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SECTION 5: CYBERNETICS, SYSTEMS, EPISTEMOLOGY, LANGUAGE AND PHILOSOPHY

Trivial and non-trivial machines in the animal and in man

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Abstract

Purpose – It was the aim to apply basic epistemological concepts, as presented by Heinz von Foerster, to current problems of medicine and biology.

Design/methodology/approach – The relation of genes and human behaviour is an important issue in current medical discourse. Many states and diseases are claimed to be caused by a genetical disposition. To prove the soundness of such claims, a strict methodology has to be applied.

Findings – The usual approach of combining genetical findings with observed behaviour is based on an insufficient epistemology. The neglect of recursive processes leads to misinterpretations that have far-reaching consequences, especially if disease and therapy are concerned.

Research limitations/Implications – A precise analysis of recursive traits would allow more reliable models of the relation between genetical disposition and environmental influence.

Originality/value – The paper reflects trivial or non-trivial relations in social behaviour that are often neglected.

Keywords Epistomology, Cybernetics, Behaviour, Genetics

Paper type Conceptual paper

Introduction

Until now the ideas of Heinz von Foerster have had no major influence on medical thinking except in psychotherapy. Nevertheless they could help a great deal to get through the jungle of medical research. As an example I would like to present some relations between behaviour and the genome. Although this is done in a sporadic and rather anecdotic way, it should shed some light on how clear ideas could help to develop more precise concepts that provide more exact results. The pair genome/behaviour has the advantage of being comprehensible to a broader audience than just medical scholars. At the same time it is a well-known topic in social discourse. The term "behaviour" in this context is used as observed "social behaviour", although the expression might denote any reaction of a system. And indeed, the principles of Heinz von Foerster presented here could as well be applied to the side effects of drugs, obesity, course of therapy, multiple diseases, etc. The question of clear concepts is not only a simple theoretical question but also a question of some billion Euros wasted on futile research.

Kybernetes
Vol. 34 No. 3/4, 2005
pp. 508-520

0368-492X
DOI 10.1108/03684920510581693

The situation

In recent years there has been an increasing number of publications proving that behaviour has a genomic cause. The *Science* magazine classified the understanding of this relation as the second ranked breakthrough of the year 2003 with the promising title "Decoding mental illness" (Science News and Editorial Staffs, 2003). But this issue is not new. It has been discussed in psychiatry for many years in the search for the cause of schizophrenia – today correlated with a variety of genes (Harrison and Owen, 2003). However, if we open a medical journal, especially *Molecular Psychiatry*, we find that a lot of behavioural patterns are reduced to genetic causes. Here are some arbitrary examples:

- There is a strong connection between ADHD (attention-deficit hyperactivity disorder – diagnosed according DSM-IV) and the allele 4-repeat of the IL-1 RA gene, coding Interleukin-1 antagonists, whereas the allele 2-repeat is associated with a reduced risk (Segman *et al.*, 2002).
- There is a significant linkage of "simple phobia" to chromosome 14 markers (Gelernter *et al.*, 2003).
- The Grp-gen is regulating "Pavlovian learned fear" by influencing the lateral nucleus of the amygdala (Shumyatsky *et al.*, 2002).
- Individuals with one or two copies of the short (s-) allele of the 5-HTT gene are much more likely to develop depression than individuals with the long (l-) allele when confronted with emotional stressors between the age of 21 and 26 (Caspi *et al.*, 2003).

Of course this is the resumption of the old discussion whether behaviour is inherited or acquired. Already in 1960s fierce quarrels erupted on this subject. Results have been forged (Di Trocchio, 1993), professors have been beaten, in short: real life roared around this issue.

Since then, some decades have passed, and science has made incredible progress. Not only has the genetic research made progress, but knowledge and understanding of (self) organisation of life have been successful as well. It was especially Heinz von Foerster and the members of his Biological Computer Lab (BCL), Maturana, Ashby and Günther, just to name a few, who influenced these ideas. Revisiting some of these ideas gives us the tools to judge whether the findings of genomic behaviourism are sound and to what extent they can be helpful as to the understanding of the mechanisms of disease.

Inherited or acquired?

The ant *Pheidole kingi instabilis* is characterized by a large variety of forms. The morphology of each animal depends mainly on its function. The whole population is interconnected by exchanging stomach content, a process called *trophylaxis*. In this way some substances are distributed among the whole population. As a result, some information about the composition and situation of the population is provided to all ants. The group has a certain kind of "consciousness" of itself. Removing the queen provokes a reaction in the other ants: The workers feed a special nutrition to some larva in order to raise a new queen (Maturana and Varela, 1984).

