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Why Electromagnetism Is the Only Causal 'Spook' Required to Explain Completely Any Human Behavior or Institution'

from

The Relationship of Verbal and Nonverbal Communication

Edited by
Mary Ritchie Key

Contributions to the Sociology of Language

25

Joshua A. Fishman
Editor

1980
MOUTON PUBLISHERS • THE HAGUE • PARIS • NEW YORK

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Why Electromagnetism Is the Only Causal 'Spook' Required to Explain Completely Any Human Behavior or Institution'

In *Chance and Necessity* (1971), Jacques Monod has successfully demonstrated, I think, that ontogenesis — the process by which the ontogeny of an organism occurs — is merely an extension of the process by which atoms unite into simple molecules and simple molecules unite into more complex molecules. This is the process of formation of electron bonds, covalent arid noncovalent; its outcome in each case is determined by two things: (1) the structures and spatiotemporal relations of the material structures that went into it, and (2) the mysterious universal causal force or fundamental principle called Electromagnetism. My purpose here is to show that all human behaviors and institutions, too, are determined entirely by (1) entering material structures and relations and (2) Electromagnetism.

SPOOKS

Before I go on discussing ontogenesis, let me digress for a moment to discuss mysterious universal causal forces and fundamental principles or, to use a good old American term in a way borrowed from Wes Jackson (personal communication), 'spooks'. Physicists today generally recognize four spooks; namely, Gravitation, Electromagnetism, and the Strong and Weak Interactions in the atomic nucleus.' Reducing the number of spooks is a main goal of physics. In the last century, Maxwell showed that electricity and magnetism can be reduced to one spook, Electromagnetism. Early in this century, Einstein reduced inertia and gravitation to Gravitation alone, and he died trying to unite Electromagnetism and Gravitation under his unified field theory. Today, some theoretical physicists are attempting to reduce the Weak Interaction and Electromagnetism to a single spook; they are encouraged by the fact that these two forces have about the same strength, whereas Gravitation is much much weaker, and the Strong Interaction much much stronger, than they.

To anticipate a bit: the characteristics of any living thing or any product of a living thing — including a behavior, an artifact, or a social organization — are entirely the outcomes of two sequences of events that happen to that thing and/or product — its ontogeny and its phylogeny. Very roughly, its ontogeny is the sequence of events through which the thing develops from seed, and its phylogeny is the sequence of events through which the seed acquired its characteristic ontogeny in the first place. What I aim to show in this essay is that both the ontogeny and the phylogeny of any thing or product can be completely explained without reference to any spook other than those now recognized by physics. In fact, of those four we can make do practically all the time with Electromagnetism alone. There are no special spook principles that apply to organic life — I think Monod shows that — and there are no special spook principles that apply to human affairs; no Duality Principle, no Principle of Equivalence of Siblings, no Principle of Least Effort, no Principle of Logico-Aesthetic Integration, no Principle of Adaptation or Anticipation; we don't need them to explain human affairs scientifically, so to invoke them is to violate the rule of science that entities must not be multiplied beyond necessity: Occam's razor.

EXPLANATION

To the extent that they are valid as empirical generalizations, some of these 'principles' may be useful in scientific activities, and in practical affairs, as mnemonic devices. But the purpose of science is to *explain* events and their relations, not merely to generalize relations between classes of events, although such generalizations may be useful steps in the enterprise of developing explanation. For example, 1 think we *explain* the movement of the dial on a 'pressure' gauge, of a certain closed cylinder of gas which is being heated, by means of the kinetic theory, i.e. by Electromagnetism. We describe (among other things) the changes in state and activity of the gas molecules in that very cylinder, and show how the gas molecules strike the piston or diaphragm of the gauge with increasing velocity and frequency. We do not explain the event, the movement of the dial, by deduction from Charles' Law; indeed, we do not introduce terms like 'temperature' or 'pressure' into our explanatory discourse at all. Charles' Law, which states the relationship between the temperature and pressure of an enclosed gas, is not an explanatory principle; it is, rather, a general proposition based, first, on generalization and extrapolation from a number of specific experimental instances and, second, on its predictability from the kinetic theory; it does not,

however, enter into the *explanation* of specific experimental instances. From the point of view of the history or evolution of science, of course, Charles' Law was very important; its discovery and confirmation led others to work Out the kinetic theory. And, of course, it could be very useful in designing, say, a boiler.

I think that microtheories (such as the kinetic theory) are often slow to be accepted because they are contrary to our habits of thought. Confronted by a macroevent, such as the movement of a dial, we prefer to invent, and to cling to, a spook like 'pressure' — something that will give us a macroexplanation. As the Reverend Mr. Hale says, in Arthur Miller's *The Crucible*, 'Man, we must look to the cause proportionate.' A recurring example of this practice, in evolutionary studies, is the `argument from Design'. But the ubiquity of the inverse square law suggests to me that *all* macroevents have microexplanations. If that is true, we are wasting our time when we look for correlations between classes of macroevents without simultaneously seeking microexplanations to provide the causal links; heating the cylinder was a determinant of the moving of the dial not because 'heating an enclosed gas always increases its pressure', but because we have a series of microexplanatory links between the heating and the moving.

ONTOGENESIS

For purposes of this article, ontogenesis is not the same thing as ontogeny. The word `ontogeny' refers to a specific sequence of events in a specific organism, from conception to adulthood and conception again; then on to senescence and death. (The ontogeny of a bacterium, I suppose, begins with cell division and ends with the next cell division.) An initial characterization of `ontogenesis', on the other hand, would be `the underlying process by which ontogeny takes place'. But that characterization would be a very bad definition, or really no definition at all; the 'by which' part spoils it, turning it into nothing but a label covering ignorance; as soon say lithogenesis is the underlying process by which lithogeny (rock-formation) takes place'. By studying the ontogenies of organisms of various kinds, however, we begin to get an idea of what ontogenesis *is*, and not just what it does. We see the same things, the same variants of the ontogenetic process, occurring again and again.'

