# Notes on Relational Biology and Elementary Category Theory

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**Abstract.** This paper is the first step in an exploration of the mathematical modelling of autopoiesis, using Robert Rosen's $M$-$R$ system as a central reference point. Category theory is introduced only informally in order to render the material more accessible to a wider audience. Rather than breaking any new ground in terms of mathematical results the paper aims to contextualise the concepts discussed within digital ecosystems and autonomic computing research.

**Keywords:** $R$-$M$ system, autopoiesis, digital ecosystems, autonomic computing, mathematical biology

## 1 Introduction

Software systems can be open to information flow in a manner that is similar to how biological systems are open to a flow of materials and energy. The physical substrate and laws that drive the construction of order in biology, however, are absent in software. Therefore, one cannot speak of a software system as being 'far from equilibrium' in the Prigogine sense:

> The destruction of order prevails in the neighbourhood of thermodynamic equilibrium. Creation of order *may* occur far from equilibrium provided the system obeys to non-linear laws of a certain type. ((Nicolis & Prigogine 1977): 25) [emphasis in original]

Luckily, the activity of the users provides an information flow that is somewhat analogous to the sun's energy driving the biosphere. However, how the non-linear character of the interactions that appears to be so important to organised behaviour could be replicated is not immediately apparent.

Just as the sun's energy generates order as it is transformed hundreds of times while trickling down to high-entropy matter – which is then reabsorbed by the roots of plants – so we would like the users' activities and inputs to be propagated through cyberspace causing useful structures to form and useful behaviour to emerge spontaneously. With present-day technology useful behaviour in software can be triggered in a similar manner, but will actually occur only if it has been explicitly and deterministically specified in advance. In our view spontaneous order construction or 'autonomic behaviour' can only happen if among the possible state changes induced or triggered by the propagation of these signals we 'channel' system behaviour in particular directions that are conducive to what we perceive as 'order' and not in others. Thus we need to understand what constraints to impose locally in order to induce globally ordered behaviour.

Order construction in nature results, ultimately, from the regularity of physical laws and the stability of atoms. Anthropocentrically, we perceive such order as recognisable and repeatable patterns, which therefore acquire subjective and context-dependent informational value and content. They 'mean' something within and relative to a particular environment composed of similar patterns, among which ourselves. The fundamentally relativist nature of 'order', therefore, highlights the equally fundamental interactive character our model of computation must implement if it is to support distributed and spontaneous order construction processes. The apparently unavoidable interactive and non-linear character of the dynamic architecture of ecosystems points to the importance of a dynamical systems perspective in the development of the mathematical modelling of this class of phenomena.

If we focus on the 'digital' nature of cell biology, we cannot help noticing the surprisingly discrete character of many of the processes and phenomena inside the cell as enabled and mediated by a physical substrate that relies on interaction forces, entropy maximisation, and so forth to realise certain specific 'algorithms'. When dealing with software systems, on the other hand, the physical substrate is radically incongruous and independent of the computation being performed. In fact, this is by design: the 'general-purpose' computing machine is supposed to be able to compute anything with the same architecture: the engineering principle is abstraction and making the layers independent of each other. There is no causal

link between a particular state transition and the physical laws of the hardware components, beyond their generating and mediating the voltage levels that encode an arbitrarily chosen binary alphabet. An example of the converse of this situation are the old analog computers, whose computational functions were entirely dependent on 'analogous' electronic phenomena within their components. So in this sense computation with today's computers does not have to obey any conservation laws. It is independent of the physical constraints of the physical substrate that makes the computation possible. These constraints are present at a lower level of abstraction and manifest themselves for instance as clock speed rather than in favouring a particular algorithm over another.

The fact that software does not obey fundamental physical laws in the above sense does not stop us from enforcing an organisation of patterns, if we so wish, that is analogous to what results from the physical laws that underlie the digital biological behaviour we would like to emulate. The study and formalisation of patterns is fundamentally and effectively done by mathematics and in particular by algebra. Therefore, the study of the construction of order needs to integrate a dynamical systems perspective with algebra. The theory that addresses and achieves this integration are the symmetry methods or Lie group methods applied to the solution of systems of coupled non-linear differential equations. The challenge in bio-computing research is to translate these results from mathematics and physics into a language appropriate to computer science, i.e. automata theory and logic.

Our current and recent work (Dini & Berdou 2004, Dini 2007, Dini & Schreckling 2007a, Dini & Schreckling 2007b, Dini, et al. 2008a, Dini, et al. 2008b, Dini, et al. 2008c) started from a perspective informed by statistical physics and non-linear dynamics and has gradually migrated towards a perspective informed by algebra. We are currently investigating these questions from two points of view: the formalisation of cell metabolic and regulatory pathways through algebraic automata theory, which we can call a local perspective; and the characterisation of the mathematical properties of the cell as a whole through category theory, which we can call a global perspective. The local perspective is very much inspired and guided by the work of Nehaniv and co-workers (Egri-Nagy & Nehaniv 2008b, Egri-Nagy & Nehaniv 2008a, Egri-Nagy, et al. 2008, Dömösi & Nehaniv 2005, Nehaniv & Rhodes 2000), which builds on the original work from the 1960s on the prime decomposition of transformation semigroups (Krohn & Rhodes 1965, Krohn, et al. 1967). We are only at the beginning in the building of a global perspective. In this paper we summarise informally some of the pioneering work of Robert Rosen (Rosen 1958a, Rosen 1958b, Rosen 1959, Rosen 1972b). We have recently become aware of the work of Cornish-Bowden and Cardenas (Cornish-Bowden & Cardenas 2008), as well as of Nomura (Nomura 1997, Nomura 2006, Nomura 2007), both of which are well ahead of the material presented in the present article.