In an article with the remarkable title "Identification of a Major Gene Regulating Complex Social Behavior" the authors show that a difference in the Gp-9 genotype of the fire ant (*Solenopsis invicta*) is responsible whether one or several queens are raised (Krieger and Ross, 2002).

These short examples of the world of ants are enough to show the complexity of our issue: The status of a queen is acquired, not inherited. However, it is genetically determined that a queen has to exist. This genetic determination is to be found in the workers and not in the queen herself. Whether or not a larva becomes a queen depends on environmental influences and is stochastic.

It is amusing to directly translate this into human behaviour: It is not very likely for a person to become the leader of a country. One does not need any special genes, one must only be fed properly.

The more serious consequences of this short, but not superficial look on the organisation of ants shows that there is no clear distinction between genetics and environment, that genetic determinations are sometimes more easily changed than acquired characteristics, that a broad social context can be responsible for the morphologic expression of a genetic possibility, etc.

If we look for comparable situations in mankind we find it, e.g. in the acquisition of language. Up to the age of 12-14 the syntax of (native) language is easily learned. If this first acquisition does not happen (i.e. in the deaf), it is impossible to recapture it later (Sacks, 1989). It is, without doubt, a critical age. Afterwards (or even earlier) someone is French, English, German or Greek. Noteworthy is that the prognosis of ADHD beyond this critical age is considered as bad (Lösslein and Eike-Beth, 2000). It is foremost a question of definition to call it a morphological consolidation or a reduced plasticity of the brain. Adolescence does not only close a chapter, it opens up a new one with other topics and with an increased neuronal plasticity for them (McCrone, 2000).

Social and sexual themes become predominant. Also schizophrenia is known to not start until adolescence. However, "clinical studies show that patients with schizophrenia manifest minor behavioural abnormalities in childhood even before the onset of schizophrenia" (Sawa and Kamiya, 2003).

What happens in adolescence? Are some genes switched on and suddenly produce schizophrenia? Or do they become more important as minor changes have already been noticed earlier? Does the environment prevent the development of a coherent personality by incoherent communication (Bateson, 1972; Selvini Palazzoli *et al.*, 1989)? But why after adolescence? Do we have to live with such vague terms such as "multi-factorial disease", actually confessing ignorance?

Furthremore, plasticity of the brain might resume to some extend even in the elder people after suffering a stroke (Kluska *et al.*, 2004). That is, strong influences might reverse morphological consolidation.

Recursivity and behaviour

Recursivity is the foundation of biological organization (Maturana and Varela, 1984). Stable behaviour – *eigen-behaviour* as von Foerster called it (Figure 1) – can be understood by analysing recursive processes.

The acquisition of language is, again, a suitable example. Language is learned through a recursive process. Meaningless sounds of infants are reinforced, giving them simultaneously an intersubjective meaning: "mamamama" – "Oh yeah, say 'mama'"; or: "This is a spoon" – "ooon" – "Very good, spooooon" – "ooon". The stable eigen-behaviour is the common language.

The acquisition of language does not necessarily follow this pattern. Children cannot be kept from learning a language even if there is no reinforcement. But they

Recursive processes use the result of an operation for the same operation (von Foerster, 1984, illustration by von Foerster). If the result of a first operation $x_1 = Op(x_o)$ is processed again with the same operator $x_2 = Op(x_1)$ it leads to the formula

$$\lim_{n \to \infty} Op^{(n)} = Op_o$$

The result of the limes operation might be endless, bi- or multistable. It also might lead to a definite stable value which von Foerster called *eigen-behaviour*. The eigen-behaviour of a recursive operation does not depend on the operand but on the operator.
Von Foerster described it by extracting the square root. Irrespective of the starting number (n>0), the result will always be 1.

Note: Drawing by Heinz von Foerster

Figure 1. Recursivity and eigen-behaviour

need to be in communication. Just hearing a language without recursive interaction does not show any effect (Kuhl *et al.*, 2003).

A striking example is the children in a deaf school in Nicaragua who developed their own sign language with its own grammar (called a "big bang of language") just communicating with each other. The only thing needed to generate a language is enough people who communicate. A deaf child in a family without a supporting system is not able to do this (Breuer, 2000). It stays without the possibility of complex thinking. Deaf children remain in a state of debility if they do not learn to communicate (Sacks, 1989). The same is true for totally neglected and deprivated children that had no "social input" (Zimpel, 2005).