As one variant, we see two or more structures that have drifted close together suddenly aligning themselves in a certain relationship and bonding together to form a larger structure; we see this repeated again and again — when two structures similar to those meet they align and

form a similar larger structure. We see this in the assembly of the protein coat of a tobacco mosaic virus, where the initial structures are all the same kind of protein and they form a cylinder by bonding together in a tight helix. We see it also in the spontaneous linking up of the disparate parts of a T-4 bacteriophage, and ribosomes may be formed from protein molecules and RNA nucleotides by this automatic 'self-assembling' process.

A second variant of the ontogenetic process occurs when a relatively large structure unites temporarily with a small structure, as in the first variant; but this time a bond within the small structure is broken, and then the two components of the small structure are released separately, leaving the large structure in its previous form; in other words the large structure, an enzyme molecule, breaks the small structure down into two of its constituents. Examples include a chlorophyll molecule breaking a water molecule down into hydrogen and oxygen ions, and a digestive enzyme breaking a starch down into a sugar and a something else.

A third variant is an opposite of the second. An enzyme forms a temporary structure with *two* small structures which bond together while attached to the enzyme and are released as a single new structure, again leaving the enzyme as before. A series of such reactions, each involving a different enzyme and two structures constructed by previous enzymes in the series, builds up quite large structures — structures with as much as one percent of the mass of the enzyme itself. Indeed, structures constructed this way include *components* of enzymes and other proteins; namely, amino acid molecules. They also include nucleic acid molecules, vitamins, sugars, fatty acids — in fact, any 'middle-sized' molecule. Each enzyme, of course, is different in structure from any other enzyme and, according to its structure, ordinarily enters into just one constructive reaction with just two specific small structures.

A fourth variant is as follows. DNA nucleotides in a strand bond to free nucleotides of DNA or RNA and then release the latter as a complementary strand, forming a gene or gene-partner, an RNA messenger, or perhaps the RNA component of a ribosome or amino-acid transfer unit.

A fifth variant is really a sequenced combination of all of the first four. One end of an RNA messenger-strand bonds to a ribosome, then its first codon bonds to the anti-codon of a transfer unit. The messenger then moves one codon-length into the ribosome, its second codon bonds to another transfer unit, and the amino acid bonded to the first transfer unit bonds to that carried by the second. As this process is repeated over and over, the messenger ratchets through the ribosome, the transfer units unbond from their amino acids, and a strand of 100 to 300 amino acids, a polypeptide, emerges from the ribosome.

A sixth variant, practically the same as the first, is the automatic folding of the polypeptide into its characteristic globular, protein form, as various free bonding-sites on its constituent amino acids form bonds with each other.

A seventh variant is the temporary bonding of a gene or protein by a small molecule which prevents, or permits, or alters, its formation of other bonds. The latter may include those bonds entered into in the sixth variant; thus the temporary bonding or the subsequent unbonding may change the gross shape of a protein.

An eighth variant is alteration in any of the above reactions by an increase or decrease in the activity of atoms in the entering structures, caused by electromagnetic radiation from other, near or distant structures — as when sunlight activates chlorophyll in a green plant or stimulates enzymes to form vitamin D in an animal.

There may be other variants that I haven't remembered to mention. It seems clear, however, that all the microprocesses of ontogenesis are based on the formation and de-formation of chemical bonds between material structures of various kinds and of various submicroscopic and microscopic sizes: The only spook involved is that which causes bonding to take place — Electromagnetism.

It is not difficult, moreover, to imagine how an ordered sequence of such microprocesses, in series and parallel, can result in an ordered sequence of macroevents, producing a structure as complicated as a virus or a cell, stocking it with substructures of various kinds, and even endowing it with what for it is gross behavior. And, of course, the ontogenetic process of mitosis, or cell division, is well understood and, again, it is not difficult to imagine several, or even several dozen, cells remaining bonded together, membrane-protein to membrane-protein, after division. To get a true multicellular organism, then, we need only have a mechanism to control the ontogeny of individual cells — to stop and start it and to produce cells of different sorts from the same identical set of genes. The details of this mechanism are not at all understood yet, although it seems clear that at every differentiation point in the ontogenetic process some form of positional cueing of genes is involved. In other words, every gene for every kind of cell is included in every individual cell, but the sequential order of microevents varies according to the cell's relationship to events occurring in other cells and in the rest of its surroundings; these events 'disturb' the cueing of its genes, that is to say they determine or prevent the bonding — and hence inactivation — of some genes, through the seventh variant of the ontogenetic process. Many genes, as a result, never participate in the fifth variant of the process at all in certain cells so that, for example, a liver cell lacks enzymes and even larger structures that a neuron has, and *vice-versa.*

THE SPECIAL ROLE OF THE GENES

Biologists are fond of saying that '(only) the genes contain all the information' for the ontogeny of a particular organism. 1 think that abstract biological expression can be dissected into the following: In any ordered set of ontogenetic processes, some variables are controlled by processes of the set and some are not; specifically, some structures entering into reactions in the set are the products of preceding reactions and some must be found in the surroundings. The latter, of course, must be found in *full sufficiency* or the ontogenetic process will not run its full course. Among the former, the structures produced by preceding reactions, genes have a sort of existential priority: An ontogenetic process can be completely reconstituted in the total absence of structures of one specific kind or even of several specific kinds *except for genes;* if enzymes of one kind are completely absent, for instance, they will be assembled, in the necessary quantity, through the fifth variant of the process; if amino acids of one kind are completely absent, enzymes will synthesize them through the third variant; and so forth. But if *genes* of one specific kind are completely absent they *can't* be synthesized, so the enzyme or protein they code for can never be replaced as it is used up or worn out, and so the ontogenetic process cannot run its full course and may very well abort completely or even run wild. So, in a sense, genes are like those structures that must be found in the surroundings; unlike those structures, however, they need not be found in full sufficiency, because they (and they alone) have the ability to *replicate themselves* through the fourth variant of the ontogenetic process. Theoretically, at least, just one gene of each kind is sufficient for the construction of a trillion-celled organism or, indeed, a whole population of such.

