; cf. McElfresh *et al*., 1993.

[17] By taking into account that in the human genome the SNP frequency is 1 per 1000 base pairs, it means that in the $6 \cdot 10^6$ base pairs that differentiate two individuals there should be about $6 \cdot 10^3$ SNPs. Therefore there should be an extremely low (but different from zero) probability that two different individuals have the same SNPs. With reference to the SNPs, cf. Hartl and Clark, 1997; Collins *et al*., 1998; Przeworski *et al*., 2000; The International SNP Map Working Group, 2001; Kruglyak and Nickerson, 2001.

It follows that we do not have a sufficient condition for human identity but a necessary one, and I may state the *genetic necessary condition for human identity*:[18]

if $\boldsymbol{B}$ *is the set of human beings and* $\boldsymbol{G}$ *is the set of the possible SNPs, then*

$\forall B, B' \in \boldsymbol{B}$ *and* $\forall G, G' \in \boldsymbol{G}$, $[B \equiv B' \rightarrow G \equiv G']$.

6. Immunologic properties

While I was criticising brain transplantation thought-experiments, I recalled the importance of the immunologic aspects as to the question of human individuality is concerned. Now we must discuss them.

Probably the first explicit distinction between what is called the *immunologic Self* and the *immunologic nonSelf*, that is, what marks immunologic identity, was introduced by Frank MacFarlane Burnet in the 1940s.[19] However, with reference to our aims, the best way to grasp why immunology is relevant to human individuality is to start from genetics. Indeed in each *Homo sapiens*' genome there are some genes that encode proteins strictly correlated with its individual immunologic responses. Beyond the interesting fact that it seems that such genes have a common evolutionary history (Hunkapiller and Hood, 1989; Williams and Barclay, 1988), two gene pools are particularly relevant here: the one coding for immunoglobulins and the one, called *Major Histocompatibility Complex* (*MHC*), coding for the Major Histocompatibility Complex (MHC) proteins.[20]

Let us start with the immunoglobulins, or antibodies. These proteins are produced by a particular kind of lymphocytes: the so-

[18] It should be clear that genome (or some of its sequences), if abstractly considered, may be though of as an *individualising necessary feature*. Nevertheless, if it is considered as an actual genome of an actual human being, it must be thought of as an *identifying necessary feature*. In this second way, it is used in forensic science, for example. Of course, the individualising feature and the identifying feature must be taken well separated; only the former interests our analysis.

[19] It was introduced in the second edition of his *The Production of Antibodies*, that he cowrote with E. Fenner; cf. Burnet and Fenner, 1949. On the history of immunology and its philosophical relevance, cf. Tauber, 1991; Tauber, 1997; Tauber and Podolsky, 1997.

[20] Usually the genes are written in italic and the encoded proteins in plain style. Note that in humans, *MHC* is called *Human Leukocyte Antigen* (*HLA*).

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called B cells.[21] It is estimated that there are 10^{15} different kinds of antibodies and each kind is expressed by a specific kind of B cells. This means, on the one hand, that all the B cells expressing the same antibody can be thought of as clones of the B cell that first expressed that antibody. On the other hand, it means that there has to be a particular genetic mechanism capable of producing an enormous number of kinds of antibodies with comparatively few genes.[22]

Let us suppose that an antigen, that is, any substance capable of eliciting an immune response, enters a human body and suppose that it is the first time for that kind of antigen.[23] It happens that it is *de facto* impossible, because of the extremely high number of kinds of antibodies, that the antigen does not encounter its specific antibody. When this happens (we are in the case of the *primary immunologic response*) the antigen, or a part of it—the so-called epitope—, binds to the antibody which is on the surface of the B cell which has expressed it.[24] In this way, the B cell is activated and begins its proliferation and maturation. Such processes involve both the generation of clones—and therefore there will be always more and more B cells expressing the same kind of antibodies—and the secretion in the blood of the expressed antibodies. A part of these clones matures into memory B cells, which live in a sort of stand-by state. If the same kind of antigens, or an antigen with the same epitope, enters, the *secondary immune response* occurs and this is quicker and stronger than the first one. Now the antigen swiftly encounters the specific antibody and the immune response can be activated in a shorter time thanks to the memory B cells ready to express the right antibody.

[21] They are called B cells from *Bursa fabrici*: a bird limphoyd organ. In a human adult, they mature in the bone marrow, while in the foetal stage they mature in the liver.

[22] This is possible since each antibody is composed of two peptidic chains: the heavy chain (there are five classes of heavy chains) and the light chain (there are two classes of light chains). It happens that the two light chains and all the three heavy chains are encoded by three different pools of gene segments, and each segment encodes for a particular part of the corresponding light or heavy chain. The rearrangement of the gene segments and other genetic events, like somatic mutations, allow the incredible number of different antibodies.

[23] That is, let us suppose that it overcomes the other innate non-specific parts of the immune system, such as skin, mucous membrane, serum factors, phagocytic cells.

[24] It is estimated that a non-activated B cell has approximately 10^5 antibody molecules in its plasma membrane.