It is genetically determined to learn a language. Through the interaction with the environment a certain language is learned. It might as well be a sign language. Lately, Chomsky *et al.* gave a more specified concept of language and communication taking recursive elements into account. They differentiate between the faculty of language in a broader sense (FLB) and the faculty of language in a narrower sense (FLN). FLB is the faculty of communication, which is common in all animals and does not change through social contact. FLN is a recursive product with open end possibilities and as such only existing in human beings (Hauser *et al.*, 2002). Hauser and Fitch showed in experiments with monkeys that it is exactly the lack of recursive action that limitates the development of language in animals (Fitch and Hauser, 2004).

One of the first to analyze the recursive interaction of communication was Bateson. With his concept of *schismogenesis* he demonstrated that severe problems arise if a certain kind of interaction remains unchanged (Bateson, 1972, pp. 61-72). Fights are often not caused by objectives but are a result of a certain way of handling the objectives (Ivanovas, 2003). These models have been refined by systemic psychotherapy, showing, e.g. how whole families produce certain behavioural patterns just by repeating defined strategies (Haley, 1963). Mostly only one member of the family is symptomatic (identified patient). The processing of a symptomatic member has some similarities to the ant colony, which is bringing forth a queen:

- the illness of the identified patient is an expression of a social context; and
- when the symptomatic member is removed often another member becomes symptomatic.

Solutions in such situations are rarely attained by changing the objectives (operands) but through change of the structure of communication (the operator).

The model suggests that minor "behavioural abnormalities" before adolescence might well lead – through an unchanged communicative operator – to severe disturbances of social behaviour, e.g. schizophrenia (Selvini Palazzoli *et al.*, 1995).

This does neither exclude a biological basis for the behavioural abnormalities, nor does it exclude that the constant interaction produces brain dysregulation, e.g. oligodendrocyte dysfunction (Tkachev *et al.*, 2003). Recursive processes cannot be described in such a linear way.

A more precise analysis is necessary.

Trivial and non-trivial machines

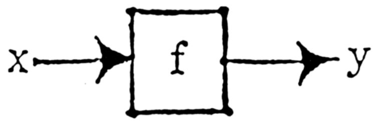
The structural connection between genes and behaviour can best be described with the concepts of trivial and non-trivial machines as presented by von Foerster (Figures 2 and 3, illustrations by von Foerster). The term "machine", introduced by Alan Turing, means a set of rules and laws how a certain state is transformed into a different state (von Foerster, 1993, p. 135).

FLB as defined by Chomsky would be a trivial machine, FLN a non-trivial machine.

A trivial machine is as well *imprinting* as first described by Konrad Lorenz (Lorenz, 1949): The baby duckling will follow the first moving object it sees at the moment it slips out of the egg. This is normally the mother. Therefore, throughout its childhood it walks behind its mother. If it sees first Konrad Lorenz it will follow Konrad Lorenz, and if it first sees a moving football, it will spend a lot of his lifetime walking behind a moving football. Imprinting is a trivial machine with just one recursive loop. Not understanding this recursive loop will lead to the following fallacy: Imprinting is genetically determined. If behaviour (falsely) is defined as "following the mother", there are these unexpected situations when it follows Konrad Lorenz. In this case the genes will not code for the expected behaviour. This is called *penetrance*. If the correlation gene-behaviour is high as in some kinds of haemophilia, the penetrance of the gene is high. If the correlation is low, then the penetrance is low. Low penetrance is normally expected when the maternal and paternal genes differ or when a behaviour is coded by many genes, thus producing a non-trivial machine. What should be demonstrated here is that a seeming "low penetrance" might be nothing else than the misunderstanding of one single recursive loop. In the duckling the behaviour "following a moving object" is 100 percent genetic, the behaviour "following the mother" is 100 percent acquired. Through a slight change in the definition the inner logic is turned upside down, although the observed process remains the same.

But recursivity can also be misunderstood the other way round.

A trivial machine has a simple mechanism: A certain operation (*f*) is done with an operand (*x*). The result is (*y*).



Because (*f*) is determined, the result is determined and predictable (von Foerster & Poerksen, 1999, p. 57). If we insert a coin in a chewing gum machine, we get a chewing gum, not one time a peppermint, next time a condom.

Trivial machines are

- synthetically deterministic,
- history independent, i.e. every following operation is according to the operation before,
- analytically determinable, i. e.knowing some *x* and *y,* we can reconstruct the operator *f*,
- predictable.

