On the Biology of Learning

I. Introduction.

From a common-sense point of view, there would seem to be nothing very mysterious about learning. After all, each of us once learned to walk, and a bit later to talk; and although it was so long ago that few of us can remember exactly how we did it, there can't have been much to it. As they say, a child of *two* could do that! And now that we are grown up, we learn from our mistakes (or not, as the case may be) without really noticing what we are doing. It is only when we fall foul of a competitive educational institution that learning actually becomes a problem - a point to which we shall return at the end of this text. For the most part, learning is something that happens quite naturally, by itself as it were, and understandably enough we tend to take it for granted.

One of the qualities of science at its best is that it can prompt us to realize that things are far more wonderful than we usually imagine. Einstein once said, tongue only half in cheek, that his discoveries concerning relativity were largely due to the fact that he was.... mentally retarded! - so that questions of space and time that most children have normally understood by the age of four or five remained profoundly mysterious to him until an age when he had acquired the equipment of mathematical physics. The aim of the present text, in addressing the question of learning from a rather elementary biological point of view, is to suggest that it is a process which is actually rather more mysterious and indeed problematical than we generally think.

II. Unicellular and multicellular organisms

The first point we wish to make is that learning is a property of multicellular organisms. An individual unicellular organism such as a bacterium cannot learn. Let us take the example of resistance to antibiotics. Superficially, it looks as though bacteria can "learn", because since the widespread use of antibiotics in human and veterinary medicine over the last decades, antibiotic-resistant strains of bacteria have become prevalent (dangerously so in hospitals). However, careful analysis shows that the mechanism involved is not individual learning, but natural selection at the level of the population. A few antibiotic-resistant bacteria existed already *prior* to the advent of antibiotics (at that point, they were rare mutants); in the presence of antibiotics, the "normal" (i.e. sensitive) bacteria were unable to reproduce and to compete for resources, so the resistant bacteria flourished and automatically came to dominate the population.

Before moving on to multicellular organisms, we may note that when a unicellular organism grows and divides, as a general rule the two "daughter" cells are identical to the previous "parent" cell. This has several important consequences. Firstly, it means that although no living organism can be unconditionally guaranteed of immortality, all unicellular organisms are *potentially* immortal (growing and dividing in two is not the same as dying). This is graphically illustrated by the fact that each unicellular organism alive today is, in a certain sense, the "same" individual as its original ancestor over at the beginning of Life on the planet Earth. A second important consequence is that, if unicellular individuals were able to learn, the benefits would generally be passed on to their progeny, and we would have an example of the inheritance of acquired characteristics. According to conventional neo-Darwinian theory, this does not happen.

Learning, then, occurs only in multicellular organisms; and from a biological point of view, this means that it must be situated in the context of *ontogeny*. Ontogeny is the process, intrinsic to all multicellular organisms, which leads from the fertilization of an egg-cell, through the formation and growth of the embryo to birth, then through post-natal development to maturity and sexual reproduction, and then (if "accidental" death has not

occurred before[1]) through ageing and senescence to an inevitable death. Thus, unlike unicellular organisms, all multicellular organisms have a life-cycle which is characteristic of the species. Because of ontogeny, multicellular organisms are *individuals* in a sense in which clones of unicellular organisms are not.

III. Ontogeny.

Ontogeny, then, is a succession of stages which is "historical" in the sense that each step sets the scene for the next step, which in turn sets the conditions for the next, and so on. However, unlike human history or biological evolution on the grand scale, which are open-ended, ontogeny is a basically repetitive process: each individual goes through a very precise pattern of developmental stages which is essentially the same for all normal members of the species. Indeed, the most striking feature of ontogeny is perhaps its robust regularity. In humans, "monstrous" births in which ontogeny has gone seriously awry are rare, making up less than 1% of all births[2]. Given the highly intricate nature of ontogeny - passing from a single cell to a complex multicellular organism composed of many milliards of cells in the space of months - the success-rate of better than 99% borders on the miraculous. Teleologically, of course, ontogeny has to be reliable, otherwise multicellular forms of life would not be viable; but in science, teleology is no substitute for efficient causation, and developmental regularity calls for an operational explanation in terms of underlying mechanism. At the present time, it must be admitted that we have very little understanding of the exact nature of the underlying mechanisms[3]. However, there are many general indications that ontogeny is indeed a dynamically self-regulatory process; one of the most striking is the fact that if a developing embryo is carefully separated into two, the result is not a left-sided plus a right-sided monster, but a pair of perfectly-formed identical twins.