Two remarks are worth making here. The first concerns the fact that the aforementioned explanation of the B cell response is based on the clonal selection theory proposed by Burnet.[25] The second concerns the fact that each human being, before being exposed to any antigen, has a given repertoire of antibodies. This repertoire and the levels of different antibodies are innate, and therefore they may be thought of as genetically governed properties. Of course they are equal to those possessed by any other human being having the same genome, or at least the same antibody-gene pools, as it happens in the case of twins. However as soon as the B cells encounter the first antigens, the level of the different kinds of antibodies, and B cells as well, begins to change. So, the more the human being develops and grows, the more the B cell-governed response and the level of the antibody repertoire become individual. Since such an individualisation of the B cell-governed response is strictly correlated with the foreign microorganisms encountered by the B cells, for the first time we have come across an aspect of individualisation due to the environment in which the human being lives. That is, human individualisation linked to the B cell-governed immune response is partly due to the genetically governed properties, partly to epigenetically governed properties[26] (and therefore they are innate), and partly to environmentally governed properties due to the interactions with the environment (and therefore they are acquired and adaptative).[27]

Let us turn to the MHC proteins. They are expressed by the *MHC* genes that occupy a region of the sixth chromosome, and are characterised by one of the strongest polymorphisms of our genome. It means that any locus encoding for a MHC protein can be occupied by so many alleles that it has been estimated there are at least 10^{12} different possible MHC proteins. As a consequence, *de facto* any human being has its own MHC proteins. That is, it is

[25] The clonal selection theory is based on the assumption that any human being randomly produces an enormous number of antibodies before being exposed to any antigen. The binding of the antigen to its specific antibody activates the B cell which has expressed it, and such a B cell both proliferates and matures; cf. Burnet, 1959.

[26] I have not spoken yet about the fact that the B cell immune response is not only connected to the encountered antigens but also to the interaction with different cell populations, for example the so called T Helper cells.

[27] Note that the instantiation at a given time of the immunologic phenotype is not so different from any other phenotypic instantiation, at least from the point of view of the relation genotype/phenotype.

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individualised in a precise way by its *MHC*. Of course, there is always both the case of the twins who have the same genome, and *a fortiori* the same *MHC* genes, and the extremely rare case of two different human beings possessing the same *MHC*. However we are allowed to affirm that the MHC proteins give strong individual character to our immunologic response.[28]

There are two types of MHC proteins, the Class I MHC proteins, which are expressed in almost all the human cells, and the Class II MHC proteins, which are expressed in certain B cells, in the macrophages (a kind of white blood cells) and in the so-called antigen-presenting cells.

At this point I should recall that beyond the B cells there is another kind of lymphocytes: the T cells.[29] The T cells, differently from the B cells, neither present antibodies on their surface nor secrete them. Instead their surface has antigen-receptor proteins that recognise foreign antigens. As there are two classes of MHC proteins so there are two classes of T cells: the Cytotoxic T cells, whose antigen receptors recognise the Class I MHC, and the Helper T cells, whose antigen receptors recognise the Class II MHC.[30]

With reference to our aims, what happens in the thymus is extremely relevant. Here the T cells "learn" to distinguish the immunologic Self from the immunologic nonSelf. Indeed it happens that about 90% of the T cells die before maturing. This is a consequence of the fact that in the thymus a *positive selection* of

[28] The *MHC*, due to its great polymorphism, can be used also to reconstruct the phylogenetic history of the species; cf. Klein, 1986; Takahata, 1990.

[29] The T cells are called in this way since they develop in the Thymus.

[30] The T_c cells defend us against microorganisms that are inside our cells. The foreign proteins of such microorganisms are degradated by the host cell and then carried and presented at its surface by the Class I MHC proteins. The T_c cells recognise the complex (foreign peptide)–(Class I MHC) with their antigen receptors, and then their immune response begins by killing the infected cells. The T_H cells help us in defending from antigen in a different way. They recognise, by means of their antigen receptors, the foreign antigen when it is bound to the Class II MHC on the surface of the antigen-presenting cells, which before have ingested the foreign microorganism, degradated it and presented at their surface by means of the Class II MHC proteins. The activated T_H cells do not kill directly the antigen-presenting cells, but both stimulate macrophages to do it and help the right B cells to secrete their antibodies. It should be noted that also the T cell antigen receptors are specific, and therefore also the process concerning the level of different kinds of antigen-receptor T cells is explainable by means of the clonal selection theory.

the T cells happens. That is, only those T cells capable of recognising foreign peptides bound to the self-MHC, survive. This result, called MHC *restriction*, is therefore an acquired property of the immune system. Nevertheless, the T cells "must learn" also another important fact: they must tolerate the self-MHC and the self-peptides, otherwise, as happens in the autoimmune diseases, the consequences would be lethal for human beings. Also this process of *negative selection*, which leads to the acquired immunologic self-tolerance, takes place in the thymus by means of the elimination of the self-reactive T cells.