So we are prepared to make two assertions:

First, any microevent in the ontogeny of any organism can be explained by two things: (1) the structure of the structures that entered that event, in particular the spatial relationships of bonding-sites on their `surfaces' and the electrical charge at each bonding-site, and (2) the great spook of Electromagnetism.

Second, any macroevent in the ontogeny of an organism is nothing but an outcome of an ordered sequence of such microevents — in fact, the ontogeny as a whole is such a macroevent.

From these two assertions it follows that given a complete set of genes, a 'starter-set' of amino-acids, enzymes, ribosomes, etc., an adequate supply of certain small and middle-sized molecules and a certain range of levels of energy of different frequencies in the surroundings, and the great spook of Electromagnetism, each step in the ontogeny of a bacterium

or a rat, from fertilized egg to adult and fertilized egg, is inevitable.

Aha, one might say. Such a process might result in an adult rat, but it wouldn't be a normal adult rat; it would be a stupid, incompetent, psychologically maimed adult rat, because it wouldn't have had the *experience* a rat needs to grow up properly. And, of course, that is right; indeed, it wouldn't be a proper rat at all.

But let's look again and see what ontogenesis can do besides constructing bacteria and the psychologically empty husks of rats; perhaps, given the necessary material structures and relations, it can account for all behavior, even learned behavior, even culturally acquired behavior and its products.

BEHAVIOR

First, let's consider an act of (unlearned) gross behavior on the part of a simple metazoan.' Such a macroevent is the outcome of an ordered sequence of microevents, some occurring in parallel, some in series, all of them examples of the ontogenetic process. First, a molecule, a change in electromagnetic radiation, or some other disturbance in the organism's surroundings releases activity of an enzyme in a peripheral cell. That activity, in turn, results in the release of packets of acetylcholine or other neurotransmitter molecule into a synaptic cleft. The latter acts on proteins in the membrane of the following nerve cell (neuron), depolarizing that membrane in a chain reaction, the action-potential, which results in the dumping of neurotransmitters into the next synaptic cleft, and so on, until finally, in a motor-cell, some enzyme-manufactured small molecules bond to protein molecules in the cell-membrane causing the protein molecules, and thus the membrane, the tissue, and the organ (muscle), to contract.' That's ontogenesis, all right. We probably wouldn't want to call it 'ontogeny', however, because it doesn't have a (semi)permanent effect; that is, in a few moments the muscle relaxes again. The sequence is *based* on ontogeny, of course: an ontogenic sequence made the sensory cells, the interneurons, the motor cells, and all the supporting cells and tissues, and stocked each cell with the enzymes, neurotransmitters, and so forth, so that it took only a cue from the environment to set the whole thing off. So a behavioral event is contingent upon *two* sequences of ontogenetic processes: There is the sequence of processes through which the behavior is released but, first, there is the sequence of processes which constructed the microstructures which enable those releasing processes, i.e. which constructed the macrostructure which can do the behavior.

INSTRUCTIONS

An *instruction,* a structure which can do a certain behavior, is like any other material structure except that it has this peculiar ability to behave on cue and then return to its pre-behavior state and hence, to behave again on cue. One can say that the instruction *is* the structure which can do behavior: or, metaphorically, that the structure *carries* the instruction; or that a pre-existing structure is *programmed* with the instruction by the modification of its fine structure.

The act of gross behavior described above was performed by an instruction consisting of cells, enzymes, and so forth. But when we described the microevents that underlay it, we described a sequence of, again, behavioral acts, each performed by a different microstructure. So each neuron is an instruction, in fact each enzyme molecule and membrane-protein molecule is an instruction — components, at various levels of inclusion, of the gross-behavior instruction; the ontogenetic processes in the second sequence, through which the behavior is released, are behaviors of those instructions, the results of one behavior being the cue for the next. And the ontogenetic processes in the first sequence, which constructed the gross instruction-structure (indeed, the whole organism) in the first place, are also behaviors of instructions; those instructions include not only enzymes and other proteins, of course, but also genes, RNA messengers, ribosomes, and transfer units; they, too, are cued either by variations in the surroundings of the developing organism or by the behavioral results or products of other instructions in the series.

To recapitulate, a gross behavior is contingent upon two sequences of ontogenetic processes. Via the first sequence, instructions construct an organism and program it with instructions; in other words, ontogeny takes place. Via the second sequence, those instructions release the gross behavior, and each returns to its respective *status quo ante.*

It would appear that ontogeny does not take place via the second sequence, since no (semi)permanent structure results; there appears to be no durable thing that we can speak of the ontogeny *of,* as we could speak of the ontogeny of an organism or instruction occurring via the first sequence. In fact, however, gross behaviors of organisms do sometimes result in (semi)permanent structures, of two kinds. A structure of the first kind is an artifact, a material structure such as a spider's web, a bird's nest, a clay pot, or a house. There is no incongruity in speaking of the ontogeny of an artifact, referring to the sequence of behaviors of instructions — including, now, gross-behavior instructions — by which it is constructed.