Biological processes occur on several different time-scales. All living organisms are "autopoietic" (Maturana & Varela 1987); in other words, their most basic, permanent activity consists precisely in *producing themselves* as distinct material entities. This basic activity is metabolic, and takes place on a physiological time-scale of seconds, minutes, hours. In order to be robustly viable, an autopoietic process must necessarily[4] maintain its own organisation invariant; and so living organisms are intrinsically conservative. However, over a very long period of time, living forms do change; this is the process we call biological evolution, or phylogeny, and its time-scale is of the order of millions of years (MYr).

The origin of macroscopic multicellular organisms - both animals and plants - was itself a major evolutionary event, which occurred with dramatic suddenness around 600 MYr ago[5]. Ontogeny occurs on a time-scale of weeks, months and years. But the origin of

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[1] In civilized human society, we call death in bed from old age a "natural" death. From a biological point of view, nothing is less natural than such a death: in all natural populations of animals and plants, the "accidental" death-rate is such that very few individuals attain the chronological age of senility. Thus, the life-expectancy of a sparrow is about 6 months - independently of its chronological age! In captivity, sparrows live to age ten or more, but in the wild less than one in a thousand reach this age.

[2] The figures are comparable for all multicellular species under normal conditions. The low failure-rate at birth is partly due to the fact that serious errors in the first days of ontogeny - which occur much more frequently - lead to precocious abortion and are thus almost undetectable. However, this is in itself a remarkable adaptation, and hardly detracts from the overall performance.

[3] It is widely held that developmental regularity results from the "genetic programme" of the species. I shall argue below that this is a vacuous misconception which hinders far more than it helps the search for a scientific solution.

[4] Here also, teleology must not substitute for operational explanation.

[5] It would take us too far afield to give a proper account of this fascinating event. Key aspects are addressed by Buss (1987), Gould () and Reichholf (1993).

ontogeny not only introduced a third time-scale, intermediate between those of physiology and of evolution; it changed the nature and the pace of evolution itself, for reasons which we must now examine.

IV. Evolution

According to the Darwinian theory, evolution results from the combination of variation and selection. It follows, logically, that selection can only operate on the variation that is there in the first place; and hence that the course of evolution will depend, crucially, on the variation that can arise. In the currently orthodox neo-Darwinian version of the theory, the variation is supposed to be "random", so that the course of evolution is determined by natural selection. However, the concept of "randomness" is tricky; events cannot be "random" in any absolute sense, only relative to the background of a given set of possibilities. For example, when we toss a coin, what is random is whether it will come down "heads" or "tails"; if there is no cheating, the probabilities of these two events will be equal at 50%. But this randomness occurs on the background of the explicit list of all the possible events: the fact that if we toss a coin, it will come down either "heads" or "tails"[1]. Now in the case of biological evolution, the variation which arises, and which selection can act upon, can only be considered "random" from a narrowly gene-centred point of view. Mutations in the nucleotide sequence of DNA are, indeed, quasi-random. But from an organism-centred point of view, the variation which *can* arise is anything but random: it is completely conditioned by the initial form of the organism itself. The contrast between the two points of view can be highlighted by a somewhat facetious caricature. Why are there no cows which jump over the moon? On the neo-Darwinian view (variation is "random"), such mutant cows do occur from time to time, but since the cost-benefit result of the operation is not very favorable, such cows are selected against. The organism-centred point of view is that this particular variant just cannot occur, and therefore the question of the cost-benefit analysis simply does not arise.