By taking into account what has been said both about the B cells and the T cells, when a human being is exposed to foreign microorganisms, that human being develops its own individual immunity. This process, which can be artificially induced (for example, by vaccination), or naturally induced (as happens, for example, when a human being is non-voluntarily exposed to infective microorganisms such as viruses or bacteria), leads to the so-called *actively acquired immunity*. Instead if the immunity is transferred to the human being by transferring the specific immune cells and/or antibodies from an immune host to it (as it happens in the transmission of maternal antibodies across the placenta to the foetus, or in the transmission of maternal secretory antibodies to the newborn *via* colostrum and milk), we speak about *passively acquired immunity*.

In both cases the immunity of a given human being and its immunologic individuation are a) grounded in its genome (in particular in the gene pools encoding the proteins related to the immune response), b) epigenetically developed (with reference to the interactions among the different cell populations of the immune system), and c) environmentally fixed by its interaction with foreign microorganisms (as to the particular level of the different kinds of T and B lymphocytes is concerned).

It is relevant to note that on a genetic basis, which is already individualising, a new process of individualisation takes place during the ontogenesis and it persists all through life. Another remarkable aspect concerns the fact that on a genetic basis, the immune system of any human being individualises itself by means of the interaction with the environment in which that human being lives. Therefore, we may speak of *plasticity of the immune system* that, during the ontogenesis and then during the whole life, can be instantiated in a particular way. With regard to the question at issue, we should clearly distinguish the particular manner in which the immunologic plasticity is really instantiated in that particular real

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human being (a topic involving the *identification* of that real human being, as is well-known in the forensic medicine), and the abstract possible instantiations of the immunologic plasticity in an abstract human being (a topic concerning the *individualisation* of that abstract human being *qua* that abstract human being).

Of course we are interested in the individualisation and therefore, I may state the *immunologic necessary condition for human identity*:

> *if* $\boldsymbol{B}$ *is the set of human beings and* $\boldsymbol{I}$ *is the set of the possible instantiations of the immunologic plasticity, given by possible ensembles of MHC proteins and antibodies, then*
>
> $\forall B, B' \in \boldsymbol{B}$ *and* $\forall I, I' \in \boldsymbol{I}, [B \equiv B' \rightarrow I \equiv I']$

7. Brain properties

More or less at the beginning of the third week from fecundation, gastrulation starts. There is a reorganisation of the embryonic cell mass and the formation of three cell layers: the germinal layers. The outer layer, the *ectoderm*, will give rise, beyond the skin covering and the sense organs, to the nervous system.[31] Since that moment on the process which will lead to the cerebral cortex takes place. We may summarize it in a few steps:

1) *Neural induction* that starts with the formation of the neural plate, the future nervous system, from a cell population of the ectoderm. Moreover around the fourth week, neurulation, that is, the development of the neural tube (i.e., the precursor of both the encephalon and the spinal marrow) begins.
2) *Neurogenesis*, that is, the neuron formation and proliferation from a layer of the neural tube. It is worth noting that each neural cell precursor, the neuroblast, gives rise to a finite number of neurons and that after a certain period the neurogenesis decreases drastically.[32]
3) *Neuron migration*. The new neurons are not in their position and therefore they migrate towards their final destination to form the six layers of the cerebral cortex.

[31] The other two layers are the *mesoderm* (the central one) which will give rise to muscles, excretory organs, circulatory organs, sex organs, and skeleton; and the *endoderm* (the inner one) which will give rise to the alimentary canal, the organs associated with digestion and breathing.

[32] It was thought that the neurogenesis stops at a certain moment of the foetal development. Yet it has been found that it continues in certain brain areas all life long, cf. Kempermann *et al*., 1997; Gould *et al*., 1999.

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4) *Neuron programmed death and differentiation*. In order to give form and function to the developing cerebral cortex some neurons die, as happens for many other cells during the ontogenesis.[33] The remaining neurons begin differentiating and maturing. This means that dendrites and axons sprout from each neuron, arborising it. Then the axons begin stretching to arrive at the right target to form the right connection. Once they have arrived at their destination, they form a synapsis with a dendrite of the target neuron.
5) *Synaptogenesis*. The process of the genesis of the synaptic connections among neurons is the most important fact concerning the brain cognitive functionality. It begins around the seventh week after fecundation. It has its peak at approximately 24 months of age, and it stabilizes after about 9 years of age. It continues all through life, of course at a different rate.[34]
6) *Neural plasticity*.[35] Beyond the genetically and epigenetically governed processes of neuron proliferation and production of axons, dendrites and synapses, brain development is strongly characterised by plasticity, that is, by the fact that some synaptic connections are pruned, some are reinforced and some are created. The important aspect is that such *plasticity*, which continues throughout childhood and adulthood,[36] even if at different levels, is environmentally modulated. It seems that such environmentally governed plasticity may be explained by a neo-Darwinian selectionist model integrated with an instructionist model (Cf. Crair, 1999). According to the selectionist model (cf. Changeux *et al*., 1973; Changeux, 1985; Changeux and Dehaene 1989; Edelman, 1987), the environmental selection acts on the overproduced synaptic connections by both reinforcing those which are used and pruning the other ones. Instead, according to the instructionist model, the environmental inputs lead to both the

[33] It is the so called programmed cell death, or apoptosis, which gives rise to the form and function of the embryo and of its organs and parts. On the cellular apoptosis, cf. Ameisen, 1994.