Note: Drawing by Heinz von Foerster

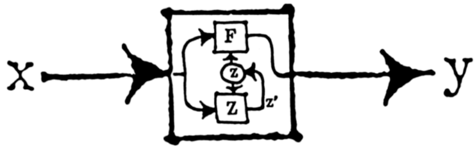
Figure 2.
Trivial machines

The raising of the queen in *Pheidole kingi instabilis* or the queens in *Solenopsis invicta* is a trivial machine. Nevertheless, from the viewpoint of the larva ("Who is going to be queen?") it is a stochastic process that cannot be predicted. But the result is determined. The environment only regulates the formalities. There are no recursive loops. If there are several queens as in the species *Formica fusa*, the workers prefer to feed the brood (larva and queen) they are genetically more related to (Hannone and Sundström, 2003). This is not a recursive process by itself, because it does not change the behaviour of the queen, the workers and the larva. A recursive effect could only be seen through generations, if according to environmental conditions the reproductive share of one queen increases. This preference of the own brood was called nepotism. But nepotism ("one hand washes the other") is a highly recursive process, which continuously changes the behaviour of all people concerned. It might have a stabilizing effect in chaotic social situations (e.g. post war) or a destabilising effect in stable conditions (mafia), it might lead the partners to the highest positions or to prison. The outcome is not foreseeable. It is as non-trivial just like the cooperation of scientists.

Trivializing non-trivial machines

Heinz von Foerster always maintained that man is a non-trivial machine. His well-known example is that of a child answering the question "How much is 2×2"

In non-trivial machines an internal logic changes the operator with every operation (von Foerster & Poerksen, 1999, p. 58). If only one step is missed by an observer, the reaction of the machine becomes unpredictable, even if the principle of the program is known.



Such a machine is
- synthetically deterministic, as it can be constructed easily,
- history dependent, because every operation changes the operator,
- analytically indeterminable, because of the non-linear equations
- analytically unpredictable.

Note: Drawing by Heinz von Foerster

Figure 3.
Non-trivial machines

with “green”. Such a child, he said, would be sent immediately to a “trivializing institution” thereafter giving the “right” answer (von Foerster, 1999).

What was initially thought of as more anecdotal – von Foerster, as he told me, was unaware of the clinical picture of *synaesthetics* – which turns out to be an exact description of how complex behaviour is trivialized. *Synaesthetics* is a state of perception where different sensual qualities are inseparably connected, shapes have colours, names have odours etc. One of these children during first grade indeed answered the question “How much is 1 plus 1?” with “dark green” (Schneider, 2003).

Synaesthetics is said to be quite common if not ubiquitous in early childhood before a more social perception is adopted (Hackenbroch, 2000). This corresponds to the finding that the infant’s ability to discriminate among native speech sounds improves, whereas the same ability to discriminate among foreign speech sounds decreases (Kuhl *et al.*, 2003). Similarly six-month old infants are equally good in recognizing facial identity in humans and non-human primates. Something they have lost at the age of nine months (Pascalis *et al.*, 2002).

Early childhood is characterized by a great variety of non-trivial, non-linear, non-formed behaviour that brings forth a stable social behaviour only through recursive interaction. This is accompanied by a stabilization of neuronal brain organisation. Biology does not discriminate between structure and function. Function changes the structure and structure shapes function. The genomic foundation only provides a very wide framework. Trivialization is the necessary condition for the development of social behaviour.

However, this organization is not fixed as brain research of the last years showed (Gross, 2000). This corresponds to the observation of Keeney (2005) that synaesthetic perception can be achieved by certain rituals.

Perception can hardly be called "learned" or acquired. But its organization is much more flexible than we had expected. It is based on recursive social interaction.

The genome is not a trivial machine

Until here we assumed that the genome is a trivial machine producing trivial machines and creating a frame for non-trivial machines. But this is not the case.

Epigenetics is the slogan, which is shaking old beliefs.

In the last years there has been more and more evidence indicating that acquired characteristics can be inherited. One mechanism is *genomic imprinting*, where one parental gene finds a mechanism to express itself more then the other gene (Clayton-Smith, 2003).

But even more it seems that genes are "only marionettes in the hands of enzymes, turning them on and off" (Kaati, 2002, my translation). The mechanism of inheriting acquired characteristics is due to the small RNA (sRNA) that can activate, deactivate or even cut off parts of the genome. This discovery was classified as "The Breakthrough of the Year 2002" by *Science* magazine (Couzin, 2002).

Epigenetics has been proved in the animal and in humans.