More seriously - and more to the point of this article - the great significance of multicellular organisms with an ontogeny is that they can vary not just in terms of cell physiology, but in terms of the developmental process. As a graphic illustration of the fantastic variation that can be produced in this way, we can simply consider the incredible variety of life-forms and life-strategies of multicellular animals, plants and fungi that exist today - from mice to oak trees, elephants to crabs to daffodils, bats and whales and eagles and sparrows to bees and spiders, from snails and worms and squids and sea-urchins to corals and toadstools[2]. Compared to this, unicellular organisms are inevitably monotonous. Basically, the only thing they do is to feed themselves so as to maintain their own metabolism and cellular autopoiesis. This is, of course, already a tremendous achievement, and we are still far from understanding scientifically how it is possible; but the hard fact remains that there are not very many different ways of doing it, and so variation in the life-forms and life-strategies of single-celled organisms is inevitably very restricted. With the advent of multicellular organisms, the rate and indeed the very nature of biological evolution changed dramatically[3].

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[1] In principle there are other possibilities - for example, the coin might land on an edge and stay that way, or it might roll away and get lost, or it might not come down at all if someone catches it and walks away. In this case, non-zero probabilities will be assigned to these additional possible events. But this only serves to strengthen the general point: to say that an event is "random" presupposes that we have a complete list of all possible events.

[2] All this variation is of course only the tiny subset of possible variation which has (a) actually arisen, and (b) been selected; but this only serves to strengthen the general point.

[3] The dramatic suddenness of the appearance of a whole range of multicellular animals in the "Cambrian explosion" (Gould 1991) is itself an illustration of this.

V. Developmental constraints and possibilities.

Ontogeny, then, is tremendously enabling: it opens up whole new worlds of possibilities. But *at the very same time* it is immensely constraining; and it is most important to understand the reasons for this. We have already noted that the only forms that can arise to be selected are those that result from a modification of the developmental process. And since evolution is a historical process which proceeds one step at a time, the only modifications that can be incorporated in a revised developmental process are local, elementary perturbations of the previous process. Now we have also already noted that the ontogenetical process is necessarily a robustly self-regulated dynamical *system.* More precisely, it is a highly non-linear dynamic system; and as Saunders (1989) has pointed out, such systems have the quite generic property possessing a number of distinct "attractors". This conceptual framework leads us to expect what is indeed observed experimentally: many perturbations of the developmental process have virtually no effect, because the dynamical regulation compensates for them; but certain perturbations can have major and apparently multiple effects[1]. It is most revealing to note that the effects depend not on the nature of the perturbation, but on the precise developmental stage at which the perturbation occurs. A classical example in *Drosophila* concerns an abnormal form with a double thorax (and hence an extra pair of wings) appropriately known as "bithorax". This abnormality can be produced either by a thermal shock, or a chemical shock with ether vapour, or even a mechanical shock with a pin-prick - on the sole condition that the shock be administered in a precise time-window during development of the pupa. The same anomaly can also be produced by a genetic mutation - which presumably produces its effect at the same point in time. This phenomenon - the *same* anomaly being produced indifferently by a genetic mutation or by an environmental perturbation - is known as "phenocopy". In the 1940's, the geneticist Goldschmidt produced phenocopies of all the genetic mutants known in *Drosophila* at that time. We may summarize all these phenomena by saying that the course of development follows a definite *epigenetic landscape* which determines the possible outcomes of the process. This "landscape" is a property of the developmental system as a whole; it determines, in particular, the nature of the variation which can be produced, largely independently of whether the cause of the variation is genetic or environmental.

It is a characteristic weakness of neo-Darwinian biology that it talks of "genes 'for' this or that character" in an abstract way, as though any "character" whatsoever could be "produced" with the "right" gene. This easily leads to nonsense and just-so stories with no basis whatsoever in biological facts. Of course, on condition that all other things are equal, *certain* phenotypic differences can be caused by genetic differences. But a genetic difference can only cause a phenotypic difference that can itself result from a modification of the developmental process; and if the phenotypic difference in question can be produced, as a general rule it will also be possible to produce it by an environmental modification. As a result of the form of the epigenetic landscape, certain "characters" are malleable; such characters will usually be affected both by genetic and by environmental causes of variation, and so their "heritability" will be intermediate. Other "characters" are

[1] An interesting example occurs in the development of flat-fish, such as sole and plaice, which as adults have an asymmetrical body-plan: the eyes, mouth and other parts of the head are all on the "upper" side. Superficially, this looks like a problem for evolution by single steps: all these changes have to occur at the same time, since a form in which only the eyes but not the mouth (or vice versa) were on one side would be obviously unviable. The answer comes from the dynamics of development: the newly hatched fish swin upright and are perfectly symmetical like normal fish; then, *quite suddenly,* in the space of a few hours, the developing fish "flips over" and *all* the head organs are placed on the same side. Thus, contrary to appearances, these apparently unrelated changes are the consequences of a *single* developmental modification.

highly canalized; in this case, neither genetic nor environmental differences will be able to affect them, and so their heritability will be neither high nor low but indeterminate.