[34] On the brain development, cf. Huttenlocher, 1979 and 1990; O'Leary, 1992; Quartz and Seinowsky, 1997; Rakic, 1988 and 2000; Rakic and Zecevic, 2000; Zecevic, 1998; Marin-Padilla, 1990.

[35] On the historical bases of brain plasticity, cf. Konorski, 1948; Hebb, 1949.

[36] The adult's plasticity is limited and no longer arrives at deep structural changes of the synaptic connections; cf. Singer, 1987 and 1995; Tucker, 1992.

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formation of new synapses (Quartz and Seinowsky, 1997) and the separation of the cortical areas (cf. Neville, 1990; Neville and Lawson, 1980).[37] These two models together seem to well-cohere with two periods of the environmentally governed development of the brain (Greenough and Black, 1992).

a) The *experience-expectant period*, during which particular synaptic connections are expected to be reinforced and stabilized by particular environmental inputs. It is supposed that such particular synaptic connections are the evolutionary outcome of environmental selections connected with species-specific inputs. Therefore the neglect and the failure of such environmental inputs may lead to a permanent deficit in the brain structure, and thus in the cognitive abilities.[38]

b) The *experience-dependent period* during which particular synaptic connections are reinforced or produced in response to unexpected environmental inputs.

At this point, before tackling the role of the synaptic connections and therefore of the cognitive functions, it is necessary to underline the role of the genes during the ontogenesis. Ontogenesis is an extremely complex process of cell proliferation, cell differentiation and cell death. It does not begin thanks to the self-expression of the genes contained in the zygote nucleus, but by means of their expression due to the proteins contained in the zygote's cytoplasm. Since the zygote's cytoplasm is nothing but the maternal ovum's cytoplasm, the early ontogenetic events are governed by the maternal genes that have encoded the zygotic cytoplasm's proteins.[39]

[37] Therefore, even if the first two, three years of the infant's life seem extremely important, they are not important because what it learns in those years is what it can learn, but because what it learns is the basis of what it can learn in the future. That is, it was thought that during the first two, three years the infant's synaptic connections were determined, with small changes, once and for ever. Instead it has been found that the infant's synaptic connections, which have been mostly epigenetically produced, are the basis on which, and from which, life long plasticity starts; cf. Bruer, 1999; Gopnik *et al*., 1997.

[38] It is extremely well known the case of the blocking of the visual input of a cat's eye during the first period of its development. This blocking, even if it is later removed, produced an irreversible change in the visual cortex that led to vision damages. Cf. Wiesel, 1982; Hubel and Wiesel, 1979; Scarr, 1993; Taylor and Taylor, 1979.

[39] Note that other maternal "information" is organised as mRNA. On the misuse of the term "information", cf. Boniolo, 2003.

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Not only are the zygotic cytoplasm's proteins important in themselves, but also the concentration of some of them is relevant. It is such concentration, indeed, that permits the correct embryo's morphogenesis. Of course, at that point also the new nucleus genes move into action. In particular the so-called homeotic genes are important. These contain a 180 base pair long nucleotide sequence (the homeobox) that encodes for 60 amino acids, which concur to form transcriptional factors, that is, proteins controlling the expression of other genes relevant to the development. In such a way these homeogenes control human development, in particular brain development (cf. Boncinelli, 1999; Reichert and Simeone, 1999).

It must be pointed out that the ontogenetic process, even if genetically based on the aforementioned developmental genes, is not a pure succession of genetic events. Instead it must be considered both as a process in which first the genes of a given nucleus and the biochemical elements of its cytoplasm interact, and, then, as a process in which a given cell expresses some of its genes thanks to the interaction with surrounding cell populations. That is, the ontogenetic process is partly a genetic process, partly an epigenetic process.[40]

So far I have briefly sketched how brain development occurs by means of genetically, epigenetically, and environmentally governed processes. It should be already clear that these processes lead to the formation of a brain structure unique for each individual. Nevertheless another step is worth making, and it deals with one of the functions of such a brain structure: memory.

Since Augustine, a lot of philosophers have considered memory as one of the main, if not the unique, features of human identity, even personal identity. But very few of them have inquired about what memory is from a neurobiological point of view. This will be exactly our next step.

With a motto, we may affirm that *memory is nothing but synaptic connections*. We have said that each human being's synaptic connections are the result of genetically, epigenetically, and environmentally governed series of processes that are unique for each human being. Therefore each human being's memory is the result of genetically, epigenetically, and environmentally governed series of processes that are unique for each human being.[41]

[40] On the genetic and epigenetic bases of ontogenesis, cf. Le Douarin, 2000; Minelli, 2003. On the genetic and epigenetic bases of brain development, cf. Schlaggar and O'Leary, 1991; Chan and Jan, 1999.