SOCIAL INSTRUCTIONS

A structure of the second kind is a *social* structure or group or organization, constructed and maintained as follows: Some of the gross behavior instructions of an organism are (1) cued by the results of gross behavior by a conspecific (an organism of the same species); or (2) `directed toward' a conspecific, e.g. when they behave, they move the behaver along a vector determined by the location of the conspecific or they cue another instruction carried by the conspecific; or (3) both. Among these *social instructions* are many whose principal or even sole behavioral outcome is the bringing or keeping of organisms into certain spatial relationships (usually, but not always, including proximity). An ordered sequence of such *sociogenic* instructions, acting in series and in parallel, constructs and maintains a social organization in a manner quite precisely analogous to the way in which genes, messengers, enzymes, etc., construct and maintain an organism. As with artifacts, there is no incongruity in speaking of the ontogeny of a social organization or in asserting that that ontogeny is ontogenetic, i.e. based entirely on chemical bonding processes, hence on Electromagnetism.

While a social orgamzation is maintained, of course, it has effects on the behaviors of individual organisms, both members and nonmembers. These effects fall into two categories: In the short term, group-behaviors release or cue an instruction carried by one or more individual organisms; the resultant behavior may itself be sociogenic, as when a group-member, cued by being in the group situation, directs some sanctioning behavior toward another member cueing, in turn, some conformist behavior on his/her part — social control, in a phrase. In the longer term, the social orgamzation and its behaviors make up a salient part of the environment controlling the subsequent *evolution* (phylogeny) of the instructional *repertory* of the population — again including sociogenic and other social instructions. I will return to this evolutionary/environmental role of social organizations later.

As implied above, not all social instructions are sociogenic; not all participate in the *ontogeny* of a social organization. Many participate in the gross behavior of the social organization instead (or in addition). In other words, just as there are two sequences of ontogenetic processes — behaviors of microscopic instructions — in the organism, so there are two sequences of ontogenetic processes — behaviors of gross orgamsmic instructions — in the organization.

For example, the greeting and allo-grooming instructions of *ants* and the instructions that bring them home after foraging behave in the first sequence, being ontogenetic of the ant society; the instructions that build

the nest (an artifact, n.b.), swarm out to attack predators, cooperate to feed the queen and larvae, etc., behave in the second sequence. The latter instructions are thus components of the gross-behavior instructions of the society.' The ontogeny of the ant society and the social behaviors of both kinds, like the ontogenies and behaviors of the individual ants, are composed entirely of variants of the single ontogenetic process (summarized above), and Electromagnetism is the only spook involved.

LEARNING

The information I have presented so far to back my arguments is quite certain, but now I must leave the certain for the plausible. In the above discussion I remarked that 'an ontogenic sequence (first sequence of ontogenetic processes) made the sensory cells, the interneurons, the motor cells, and all the supporting cells and tissues, and stocked each cell with the enzymes, neurotransmitters, arid so forth, so that it took only a simple cue from the environment to set the whole thing off (following the second sequence of ontogenetic processes)'. Now, that description will do for a gross behavior instruction which is (entirely) genetically programmed. What I think happens in learning, or environmental modification of behavior, is this: There are a great many possible, incomplete sets of neurons which, if complete, would compose a gross behavior instruction; which, that is, would comprise a complete *neural routeway* and thus could enable a sequence of ontogenetic processes of the second, or behavior releasing, kind. But each of these neural routeways is incomplete because one or more of its constituent interneurons lacks a certain enzyme; the enzyme, for instance, that does an essential step in the assembly of neurotransmitter molecules, or of the molecule that controls the release of neurotransmitter into a following synaptic cleft. Anyway, because of the lack of the enzyme, an action potential (behavior) of that neuron does not cue the following neuron(s). Learning, then, involves 'activation' or 'programming' of a set of neurons through cueing the production of the lacking enzyme in each. Since every neuron, like every other cell, contains a complete set of genes, each neuron in question contains the gene that codes an RNA messenger to produce that enzyme, but the action of that gene is blocked because it is bonded by a molecule — let's call it molecule X. Molecule X was produced, I suppose, by an enzyme process early in ontogeny, a process which has since been permanently inactivated. (So to say that a neural routeway is 'genetically programmed' is really to say that, by positional cueing, molecule X was never produced in any of the neurons

in that routeway.) So, if this neuron is to be able to cue the other neurons that synapse upon it, we need only get molecule X to let go of the gene. A simple way to do that is to introduce into the cell another molecule for which molecule X has a greater affinity than it has for the gene, much as iron ore is reduced by attracting its oxygen atom away with a carbon atom. Now, how do we introduce this molecule — let's call it Y — into a neuron? Since the neuron is deep in the nervous system, and since we must address it very precisely, the only way I can think of is through a neural routeway.

In short, I propose that a completely programmed (`standard') routeway and an incomplete routeway run close together for part of their lengths. Suppose that the standard routeway is responding to cue C, each of its constituent neurons firing in sequence and a certain gross behavior occurring. When it fires, each neuron of the standard routeway releases molecule Y into its immediate vicinity. Suppose, next, that in that vicinity is a nontransmitting neuron N cued, via a partially complete neural routeway, by cue C'. Because C' is occurring (as well as C), N is firing, and *only because it is firing* it absorbs the Y, from a nearby standard routeway neuron, through osmosis.' Y then bonds to X, unblocking the gene, which proceeds to code RNA messengers, which construct the enzyme which produces neurotransmitter. N thereby becomes a *transmitting* neuron; whenever it fires, thereafter, it cues the neurons which synapse upon it. A few milliseconds later, one of those following neurons is programmed by the same process, which is repeated over and over again until a new routeway is completed. So, with serial and parallel repetitions of this process, an environmental cue, to which the existing standard routeway responds, directs the programming of a *new* routeway which responds to a different environmental cue, the one that was firing the neurons in the new routeway as it was programmed; and the change is permanent for the life of the organism. The organism is carrying a new gross behavior instruction.