To sum up: ontogeny is both enabling and constraining. The possibilities, and the limitations, can be explored empirically; in order to understand them scientifically, there is no substitute for an explanation of the epignetic landscape as the property of a dynamic system[1].

VI. Learning: enabled and constrained

This, then, is the backdrop: "learning" is a phenomenon which takes place in the context of a developing multi-cellular organism, at a particular point in its life-history. It follows, from all that we have said so far, that "learning" can only be a modification of the developmental process; and this means that what *can* be "learned" is both *enabled and constrained* by the epigenetic landscape. In order to fully understand the implications of this, it is best to take things one at a time. We shall therefore examine, successively, the fact that learning is enabled by ontogeny; the fact that it is constrained by ontogeny; and finally, the very particular and sometimes apparently paradoxical features that stem from the fact that learning is simultaneously enabled *and* constrained.

Firstly then: learning is enabled by the epigenetic landscape. At any particular point in its ontogeny, there is a very wide range of things that the multicellular organism can *become* over the next stage of its development. In fact, the "envelope" of possibilities is open-ended; there is no pre-given limit to the number of possibilities. Of course, for those possibilities that have never been realized before, we will tend to speak of a "discovery" rather than something that we have "learned". But this distinction is, in a sense, purely contingent; in principle, anything that can be discovered lies within the envelope of developmental possibilities, and can therefore subsequently be learned. And in the same "enabling" vein, anything that can be learned can be learned rather easily: it is sufficient to put the developing organism in the particular conditions that trigger the particular developmental path in question[2]. Development - and therefore learning - is essentially an endogenously self-generating process, and it is therefore unnecessary to "instruct" it from the outside. Common-sense is not completely wrong when it has the impression that "learning" is something which "just happens".

At the same time, learning is *also* constrained by the epigenetic landscape. At any given developmental stage, the number of things that *can* be learned at that stage in that species is severely restricted[3]. And among the things that can be learned, many that can easily be learned at the appropriate developmental stage can only be learned with difficulty - or sometimes not at all - at a later stage, as many of us who have struggled to acquire a second or third language in adult life know to our cost. This is, in fact, a rather generic characteristic of developmental processes. It happens very frequently - and it is actually quite easy to understand - that the consequences of modifying the process at a particular stage can *only* be produced by modifying the process *at* that stage.

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[1] In particular, it is widely held that the robust regularity of ontogeny is "explained" by the existence of a "genetic programme". However, as Oyama (1985) has most carefully and convincingly argued, this is a pseudo-explanation on a par with the "dormitive virtues" of opium. Briefly: there is no good reason to suppose that the "programme" is localized in the genes: in fact, it is clearly distributed over all the components which interact during the developmental process. Secondly, the "programme" does not pre-exist the process it is supposed to "direct"; as we have already noted, each stage in the process sets the conditions for the next stage, so that the process in a very large part "organizes itself".

[2] This is not contradictory with the fact that there are *some* things that can only be learned by long and/or painful experience that result in "a sadder but a wiser man".

[3] This is not contradictory with the previous statement that the number of possibilities is unlimited. For example, there is no limit to the number of prime numbers; but they are only a fraction of whole numbers, and an infinitesimal fraction of real numbers.

That the twin facets of ontogeny - enabling and constraining - both apply to learning is an empirical fact, well known to experienced teachers. And if we have succeeded in our argument so far, this fact may actually seem unsurprising, to the extent that it may seem we are making heavy weather of the obvious. Well, it would be nice to think so; but the hard fact of the matter is that the point of view just put forward runs directly counter to some very widespread common-sense notions. Specifically, these are: (i) the "innate vs acquired" dichotomy; and (ii) the notion that "learning" is a process of "instruction", by which is meant a process of information transfer from teacher to pupil. These two notions are actually interrelated, as we shall see, but we shall examine them critically in turn.