[41] From this point of view, the position of those stating that we are our synaptic connections is understandable (cf. Le Doux, 2002), if not totally sharable since it takes into account only one of the individualising features.

As far as we know, thanks to Brenda Milner's seminal works in the 1960s, human memory is thought of as a set of mnestic systems (cf. Cohen and Squire, 1980; Squire, 1987; Sherry and Schacter, 1987):

1) *implicit memories* (or *non-declarative memories*), connected with motor, visual, and cognitive capacities which may be activated unconsciously. There are three subsystems: a) *procedural learning memory* (tentatively located in the striatum and in specific cortical areas); it has to do with the knowledge acquisition of the structural properties of the relations among objects and among events; b) *conditioning paradigm memory* (tentatively located in the cerebellum); it is connected with the conditional learning that allows the individual to anticipate an event by detecting its precursor signal; c) *priming paradigm memory* (tentatively located in specific cortical areas); it concerns the detection, and the improvement of the detection, of an object recently experienced.
2) e*xplicit memories* (or *declarative memories*) are the memories you can declare since they refer to something that can be brought to mind, that is, something we are conscious of. They may be subdivided into: a) *pre-explicit memory* (tentatively situated in the hippocampus); it is related to the novelty detection; b) *semantic memory* (tentatively located in particular cortical areas); it is connected with what we can know without having direct experience; c) *episodic memory* (tentatively placed in certain cortical areas); it concerns the knowledge of what is directly experienced.

All the processes leading to the development of the different memory systems are not well understood yet (cf. Nelson, 1995, 1997 and 2000). Nevertheless it seems quite clear that these processes are epigenetically and environmentally governed, and that they can be thought of as mechanisms concerning the acquisition, the retention, and the retrieval of knowledge. Moreover, thanks to Eric Kandel (cf. Kandel and Spencer, 1968; Kandel, 1976), we know that these mechanisms are strictly connected with changes in the synaptic connections. Therefore they can be studied on genetic bases (cf. Alberini, 1999; Mayford and Kandel, 1999; Laroche, 2000). Indeed, both explicit and implicit memories are long-term memories, that is, memories whose acquisition, retention, and retrieval mechanisms are due to changes in the number and in the organization of the synaptic connections by means of the activation of certain genes, and therefore by means of the encoded proteins.[42]

[42] Also the short-term memory concerns synaptic modifications; however such modifications have to do with the synaptic activity thanks to modification of already existing proteins.

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Some interesting conclusions may be drawn from what has been said above. First of all during the epigenetically governed part of the brain development it seems that the so-called species-specific mnestic mechanisms are located in the deeper cortical areas (cf. Fuster, 1995 and 1997). They should concern the experience-expectant mechanisms and, it seems, the implicit memory (Reber, 1992). Secondly, I have outlined that the memory systems are the complex outcome of our phylogenesis and ontogenesis. Moreover each different system seems to be located in a different part of our brain, and therefore to have a different developmental history. It is supposed that the thalamus and the hippocampus begin differentiating around the end of the fifth week from fecundation; the cerebellum starts developing around the end of the sixth week from fecundation; and the first synapses form approximately in the seventh week from fecundation. If this holds and if the brain location of memory systems holds, a human being can begin developing its own (both implicit and explicit) memory only after a certain time from fecundation and thus before its birth.[43] Finally, all the mnestic mechanisms are synaptically based and those concerning long-term memory have also a genetic substrate (even if, of course, they are not genetically determined, but epigenetically and environmentally governed).

If long-term memory is a particular stable synaptic organisation, therefore anytime we intervene to retrieve memories, our mnestic action is performed by a brain different from the brain that stored them. Anytime we reorganise our memories, we change the synaptic connections, also by creating new ones, as, unfortunately, it sometimes occurs when we are the object of bad psychotherapy (cf. Loftus and Ketcham, 1994; Kassin and Kiechel, 1996; Rubin, 1996; David, 1996).[44]

By taking all this into account, it is extremely hard to understand what the real bases of the brain transplantation thought-experiments are. Actually, scientifically, and not science fictionally, we may state that each human being has its own way in which its species-specific neural plasticity is instantiated all through its life by means of individual epigenetic interactions and individual environmental inputs grounded in an individual genome.

[43] Of course to control the actual foetus' mental contents is extremely difficult. However, it has been found that there are memories in the newborn due to input during the foetal period.

[44] It is nice to recall that also Leibniz discussed the topic of false memories, cf. Leibniz, 1765, Book II, Ch. XXVII, § 9.

Therefore, I can state the *neural plasticity necessary condition for human identity*:

if ***B*** *is the set of human beings and* ***N*** *is the set of possible instantiations of the neural plasticity, then*

$\forall B, B' \in \boldsymbol{B}$ *and* $\forall N, N' \in \boldsymbol{N}$, $[B \equiv B' \rightarrow N \equiv N']$.