Needless to say, this description is vastly oversimplified, both as to the quantity and quality of activity that must take place in the environmental programming of a single gross behavior instruction, and as to the variety of results.' But it explains or at least accounts for a couple of phenomena; namely, the increase in RNA that has been observed to take place in neurons during intensive learning, and the ramification or diffusion of similar experience-acquired instructions throughout large portions of the brain — since each experience-acquired routeway can serve as a *standard* routeway for subsequent events of learning, when C and C' are the same as well as when they are different. Also, since one must begin with genetically programmed routeways (albeit perhaps a

very large and variegated endowment thereof), it also accounts for the fact that learning is species-specific; e.g. rats learn mazes easily but can't be trained to copulate for a food reward, and certain birds that navigate by the sun easily learn to look for grain at different times of day in what they calculate is a certain direction (say, east), but find it difficult to learn to look for grain simply in the direction of the sun.

More important, from my point of view, this account describes processes both ontogenetic (chemical) and ontogenic (helping produce normal adult animals). It can easily be integrated with the account of the ontogeny and gross behavior of social organizations, as well, since the environmental cue for existing and for learned routeways may be a conspecific's behavior, and the behavioral outcome of either routeway (or both) may be 'directed toward' conspecifics; in other words, social instructions may be acquired this way just as well as other gross behavior instructions.

So it seems plausible, to reiterate, that, 'given a complete set of genes, a 'starter-set' of amino-acids, enzymes, ribosomes, etc., an adequate supply of certain small and middle-sized molecules . . . a certain range of levels of energy of different frequencies, ***and an adequate supply of sufficiently variegated sensory cues*** in the surroundings, and the great spook of Electromagnetism, each step in the ontogeny of a bacterium or a ***healthy*** rat ***and even a healthy rat society*** is inevitable' (repeated from above, emphasized portions added). Learning, in short, is another outcome of the ontogenetic process.

CULTURAL ACQUISITION

All we need to add, in order to substitute 'human' for 'rat', in the above, is a means for transmitting an extragenetic tradition, or behavioral heritage. If my ontogenetic model of learning is plausible, one can easily imagine an expanded learning brain in which events like these take place: A certain genetically programmed routeway is fired by a cue consisting of (light rays reflected from) a conspecific ***animal who has just completed a behavioral act.*** The ***result*** of that behavioral act is also, simultaneously, cueing a neural routeway, but one which is heretofore incomplete. Hence the first, genetically controlled, routeway completes the programming of the second routeway. Subsequently, the second routeway, the one which responds to the behavioral ***result,*** serves as a standard routeway for ordinary learning. Thus the observing animal's behavior is shaped to approximate that of the demonstrator; i.e. he carries a set of neural instructions analogous to a set carried by the

demonstrator. He has learned from observation. With repetition of such learning, genetically identical animals of different populations can come to carry different gross behavior instructions, with a high amount of variation between populations and a low amount of variation within populations.

This observational learning capacity goes to a sort of limit in a brain large enough (enough free neurons) that the observer records the results of the demonstrator's behavior at a rate varying around about ten per second. At this rate, each behavioral result is 'recorded' as the cue which released the behavior which led to the next behavioral result, so observational learning in such an animal is very smooth, very swift, and very accurate.

— Social groups of such animals develop and maintain extragenetic traditions or behavioral heritages or cultures, consisting of instructions programmed as above. Being sort of traditional myself, I call neural instructions so programmed 'cultural instructions'.

—Some sets of such cultural instructions are in ontogenic pathways leading to the construction of artifacts (e.g. a tool, a pot) and social organizations (e.g. a hunting party, a university).

—All elements in that cultural process are variants of ontogenesis.

But in this animal, we have a new requirement for the ontogeny of a healthy animal and society, besides the ones we arrived at immediately preceding. In a sense, this is a special case of any learning species' requirement for 'an adequate supply of sufficiently variegated sensory cues in the surroundings', but in the cultural case, those 'cues' do more than stimulate a lot of self-programming in the animal — they 'contain all (or nearly all) the information' that the learning animal thereby acquires. If that sounds familiar, it should. Ontogenically, cultural instructions are precisely analogous to genes. In a preceding section, `The Special Role of the Genes', I dissected the statement that 'the genes contain all the information'; to paraphrase that dissection, this time including cultural instructions: 'In any ordered set of ontogenetic processes, some variables are controlled by processes of the set and some are not; specifically, some structures entering into reactions in the set are the products of preceding reactions and some must be found in the surroundings. The latter, of course, must be found in full sufficiency or the ontogenetic process will not run its full course. Among the former, genes ***and cultural instructions*** have a sort of existential priority: An ontogenetic process can be completely reconstituted in the total absence of structures of one specific kind or even of several specific kinds, ***except for*** genes and ***cultural instructions;*** if social groups of one kind or tools of one kind are completely absent, for instance, they will be assembled,

in the necessary quantity, by (carriers of) the appropriate cultural instructions. But if *cultural instructions* of one specific kind are completely absent (e.g. those for tying the blade of the tool to its shaft) they can't simply be made up, so the tool or social group they help construct can never be replaced as it wears out or breaks up, and so the ontogenetic process cannot run its full course and may very well abort completely or even run wild. So, in a sense, cultural instructions are like those cues that must be found in the surroundings; unlike those cues, however, they need not be found in full sufficiency because they (and they alone, among such cues) have the ability to replicate themselves, through the learning process just described. Theoretically, at least, just one cultural instruction of each kind is sufficient for the construction and maintenance of a complete functioning human society of millions of people.'

So genes and cultural instructions have important characteristics in common: they are behaving structures which, when appropriately cued, enter into ontogenetic processes, often ontogenic processes. Because they are the only structures capable of replicating themselves, they play a role in those processes fundamentally different from that played by other structures; metaphorically, only they 'contain all the information' necessary for constructing the complete set of routeways. Finally, their components, although these are instructions (e.g. nucleotides for genes, activated neurons for cultural instructions), are not capable of replicating themselves; so genes and cultural instructions are *elementary self-replicating instructions* (SRI's, for short). Given complete sets of both, and the necessary environmental stuff with which to get started and keep going, each step in the ontogeny of a total human society, including its personnel, its institutions, and its traditions, is inevitable.