VII. The innate vs acquired dichotomy.

The innate vs acquired dichotomy is so deeply ensconced in our culture that although opinions differ, sometimes violently, as to whether a given character (such as "intelligence" or "schizophrenia") is "innate" or "acquired", even at the height of the battle the protagonists are actually in complete agreement that the character *is* either "innate" or "acquired"; or in somewhat more sophisticated terms, that the determination of the character can be partitioned between "genetic" and "environmental" causes, and that the more is due to heredity, the less is due to the environment. In fact, the very use of the words "innate" and "acquired" implies, conceptually, that there are two *sorts* of development: one sort that is controlled (or "programmed") by the genes, and another sort that is controlled by the environment. Oyama (1985), in a most careful study of this vexed question, has presented in detail the arguments against this sort of developmental dualism: there is, and can be, only *one* sort of developmental system. She diagnoses the source of the mistaken innate-acquired dichotomy in the distinction, even more deeply rooted in our culture, between Matter and Form. In classical Greek philosophy, "Forms" exist transcendentally, typically in a Platonic "heaven of ideas". "Matter" is conceived as basically inert, or at most chaotic; left to itself, it can do nothing significant. In order for an *organized* material process to occur, the matter involved must be literally "in-formed", i.e. it must receive its "form" from an external source. Applied to ontogeny, this means that the developing embryo, as a material entity, cannot possibly be an autonomous self-organizing system: it must receive its "in-formation" from an external source; and there are then two possible repositories for this information, the genes[1] and the environment. It is in this conceptual framework that it does indeed make sense to evalute the relative contributions of "genetic information" and "environmental information". We have argued here that this dualistic conceptual framework is deeply mistaken; and indeed it leads to a curious ressuscitation of vitalism, with talk of "selfish" genes "reproducing themselves" and "directing the development of the organism"; of genes "for" all sorts of characters, from diseases to physical or intellectual abilities. There are all sorts of reasons why this is wrong, some of which we have already mentioned; but a major reason, which we shall now examine, is the concept of the "transmission of information".

VIII. Information transfer

Perhaps the best way to see what is wrong with the concept of the "transmission of information" as an explanation of biological phenomena is to start with the situation where it is correct: that of telecommunications engineering. It is in this context that

[1] The genes are of course situated in the nuclei at the centre of each cell, and so it may seem strange to consider that they are "external" to the developing organism. However, what is at stake here is not simply the geometrical location, but the conceptual relation; and ever since Weismann's distinction between the germ-plasm and the soma, the "germ-plasm" has been *conceptually* exterior to the somatic processes which it "instructs" without ever being affected in return.

Shannon's precise definition of "information" makes sense[1]. To spell things out, the Shannonian schema *presupposes* :

i) an "emitter", an entity with a finite number of alternative states; this specifies the information to be transmitted;
ii) a "receiver", a different entity but which must have the same set of alternative states;
iii) a "signal", a third entity which has a set of states which can be put into precise correspondance with the states of the emitter and receiver;
iv) a "code" specifying the correspondance relations between the states of the emitter/receiver on one hand, and the states of the signal on the other hand;
v) a mechanism for "encoding" the state of the emitter into a state of the signal;
vi) a mechanism for "decoding" the state of the signal into a state of the receiver;
vii) a mechanism for physically transmitting the signal from the site of encoding to the site of decoding.

It is to be noted that this schema is purely syntaxical and formal, and that the "meaning" of the message does not enter into the account. This is, indeed, correct: the "meaning" of the signals transmitted by a telephone line (for example) is not the concern of the telecom engineer.

Now there are a certain number of biological situations which can, at least on the face of it, be considered as examples of "information transfer" in this precise sense: for example, DNA nucleotide sequence to protein amino-acid sequence via mRNA sequence; cell-cell signalling, by endocrine or autocrine molecules; animal communication by visual, auditory or olfactory signals; even human language (although the dangers and possible misconceptions here have been clearly exposed by Reddy 1979). However, from a biological point of view, the Shannonian schema *presupposes* practically everything of interest. *How does it come about* : *that* the states of the "emitter" *and* the receiver can be categorized in just this way? ; *that* there is a "signal" which also has the requisite number of states? ; *that* the encoding and decoding operations can be realized materially? ; *that* the signal is transmitted from the emitter to the receiver? And finally, above all, there are the pragmatic and semantic dimensions: how does it come about *that* this "transfer of information" actually results in a co-ordination of actions between the emitter and receiver that contributes significantly to their mutual viability? The biological value of information cannot be measured in Shannonian "bits": billions of bits can be useless, one bit can be crucial.