8. Human identity

At this point we may put together what has been found. We have seen that any human being is necessarily characterised a) by a partially individualizing worldline W describing its position in space and time during all its life (and not only); b) by a partially individualizing integrate metabolism L; c) by species-specific nucleotide sequences S; d) by partially individualizing nucleotide sequences G; e) by a partially individualizing instantiation of its immunologic plasticity I; and f) by a partially individualizing instantiation of its neural plasticity N. That is, given two human beings B and B', we have

$\forall$B, B′$\in$**B** and $\forall$W, W′ $\in$**W**, [B$\equiv$B′ $\rightarrow$ W$\equiv$W′],
$\forall$B, B′$\in$**B** and $\forall$L, L′$\in$**L**, [B$\equiv$B′ $\rightarrow$ L$\equiv$L′],
$\forall$B, B′$\in$**B** and $\forall$S, S′$\in$**S**, [B$\equiv$B′ $\rightarrow$ S$\equiv$S′],
$\forall$B, B′$\in$**B** and $\forall$G, G′$\in$**G**, [B$\equiv$B′ $\rightarrow$ G$\equiv$G′],
$\forall$B, B′$\in$**B** and $\forall$I, I′$\in$**I**, [B$\equiv$B′ $\rightarrow$ I$\equiv$I′],
$\forall$B, B′$\in$**B** and $\forall$N, N′$\in$**N**, [B$\equiv$B′ $\rightarrow$ N$\equiv$N′].

My proposal about human identity concerns exactly the above mentioned six necessary conditions. That is, I am suggesting that all of them make up a sufficient condition for human identity. Thus

$\forall B, B' \in \boldsymbol{B}$, $\forall W, W' \in \boldsymbol{W}$, $\forall L, L' \in \boldsymbol{L}$, $\forall S, S' \in \boldsymbol{S}$, $\forall G, G' \in \boldsymbol{G}$, $\forall I, I' \in \boldsymbol{I}$, $\forall N, N' \in \boldsymbol{N}$,
$[(W \equiv W' \,\&\, L \equiv L' \,\&\, S \equiv S' \,\&\, G \equiv G' \,\&\, I \equiv I' \,\&\, N \equiv N') \rightarrow B \equiv B']$.[45]

Therefore, let us come back to the first initial questions:

[45] With reference to this statement, it should be noted that each element of the 6-pla is not totally independent from the other ones; for example, both the immunologic and the neural element are connected to the genetic element, and there is a connection between the immunologic element and the neural one. Not to consider all the 6 elements in their complex relations would be a mistake and it is just for the sake of exposition that I have considered them one by one.

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"Given two human beings, B and B′, which is the set of the sufficient properties $<P^B_i>$ of B and $<P^{B'}_i>$ of B′, such that, at a certain time t, if $<P^B_i> \equiv <P^{B'}_i>$ then $B \equiv B'$?".

Now I would suggest answering as follows:

"Given two human beings, B and B', and given, at a certain time t, two sets <W, L, S, G, I, N> of properties of B and <W′, L′, S′, G′, I′, N′> of properties of B′, it is sufficient that <W, L, S, G, I, N>≡<W′, L′, S′, G′, I′, N′> so that B≡B′".

Now, we must come to the time dependent question:

"Given a human being B, which is the set of the sufficient properties $<P^B_i>_t$ and $<P^B_i>_{t'}$, where t′>t, such that if $<P^B_i>_t \equiv <P^B_i>_{t'}$ then $B_t \equiv B_{t'}$?"

Here we should pay attention because among the elements of the set <W, L, S, G, I, N> there are properties which are time dependent and properties which are time independent. Of course we are looking for something which is time invariant, something which persists during human beings' life. Let us consider the elements of the just mentioned set one by one:

1) W is the property of having a possible worldline, which, as said, has to be continuous and without time loops. Let us call 3_t the 3-dimensional space section of W at t. If at t B occupies 3_t and at t′ B occupies $3_{t'}$, and if between 3_t and $3_{t'}$ there is no solution of continuity, then at t and t′ B occupies two different space sections of the same worldline W.
2) L is the property of possessing certain functioning integrated metabolic processes; that is, to be alive. This is a time independent property.
3) S is the property of having a given species-specific DNA sequence. This is a time independent property.
4) G is the property of having a given SNP. Also this is a time independent property.
5) I is the property of having a particular instantiation of the immunologic plasticity. This is a time dependent property, and it is useless for our aims. Actually we have a time invariant, that is, immunologic plasticity *qua* immunologic plasticity. Let us call it P^I.
6) N is the property of having a particular instantiation of the neural plasticity. Also this is a time dependent property, and it is useless for our aims. However we have a time invariant, that is, neural plasticity *qua* neural plasticity. Let us call it P^N.