NATURAL SELECTION, THE ONTOGENETIC PROCESS OF GENETIC AND CULTURAL CONTINUITY AND EVOLUTION

To finish my task of presenting a method of `explain(ing) completely any human behavior or institution', promised in the title of this article, there remains only to sketch out the process by which a set of SRI's — of elementary self-replicating genetic and cultural instructions — is compiled and maintained; the process, in other words, which underlies the phylogeny and continuity of a population's genetic and cultural repertory. This process, too, will be found to be entirely ontogenetic, i.e. chemical, the only spook involved being Electromagnetism.

As SRI's, genes and cultural instructions have certain characteristics which determine their role in the phylogenetic process. A gene is a string

of roughly 300 to 900 DNA nucleotides (of which there are four different kinds) which codes a messenger (fourth variant of ontogenetic process), which codes a protein (fifth variant). Of all the astronomical number of possible DNA-strings of that length, only a tiny fraction are genes (the others don't code protein). Of all the huge number of genes, only a tiny fraction are equivalent; i.e. only a tiny fraction code for the same protein. So the *a priori* probability that any given gene (or its equivalent) will be part of even a large set of SRI's is vanishingly small. Yet at least a tenth of all DNA nucleotides found in strings in nature are elements of genes, and those genes that are found together in sets are, most improbably, the very genes that interact in ontogenetic processes that produce elaborate and intricate organic, social, and artifactual structures — structures superbly adapted to survive, endure, and prevail, in the peculiar particular environmental situations in which they are found. How are these genes 'selected' from the total universe of actual genes, theoretically possible genes, and nonsense DNA- strings?

A cultural SRI is a set of activated neurons synapsing on one another to form a reticulated set of strings through a primate brain, linking a huge number of sensory fibers to a huge number of motor fibers. There is a practically infinite number of such sets possible in any brain. Of these, only a tiny fraction are actually cultural SRI's, because only that tiny fraction, cued by some coherent sensory stimulus, in turn cue some coherent motor activity. Of all the huge number of cultural SRI's, only a tiny fraction are equivalent; i.e. only a tiny fraction respond to the same cue and produce the same behavior. So the *a priori* probability that any given cultural SRI (or its equivalent) will be part of even a large set of SRI's is vanishingly small. We have no way of estimating what proportion of the programmed neurons in a human brain are components of cultural instructions, but it's clearly substantial; and those cultural SRI's that are found together in sets are, most improbably, the very cultural SRI's that interact with each other and with genes in ontogenetic processes that produce elaborate and intricate behavioral, social, and artifactual structures — structures superbly adapted to survive, endure, and prevail, in the peculiar particular environmental situations in which they are found. How are these cultural SRI's selected from the total universe of actual cultural SRI's, theoretically possible cultural SRI's, and nonsense networks of activated neurons?

To begin, it might be well to recall that SRI's are themselves very intricate and fragile material structures. SRI's of both kinds, and their components, are highly vulnerable to electromagnetic radiation of various frequencies, to dismemberment by chemical agents of various kinds, to desiccation, to mechanical damage, and so forth. In other

words, they can exist only at locations in space/time where certain very narrowly defined conditions obtain — where the value of each of a large number of physicochemical variables falls within a very narrow range. And — it seems obvious to say it — SRI's can exist only at locations where all their components not only *and* occur but *do in fact* occur.

Now, let's perform a thought-experiment. Prevent the SRI's on the planet Earth from behaving for a few days or a week, until all ontogenetic activity ceases. Then try to find a location, anywhere on Earth, where the conditions of existence of an SRI are met. There will be very few, if any, such locations. That's strange; a week ago there were billions of such locations. How could that have been? The answer seems obvious: as a rule, SRI's can *exist* on this planet only when the *behaviors* of SRI's, through ontogenetic processes, have met the conditions of their existence.

We must keep in mind, however, that there are many different sorts of locations on the planet, in terms of the exact values of the different essential variables at each. And the behavior of an SRI, and hence its effect in ontogenesis, is highly specific, and is a strict function of its fine structure. So for any given spatiotemporal location, certain *specific* SRI's must occur and behave 'nearby' if the conditions of SRI existence are to be met at that location. Then, and only then, can SRI's replicate themselves into that location or survive there; in general, because of sheer proximity, these will include the same SRI's that behaved and met the conditions there. In general, also, they will remain there only so long as they, or their ontogenetic products, continue to help meet the conditions there.

ENS: THE EVENT-SET OF NATURAL SELECTION

Each time a certain SRI occurs in a location, and occurs there only because it has behaved in a nearby location, I call that an ENS, an *event-set of natural selection,* of the SRI. An ENS includes, then, (a) a set of ontogenetic events, including the SRI's behavior, through which an SRI helps meet (or maintain) a condition of its existence at some location, and (b) an occurrence of that SRI at that location, i.e. the meeting of all the rest of its conditions of existence there.