To sum up: the limitation of the Shannonian schema is that it *presupposes* practically everything that requires explaining. Shannonian information is contentless and therefore of no use for functional explanations. It is also of little use for operational explanations, since once it has been explained how to get to a situation in which one may legitimately speak of Shannonian information being transferred between two communicating organisms, all that needs to be explained has already been explained (Di Paolo 1998). Shannonian information can specify which one of a finite number of pre-defined possibilities will be realized; but it does not, and quite fundamentally cannot, specify what these possibilities are in the first place.

This has important implications, both for genetic information and environemental information.

Genetic information, in the strict Shannonian sense of the term, is encoded in the nucleotide sequence of DNA. This information can, and does, specify the linear sequence of amino-acids in proteins. However, this "information" does not *and cannot* specify the

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[1] I, the information content of a message composed of n elements, is defined by the formula : $I = - \Sigma p_i.(\log p_i)$, where p_i is the probability of element i (i=1, 2,...n) in the message. This is the *only* scientifically precise definition of the concept "information", and we propose to adopt it.

genetic code, and even less *that* it is the amino-acid sequence of a protein that will be specified. Thus it is quite incorrect to say that "DNA codes for proteins". However, if DNA does not "code for" proteins, it codes even less for the development of a multicellular organism.

Thus, genetic information, although important, does not have the quasi-magical properties often attributed to it. What then can be said about environmental information?

The *same* limitations that apply to genetic information also apply to environmental information. Such information can, and does, specify which of a number of pre-defined possibilities will be realized. However, it does not *and cannot* specify what those possibilities are in the first place. This means that *instruction*, in the strict sense of the term, is radically impossible.

IX. Conclusions

What are the implications of all this for the principal subject of this article, learning? Well, if instruction is impossible, then the only things that can be "learned" are things that were *already possible* at that stage of development. This is reminiscent of Vygotsky's notion of "region of proximal development" (Vygotsky 1962).. Another illustration is given by Stern (). Human parents often have the intuitive gift of attributing a capacity to their infant *just before* the infant is actually capable of the performance in question. To come back to our examples in the introduction: we attribute to a one-year old baby the capacity to walk *just before* she is actually able to take her first steps; we attribute to a 2-year old the capacity to understand what we say, and we interpret his response as an intelligent, meaningful expression, *just before* this is spontaneously the case. What is fascinating, of course, is that by doing so, the parents actually trigger the emergence of the capacity in question. Another example is provided by the training of performing animals. Gifted trainers do not even attempt to train an animal to do an arbitrary trick; based on a fine observation of what the animal already does spontaneously, they have a developed intuition as to what the animal can *almost* do already, so that what is required is a minimal increment or modification of the naturally occurring developmental process.

The limitations on what can be learned do not stem from the fact that there are certain bits of information which are "in the genes" and not in the environment. They stem from the fact that at any particular stage in the dynamical process of development, only a very restricted set of "next steps" are possible. This will, however, only be cause for disheartenment if one persists in trying to teach the impossible. Ontogeny, as we have said, is constraining; but it is also enabling, and at any given point in development, the range of "next steps", although severely restricted with respect to "everything", is in fact very large, and certainly far larger than what can be actually realized. In other words, there are many things that can be learned, but will only be actually learned if the appropriate conditions are provided. A key element in the art of teaching lies in proposing a series of graded exercises which put the learner in a position to accomplish a series of tasks which are each within the realm of what she can *already* do, but which culminate in the accomplishment of feats which could never have been done immediately. This applies to skills of all sorts: from motor skills, such as learning to ride a bicycle or to ski, to intellectual skills such as learning to play chess or to do mathematics, to artistic skills such as learning to play a musical instrument or to paint or to dance.

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