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At this point I can answer the last question:

> *"Given a living (that is, possessing the property L) human being B at a time t and at a time t', so that* $B_t \equiv B_{t'}$ *it suffices that* $<S, G, P^I, P^N>_t \equiv <S, G, P^I, P^N>_{t'}$*, of course if* B_t *and* $B_{t'}$ *occupy different space sections of the same worldline."*

Some may object I have not argued that the six necessary conditions truly make up a sufficient condition. If we were looking for an *a priori* justification, the objection would be sound. But if we wish to follow Locke's indication for "Modesty of Philosophy" and therefore "not to pronounce Magisterially, where we want […] Evidence", I believe that we have not many other possibilities. This is what biological evidence teaches us, and we must be contented. However, we may pour the argument on the critics and ask them: "Please, find a case showing that the six necessary conditions together do not make up a sufficient condition".

9. Some philosophical consequences

From what has been said, we may draw some interesting philosophical remarks. First of all, we may inquire if it is really sound to claim that we are an animal plus a person, as many philosophers suggest (cf. Shoemaker, 1984). *We know that to define what a person is, is not so easy and unproblematic*: 'person' is one of the many philosophical terms which have a really long history of different definitions. However, for the sake of discussion let us assume that a person is identifiable by its mental contents, even though *we do not exactly know even what the mental contents are*.

That we are animals is sure, and, beyond some religious fundamentalist, nobody questions this statement. Actually, to claim that we are animals is not totally correct. It would be more correct to state that we, *Homo sapiens*, belong to genus *Homo*, family Hominidae, order Primates, class Mammalia, phylum Chordata, kingdom Animalia. If we like to speak in terms of kingdoms, we are animals. However, is to affirm that we are animals plus a person right? From what has been said and from what a phylogenetic and an ontogenetic analysis teaches us, that is, from the evolutionary and the developmental point of views (i.e., from the so called *evo-devo point of view*; cf. Minelli, 2003), this is simply false. We have the mental contents we have, also because we have the brain we have. And we have the brain we have also because we had both the phylogenesis we had and the ontogenesis we had. This is what

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biology teaches us and it is incorrect and misleading to forget it, especially if we believe that the (neo)Darwinian point of view has to be taken seriously into account (cf. Ruse, 1998).

It was our phylogenetic history that accidentally brought us to have a particular neural phenotype that allows us to have particular mental contents. If we like to state that we are persons just because we have mental contents nothing hinders. But 'person' is simply a name that we, *Homo sapiens* (better, a particular subset of *Homo sapiens* called 'philosophers'), give to a specific property that we may possess thanks to our evo-devo history.

Some people may affirm, by resorting to Wiggins' distinction between substantial sortal concepts and phased sortal concept, that 'person' is a phased sortal while 'human being' is a substantial concept.[46] Here, it is worth recalling Wiggins' objection according to which "all phased concepts are either latently or manifestly restrictions of underlying more general sortal concepts" (fn. 3, p. 63). *Prima facie*, I might agree that 'person' is a phased sortal which may be thought of as a restriction of a more fundamental sortal, that of '*Homo sapiens*'. But there are two problems.

The first one concerns the fact that not only may 'person' be thought of as an emerging property of *Homo sapiens*, but also that not each philosopher agrees that any *Homo sapiens* is a person. Actually I would prefer not to speak in terms of emerging properties. If 'person' is defined by resorting to mental contents and these have to do with the memory systems, I would prefer to affirm that it is the result of epigenetically and environmentally governed properties. However, this is not so relevant, it is just a matter of jargon, and evidently I have a preference for a jargon strictly linked with the biological terminology.

Let us come to the real question: "Is each *Homo sapiens* a person"? Of course this problem presupposes that we define a person by its mental contents.[47] But it can happen that *Homo sapiens* grows without having the right neural phenotype to possess mental contents, or that *Homo sapiens*, due to accidents, loses the right neural phenotype to continue having mental contents.

Let us analyse the first possibility by considering the case of

[46] For example, Olson (1997) seems to support this position; cf. Wiggins, 2001.

[47] It is worth noting that here we should indicate which set of mental contents we are considering: only the explicit ones, only the implicit ones, both of them?

anencephalic infants.[48] Even if anencephaly is a time-specific disease,[49] it is not a stimulus-specific one. This means that there could be more than one cause: genetic, infective, metabolic, chemical, radioactive noxae are possible. Let us dwell on the genetic cause. In this situation we cannot say anything but the anencephalic infant is a particular *Homo sapiens* with a particular genome that permits it to be anencephalic (or, if you like, that does not permit it to be non-anencephalic). *If we consider many diseases from an evolutionary point of view, their social interpretation changes enormously*. Therefore, if we accept this point of view, we may affirm that, due to human polymorphisms, anencephaly is one of the many possible phenotypic traits. Of course, we cannot say, from the biological point of view, that an anencephalic infant is less human than a non-anencephalic one. Both are humans, they have only different phenotypic traits, which derive also from an allelic difference between them.

The same conclusion may be reached by considering the non-genetic causes of anencephaly. It is sufficient to take into account what has been said about the fact that our neural structure is the result of both epigenetically and environmentally governed properties. From a certain point of view, we are not wrong in saying that anencephaly and non-anencephaly are two different ways in which neural plasticity can phenotipically reveal itself.