I'll give a couple of homely examples of ENS's. First: C1 is a cultural SRI, whose behavior, a carpentry technique, results in a certain feature of building contruction. C1 helps build a house in a certain location, the carpenter lives in that house, and he survives a bad winter. Without the behavior of C1, the house would have collapsed and the carpenter

perished, and C1 would have perished along with him. C1 has ENSed. Second: C2, again a carpentry SRI, helps build a house. A passerby admires the house, and hires the carpenter to build him a boat. He would not have admired the house if C2 had not behaved. With the added income and work to do, the carpenter takes on a new helper. The helper, watching the carpenter at work, is programmed with C2. C2 has ENSed. These two examples, and a comparison between them, illustrate a number of points:

1. In neither case does the carpentry SRI build a house and ENS all by itself. It *cooperates* in an elaborate ontogenetic process with other Carpentry SRI's, with the genetic SRI's that built the carpenter, with SRI's that built his tools, and so on, to build the house. I say these SRI's cooperate because (a) they all go into the house-building ontogenetic process and (b) they all ENS through that process; both (a) and (b) are necessary for cooperation, properly speaking, to take place.'
2. In the first example, ENS of CI occurred through survival of the carrying organism. C I helped build a house for *its* 'house', the carpenter, thus meeting a condition of its existence by altering the value(s) of some variable(s) of the so-called 'natural' or 'physical' environment. That is to say, the initial values of these variables were set by 'nature'. C1 might ENS that way in a wide variety of locations in, say, the so-called temperate zone. It would not ENS that way in, say, the arctic zone, because the house would collapse in spite of Cl's behavior and the carpenter and C1 would perish. It would not ENS that way in, say, the tropics, either, because the house would *stand,* and the carpenter and C1 would survive, even if C I *didn't* behave.
3. In the second example, ENS took place through a different ontogenetic pathway. Here, the behavior of C2 cued certain other cultural SRI's, carried by the passerby; the passerby's SRI's then helped meet certain conditions of existence of C2 at an otherwise 'unreachable' spatiotemporal location; to wit, the brain of the man who became the carpenter's helper. In this ENS, C2 has *exploited* those SRI's carried by the passerby; thus this ENS could have occurred only in a location where someone was carrying those cultural SRI's or, metaphorically, only in a certain 'cultural environment'.
4. If, however, hiring the C2-carrying-and-exhibiting carpenter actually makes the passerby's boat more durable than it would have been had he hired someone else; and if the boat therefore enables the passerby and his SRI's to survive, they too have ENSed, and the relation between C2 and the passerby's instructions is cooperation, not one-way exploitation.
5. An ENS does not an evolution make. Either of the above event-sets could happen just once, or maybe a few times, with C I (or C2, as the

case might be) soon being lost. Or, on the contrary, C1 could already be well established in the cultural repertory of the carpenter's population; in that case, those ENSes would be commonplace, merely *maintaining C1's* frequency.

6. On the other hand, it might be that Cl (or C2) was novel to that population's repertory, having been just recently invented or acquired through observational learning from an alien. Then, if it ENSed more than a few times (along either or both pathways) it would *become* established in the repertory — it would be added to the complete set of SRI's, genetic and cultural, that construct that population and endow it with its traditions and institutions, through the ontogenetic pathways. An event would have occurred in the *phylogeny* of that society. *Evolution* has taken place.

7. Practically any SRI has its effect on the world, and it ENSes, through a cooperative ontogenetic process; the behavioral result of almost any SRI, therefore, is determined not only by its own behavior but also by the behaviors of other instructions entering the process. The behavior of an SRI 'borrowed' from one system by another may thus have a quite unexpected result in the borrowing system; one can *ascertain* the SRI's behavioral result in either system, of course, but one can't *predict* its result, or whether it will ENS, from one system to another.

8. When it helps meet a condition of its own existence (e.g. the survival of the carpenter), an SRI generally helps meet that same condition for other SRI's in its vicinity. At the same time, if a situation of *competition* prevails, it may *unmeet* that (or another) existence condition for still other SRI's (e.g. SRI's carried by the carpenter who would have been hired to build the passerby's boat had C2 not got the job for the carpenter carrying it).

Once established in the repertory, therefore, an SRI, by its behavioral results, alters the *environment* (both 'physical' and 'cultural'), and thus helps to determine whether, and under what conditions, other SRI's — both novel and established — will ENS; and thus helps determine changes in *their* relative and absolute frequencies. So a complete set of SRI's is more than just a collection, it is a complex system of behaving structures in a dynamic balance, the behavior of each having a determining effect on the frequency of many of the others.

SOCIOLOGY

Suppose, now, that some individual acquires a certain cultural SRI novel to his/her population. Suppose, further, that that SRI, S1, is sociogenic;

i.e. its behavior enters into an ontogenetic process constructing/maintaining a group, organization, or institution G, so its behavioral result is some modification in the structure of G. *If S1* ENSes repeatedly, and thus becomes established in the population's repertory, that modification will be present in the structure of many or all groups like G.

For each such ENS, however, (a) S1 's behavioral result must meet some condition of its existence at a location, and (b) the rest of those conditions must also be met there. And behaviors of already established SRI's entering the G-making process are surely important determinants of both (a) S1's behavioral result (point (7)) and (b) the meeting of the rest of the conditions of S1's (and their own) existence (point (8)). In short, the existing sociogenic instructions, process, and outcome — the group itself — determines in large part which novel sociogenic instructions will succeed (ENS) and which will fail, and thus the group (organization, institution) exercises environmental control over the repertories of SRI's of its members and, thereby, over its own evolution."

COOPERATION, PARASITISM, DOMESTICATION

SRI's are not, of course, *inherently* cooperative; ENS refers, with iron necessity, to the ontogenetic process by which an SRI enables its *own* occurrence. To be sure, on this planet the initial environment (the one in the thought experiment) varies through space from somewhat hostile to SRI's to extremely hostile to SRI's to totally unliveable by SRI's. SRI's have progressively occupied more and more hostile territories by cooperating in ever greater numbers and building ever more elaborate organisms, artifacts, and social structures through (of course) the ontogenetic process. So for a couple of reasons, we find mainly 'cooperative' SRI's when we look around. One reason is that they are statistically vastly overrepresented in hostile territory because they ENS there. Another reason is that we detect and know SRI's by their works, and more cooperative SRI's build bigger and fancier works; the solitary parasitic freeloaders among SRI's are hard to detect. And if a parasitic freeloader SRI should mutate and start having some detectable behavioral result, that behavioral result will probably not be neutral. It will either ENS — and that will almost certainly be by cooperating with an existing system — or else it will 'anti-ENS' i.e. be lethal, prevent its own occurrence in a location where it otherwise *would* have occurred, if it hadn't behaved.