It is known that a given genotype can give rise to different phenotypes depending on environmental conditions. These responses to the environment may be expressed in the offspring rather than in the parent and might persist across a number of generations, even if the environmental factor itself has altered (Bateson *et al.*, 2004).

For example, the nutritional status of grandfathers before adolescence (!) has been found to be of major influence for the health of their grandchildren. This is not due to genetic causes, as a period of famine for example, which is a pure environmental factor, has a health protective influence in grandchildren (Kaati *et al.*, 2002).

The genome is not a fountain bringing forth life. It turns out to take part in the process of self-organization.

Research in genomic behaviourism

All this has a major impact on the outcome of genomic research on behaviour. The reliability of this kind of research has been proven to be low. The results of one group can rarely be confirmed by those of another group. Normally technical problems are held responsible (Colhoun *et al.*, 2003). But the misunderstanding of recursive processes is another main factor.

One hint in that direction is that the correlation genome-behaviour is higher in smaller studies than in larger studies (Ioannidis *et al.*, 2003). In general, smaller groups are more homogenous in their behaviour than large populations. What does this mean in terms of recursivity?

If we take a small traditional village in the Cretan mountains, the behaviour of the inhabitants, sexual self-image, expression of aggression, etc. is very homogenous, at least to the foreign observer. This is an acquired pattern as such a person changes behaviour going to the city, especially if this is done in early life. The situation is similar as in language. According to Chomsky we could postulate a "faculty of behaviour in a broader sense" (FBB), i.e. the possibility to interact, to acquire a language, to express emotions, to mate etc., and "a faculty of behaviour in a narrower sense" (FBN), i.e. the human possibility to produce a large variety of social contexts. In contrary to the duckling that has a restricted number of recursive loops, human FBN has many recursive loops that sometimes are restricted after adolescence. Investigating a small population creates the delusion that a certain kind of behaviour is stable out of biological reasons (holding it for FBB). In such a stable situation the correlation gene/behaviour might be quite high. However, it is only an effect of the frame of observation.

This is comparable to laboratory research, where variable factors are held stable in order to investigate some traits of a system. This typical reductionist approach enables to apply the concept of cause and reaction. The relation, however, will be true under these circumstances only. Unexpected behaviour will arise over and over and it will be labelled with expressions as "low penetrance of genes".

The other similarity to laboratory research is that all studies investigate the relation of *one* gene for a defined behaviour. For example, different studies show a relationship between the genome and schizophrenia, but always for a different gene (Harrison and Owen, 2003). As the studies are designed to find a trivial relation of cause and effect, they can only provide tendencies. But, since recursive processes are involved, they cannot show any kind of structural interconnection between genome and behaviour.

The problems, which are arising as a consequence shall be demonstrated with the study on depression (Caspi *et al.*, 2003): Individuals with the s-allele of the 5-HTT gene only become depressed when confronted with emotional stressors. They develop twice as often depression after having suffered from four and more stressing events. The study investigated stressors like employment, financial, housing, health and relationship. It was found that the tendency to develop depression increases with every stressing event. This is not true for individuals with l-allele. They only hardly change, if at all, even when repeatedly making painful experiences. The l-type shows (according to the criteria of the study) a trivial reaction to environmental influences (as the reaction is always the same), the s-type a non-trivial behaviour (as it changes with every new experience). This is not surprising. We all know that some individuals are sensitive and vulnerable and others are indifferent and indolent. It only would have been a surprise, if these different constitutions had no genomic differences. Now, the only conclusions allowed is that the 5-HTT gene is connected with emotional processing. (But it is involved in many other tasks as well, as it takes part in the regulation of the reuptake of serotonin at brain synapses.)

The study investigates one traitout of a complex pattern: the sensitivity for depression. It comes to the conclusion that (in a society with certain undefined values) a sensitive person is more inclined to become depressed when some defined values (job, money, spouse, health) are in question. This finding can be formulated differently: A society with certain values brings forth depression in its sensitive members. It could be well imagined that sensitive persons in a different society or with a different study design will instead of getting depressed get religious. May be then 5-HTT would turn out to be the compassion gene.

All these are rough hypotheses. But the expectation that a gene through its impact on metabolism brings forth a certain behaviour is a rough hypothesis as well.

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However, there is a great danger when such trivializing definitions of behaviour become the starting point of therapeutic interventions. This is obvious in the case of psychotic experience, where the vicinity to creativity is a well-known fact and the list of people who have been diagnosed as manic-depressive reads like a who's who of Western art (Jamison, 1993, pp. 267-70).