Let us now consider the loss of mental contents. This is a consequence of a loss of the neural phenotypic traits permitting us to have mental contents. In this case, must a human being without the latter be considered no longer as a person? If you like. As said 'person' is simply a word constructed by certain humans to refer to a particular (even if not well-defined) mental status connected with a particular (even if not well-identified) neural plasticity. However, this is not extremely relevant from a biological point of view; a

[48] Anencephaly is a malformation concerning the absence of brain, skull, and overlying scalp. It is thought to result from a failure of the anterior neuropore to close (in normal case, it should close after 25 days from the fecundation). Because these infants lack a functioning cerebral cortex, they are permanently unconscious. Brainstem function is present in varying degree, yet they exhibit many behaviours indicating their brainstem origin: responses to painful stimuli, feeding reflexes (rooting, sucking, swallowing,) respiratory reflexes, interactions involving eye movements, some facial expressions.

[49] It may arise between the XVI day from fecundation, that is, after the beginning of the development of the neural fold, and the XXV day, that is, before the closure of the anterior neuropore.

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human being is always a human being, it always belongs to the species *Homo sapiens*. This is all we can state with extreme precision, in a non-ambiguous and empirically grounded way.

Note that what has just been discussed at the neural level is strictly analogous to what happens at the immune system level, that is, at the level of another constituent of human identity. There are situations in which our genome expresses the bases for "different" immune systems. Let us consider the case of the Severe Combined Immune Deficiency (SCID) (cf. Villa *et al*., 2001). In this case we have a family of diseases characterised by the fact that certain mutant genes prevent the usual lymphocyte differentiation, and therefore they prevent the usual functioning of the immune system. Also in this case we cannot affirm that a human suffering from one of the SCID diseases is less human than one who does not. Both are humans, they differ only in their genome in the sense that they have two different alleles of the same gene and one allele permits the "correct" lymphocyte differentiation, while the other does not.

Analogously to the loss of certain mental contents due to the loss of certain neural traits, there is the possibility of losing the usual functioning of the immune system due to accidents, as happens in the case of Acquired Immunodeficiency Syndrome (AIDS).

Therefore, we can conclude that both immunologic and neural plasticity can be instantiated in many ways, due to genetic, epigenetic, and environmental situations. The particular way in which instantiation occurs concerns the identification of that particular real *Homo sapiens*, but the plasticity *qua* plasticity concerns its individuation, that is, what is relevant for us. The way in which the identification occurs is irrelevant as to human identity is concerned. It is irrelevant that there is a particular neural phenotype permitting or not permitting mental contents. It is irrelevant that there is a particular immunologic phenotype permitting or not permitting the usual defence from antibodies. Of course it is irrelevant with reference to the problem of human identity, and not as to health care! We can acquire or lose some identifying properties linked with a specific real instantiation of immunologic and neural plasticity, but this has nothing to do with our individualising properties given by our spacetime history, our genome, and our immunologic and neural plasticity.[50]

The second problem arising from the idea that 'person' is a restriction of a more fundamental sortal concerns the fact that we

[50] This approach has relevant ethical consequences that here I cannot discuss.

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should stop considering all the human properties simply as human properties. What Charles Darwin and his followers taught us and continue teaching us is that any biological property has to be seen in the light of evolution. In our case this means that what appears to be strongly ontologically grounded as human property actually is a result of at least $3{,}5 \cdot 10^9$ years long evolutionary history which has led to that property, but that could have led to a different property. From this point of view not only does each human phased sortal appears to be a restriction of a human substantial sortal, but in turn each human substantial sortal appears to be a biological phased sortal. That is, 'person' may be considered as a restriction of a more fundamental sortal concept: that one of '*Homo sapiens*'. '*Homo sapiens*' may be considered as a restriction of a more fundamental sortal concept: that one of 'Animalia'. 'Animalia' of 'living being'. 'Living being' of 'organised system of cells'. Eventually 'organised system of cells' of 'organised system of molecules and atoms'. In this way we arrive at $3{,}5 \cdot 10^9$ years ago and, from a biological point of view, nothing more can be seriously claimed.

This simple evolutionary consideration should suggest to us either to abandon any possibility of speaking in terms of human substantial properties, or to interpret "substantial" not in an Aristotelian way but in a purely evolutionary way, as something which characterises a given species, in particular, *Homo sapiens*, but which, at the same time, has to be considered as an accidental evolutionary outcome.[51]

10. Conclusion

The position on human identity just proposed is evidently non-Aristotelian both from the ontological and the metaphysical point of view. Yet this is what we may safely and modestly state, if we want to take biology seriously. However it should be noted that Aristotle gave great importance to coeval biology. Why cannot we do the same? Why, in discussing human identity, cannot we be Aristotelian in this way and therefore forget the brain transplantation thought-experiments, or, more general, science fiction?

[51] Here general ontological and epistemological remarks related to the topic of essentialism can be drawn. In particular it seems clear that it is not so easy to be an essentialist if the evolutionary theory is taken seriously.

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