But *systems* of SRI's often become actively parasitic on other systems, preying on them, exploiting them, even domesticating them. With genetic systems this is difficult to accomplish and easy to see, because each system constructs and occupies a different organism. With systems of cultural SRI's, on the other hand, exploitation is easier to accomplish and more difficult to see, because elements of several systems occupy the same brains, and elements of the same system occupy different brains. Consider, for example, the cultural SRI (or set thereof) — call it `FC' — that makes its carrier say 'A man's gotta fight for his country' and act upon that aphorism (McDermott 1967). Carried by young American working-class males, FC is an element of the sociogenic system that constructs and maintains the so-called Military-Industrial Complex. Its function in that system is literally to recruit its carriers and ensure their willing participation in the complex's activities. When FC performs its function, behaviors of 'working-class SRI's' — the cultural SRI's that construct and maintain working-class families and neighborhoods and the genetic SRI's that construct and maintain working-class people — result not in occurrences of those working-class SRI's (ENS, in a word) but in occurrences of Military-Industrial Complex SRI's, including FC. In wartime, moreover, because of FC's behavior the behaviors of those working-class SRI's sometimes result in non-occurrences of their carriers and of themselves (and of FC). FC, and through it the Military-Industrial Complex, is indeed a parasite on the working class.

But if FC is at best an exploiter of working-class SRIs, and at worst a lethal, why doesn't it become extinct? How is it maintained in the cultural repertory of the working-class population? The answer, I think, is that the Military-Industrial Complex is not merely a parasite on the working-class system. Rather, it is part of a much larger political and economic system which exercises control over the *environment* of the working-class sytem to the general effect that only workers who carry FC, and other SRI's which build or maintain that political economy, have been able to demonstrate their cultural SRI's to the young; in that environment, noncarriers of FC have usually ended up in breadlines or in prison. In an almost classic sense, the working-class sytem has been taken over and *domesticated* by the political economy, its repertory of SRI's systematically altered to the latter's service.

The structures and relations and behaviors that human scientists are interested in are determined by ontogenetic processes which are in turn controlled by the behaviors of SRI's. Each change in the composition of the set of SRI's that enters into a given ontogenetic process is determined in part by the outcomes of that ontogenetic process and in part by the `environment' — which always includes the outcomes of other on-

togenetic processes. All ontogenetic processes consist, in the final analysis, of the formation and breaking of chemical bonds; so unless human scientists want to get involved with questions of cosmogeny, geogeny, meteorology, etc., they can do their work of scientific explanation secure in the knowledge that they need consider only one spook: Electromagnetism.

NOTES

1. This is a revised version of a paper, of similar title, delivered in May, 1973, at the conference *Son of Fringe: New Directions in Theoretical Anthropology (II),* Carleton University, Ottawa, Ontario.
2. The *behavior* of these scientific spooks is quite well known, of course, and has been described in the form of laws and constants. What remains mysterious (spooky) about them is *why* they exist and/or behave so.
3. The information on variants of the ontogenetic process presented here is now part of the basic repertory of biological science. I acquired it mainly from Monod (1971) and from *Scientific American* offprints: Crick (1954, 1966), Horowitz (1956), Hurwitz and Furth (1962), Penrose (1959), Wood and Edgar (1967).
4. This information, too, is basic biology; I acquired it partly from Eccles (1965) and Kandel (1970).
5. The words 'and so on' here hardly do justice to the elaborate squence of neuronal processes actually involved in any but the very simplest behavior. Most importantly, an observed gross behavior of an organism is practically always the outcome of a whole *series* of behaviors such as are described here, the series functioning to obtain, maintain, or avoid a certain perception and thereby a certain state of affairs in the organism, in its environment, or in the relation between them. Peripheral (sensory) cells thus play two roles in a behavior: (1) they recognize the cue that releases the behavior and (2) they feed information back to the neural system which keeps the behavior going until they inform it that the perceptual situation it controls for is now the case. How such control systems operate, and how they are themselves organized into hierarchies, has been worked out in considerable detail by Powers (1973).
6. To give an example involving culturally-programmed instructions (which follow below), instructions of the first sociogenic ontogenetic sequence bring and/or keep a group of men together in a social group we would call a 'hunting party'; instructions of the second, social gross behavior ontogenetic sequence search for, stalk, wound, track, fix, and kill a large mammal and bring home the meat.
7. This process requires a synapse of a special kind, different from those through which transfer of neurotransmitter takes place, lest the standard routeway be cueing, as well as programming, the incomplete routeway.
8. The programming of a control system routeway (note 5, and Powers (1973)) takes places as follows, if my conjecture is correct: An existing control system operates until perception P (say, a sweet taste) is obtained. At the moment that P is obtained, or a fraction of a second before, perception P' (say color-and shape of a ripe berry of a certain species) happens to be obtained also. The simultaneous firings of the P-recognition routeway and of neurons cued by P' chemically program the latter into a control system that, even after, operates until P' is obtained.

9. The *function* of an SRI, its behavior, or the result thereof, is its contribution to a cooperative ENS. Thus an SRI ENSes and is propagated/maintained in the population only because it performs its function in a *system* of cooperating instructions. For a discussion of how cultural features are shaped to their function, see Cloak (1975: 169-170).
10. This sort of 'downward causation' (Campbell 1974) is precisely analogous to the control exercised by an existing organism - genotype, ontogenic process, and/or phenotype - over its subsequent evolution; since it often results in elimination of a novel SRI, I think it is the material basis of Wilson's (1975) proposed spook, `phylogenetic inertia'.
11. For a more general and theoretical treatment of these matters, see Cloak (1976).

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