> Excerpt of a list of people having been diagnosed manic-depressive:
>
> Hans Christian Andersen, Antonin Artaud, Honoré de Balzac, Charles Baudelaire, Irving Berlin, Hector Berlioz, Anton Bruckner, Lord Byron, Paul Celan, Joseph Conrad, Noel Coward, Charles Dickens, Emily Dickinson, Isak Dinesen, Ralph Waldo Emerson, T.S. Eliot, Edward Elgar, Georg Friedrich Händel, William Faulkner, F. Scott Fitzgerald, Paul Gauguin, Vincent von Gogh, Nikolai Gogol, Maxim Gorky, Ernest Hemingway, Hermann Hesse, Hölderlin, Victor Hugo, Henrik Ibsen, Henry James, William James, John Keats, Ernst Ludwig Kirchner, Heinrich von Kleist, Otto Klemperer, Wilhelm Lehmbruck, Malcolm Lowry, Gustav Mahler, Wladimir Mayakowski, Herman Melville, Michelangelo, Charly Mingus, Modest Mussorgsky, Edward Munch, Eugene O'Neill, Charly Parker, Boris Pasternak, Cesare Pavese, Sylvia Plath, Edgar Allan Poe, Jackson Pollock, Cole Porter, Ezra Pound, Alexander Puschkin, Bud Powell, Sergej Rachmaninoff, Gioacchino Rossini, Robert Schumann, Alexander Scriabin, Mary Shelley, Robert Louis Stevenson, August Strindberg, Torquato Tasso, Lord Tennyson, Dylan Thomas, Leo Tolstoi, Georg Trakl, Peter Tschaikowsky, Ivan Turgenjew, Walt Whitman, Tennessee Williams, Virginia Wolff, Emile Zola.

Intervening in the basic organisation of the serotonin metabolism (what is current practice) or gene regulation (what most likely will be done) might lead to far reaching consequences on creativity, compassion and other central human values. These consequences cannot be proved or disproved as they do not show up under the conditions of study designs. The trivialization of human behaviour might be the price for the benefit of a few.

Some conclusions

Understanding recursivity. Results of research are reliable only if recursive processes are properly understood. Otherwise unexpected behaviour of systems arises. It is not sufficient to draw some circles in diagrams or simulate them on a PC. It must as well be understood whether a process is trivial by determination or trivial by its recursivity. In the latter even a minimal change can provoke far-reaching consequences.

Relation and correlation. The relation between the genome and behaviour can be like a rigid trivial machine (one or several queens have to exist), or a trivial machine with one to multiple recursions (imprinting, acquisition of language), or a non-trivial machine (nepotism in humans). But as soon as one recursion takes places, the correlation genome-behaviour depends on our definition of behaviour (following the mother versus following a moving object). However, the idea of relation is not inherent in the observed data. It is a consequence of a theoretical construction applied to the observed data (as imprinting). There might be a strong relation but a loose correlation and vice versa.

Number of loops. The more recursive processes are genetically foreseen or the less trivial these processes are, the lower is the correlation genome-behaviour, although the relationship itself does not change. The process might be 100 percent determined, but the outcome cannot be predicted.

Trivialisation of behaviour. In a stable frame of perception recursive functions might seem trivial. A high correlation genome-behaviour might be thought of as a trivial relation. The sharp rise of ADHD the last decades this cannot be explained by the allele 4-repeat of the IL-1 RA gene. A high correlation with ADHD only means that it might be of some importance under these circumstances, not that it causes the disease. In fact the correlation is only significant, i.e. small (Segman *et al.*, 2002).

The way down is not the way up. It is always possible to analyse an organism from behaviour down to the genes. But it does not work the other way round if recursive processes are involved. Already Turing showed that it is impossible to decide in advance if there are endless loops in a program (Guerrerio, 2002). What we experience every time we use a new edition of Windows is exactly the same in trying to predict behaviour from the genome. Günther proved that in complex, non-trivial ("polyvalent" as he calls it) situations the Aristotelian logic is no longer accurate and that the classic principle "the way up is like the way own" (οδός άνω κάτω μία) cannot be applied (Günther, 1972).

Resuming all these reflections the hope of some scientists that "although the road ahead is long and winding, it leads to a future where biology and medicine are transformed into precise engineering" (Kitano, 2002) will be disappointed. It is not in line with the fundamental principles of organisation.

My conclusion is that the reliability of genomic behaviourism will always be low.

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