The material on Lie groups of differential equations for the solution of low-dimensional non-linear dynamical systems is a third strand of activity that appears to be tantalisingly close to the other two, as exemplified by the algebraic structure of DNA (Sanchez, et al. 2006). This work was only begun in the DBE project (Dini 2007) and its continuation is planned for future projects and publications. It is quite interesting that the algebraic object 'Category' is almost identical to the algebraic object 'Transformation Semigroup' (or in fact Monoid), and that the latter as the mathematical representation of automata has actually a lot in common with discrete dynamical systems, which are also amenable to symmetry analysis. Thus, a general and comprehensive mathematical framework for the unification of cell biology with new models of computation is starting to look feasible.

The next section begins to address the global mathematical character of systems that exhibit self-organisation.

# 2 Autopoiesis: Narrowing the Problem

The (descriptive) theory of autopoiesis is concerned with providing a conceptual framework for understanding the organisation of living things. Its emphasis is therefore on the characterisation of whole systems rather than on the structural decomposition and analysis of individual parts. Table 1 provides a few definitions that we may refer to in the rest of this report (Maturana & Varela 1998).[1]

In the 1960s the work of several mathematical biologists whose ideas were quite compatible with autopoiesis acquired a greater visibility partly because of the general growth of the system theory or 2nd-order cybernetics movement. 1st-order cybernetics was concerned mainly with control. 2nd-order cybernetics acknowledged that the observer, or perhaps the 'controller', was an important part of the

[1] A much more extensive discussion of the biological and conceptual basis of autopoiesis can be found in the OPAALS deliverable D1.2 (Dini et al. 2008b).

| Term | Definition |
|---|---|
| **Autopoiesis** | A type of dynamic organisation characteristic of living organisms that allows them to be continually self-producing |
| **Natural drift** | A consequence of organisms and their environment interacting and affecting each other through structural coupling. A view of evolution that gives similar weight to environment and species, in contrast to the prevailing view of the environment as the more independent cause of the evolution of the species through natural selection. |
| **Operational closure**<br><br>**or**<br><br>**Organisational closure** | A system organisation such that the system identity is specified by a network of dynamic processes whose effects do not leave that network. More properly, 'operational closure' is defined by Maturana and Varela as a concept common to first-order autopoietic systems, i.e. the cell, and second-order autopoietic systems, i.e. multi-cellular and 'meta-cellular' organisms: we shall leave open the question of whether or not metacellular systems are first-order autopoietic systems. What we can say is that they have operational closure in their organisation: their identity is specified by a network of dynamic processes whose effects do not leave that network (Maturana and Varela, 1987: 88-89). Of course, this concept can only be an idealisation since the cell or any multi-cellular system is coupled to its environment in every way. Perhaps a safer statement is to say that a good part of the effects, although not all, participate in maintaining the system's organisation. |
| **Structural coupling** | A form of interdependence between two actors or entities that satisfies the criterion of structural determination mutually and symmetrically. Conceptually similar to non-linear coupling in physics. |
| **Structural determination** | A process of change of an organism that, at any point in time, is determined by the organism's previous structure but is triggered by the environment. The same holds for the environment: the organism is a source of perturbations and not of instructions. |

Table 1: **Definitions of some Autopoiesis terms**

system. This implied that the system could not be examined just by decompositing it into its constituent parts, because by so doing one would lose the interaction with the observer.

Put simply, the difference is similar to that between killing an animal in order to dissect it and study its anatomy, and feeding or poking or stroking the same animal in order to understand its behaviour. In the second case the observer is interacting with the 'system' under study. Clearly, as this example suggests, one observes different things depending on the approach used.

The work of these mathematical biologists acknowledged the importance of interactions. For example, people like Robert Rosen drew a distinction between structural decomposition and functional organisation. Taking inspiration from Rashevsky and von Bertalanffy,[2] he started elaborating a mathematical theory of relational biology, by applying what was already known then and is still known today in abstract mathematics as Category Theory.

# 3 Structure and Function

To explain the difference between the reductionist and the systemic viewpoints, Rosen (Rosen 1972a) compares the physico-chemical approach, which since the discovery of the structure of DNA in 1953 had been hugely successful at understanding the inner workings of the cell, to his new approach:

> The first step in conducting any structural study of a biological system is to abstract away the organizational properties of the system, leaving behind a purely structural residue to be studied entirely in structural (that is, physicochemical) terms. ((Rosen 1972a): 219)

He then notices that

> systems of the utmost structural diversity, with scarcely a molecule in common, are nevertheless recognizable as cells. This indicates that the essential features of cellular organization can be manifested by a profusion of systems of quite different structure. (Ibid)

[2]Nicolas Rashevsky was a mathematical biophysicist active in the mid-20th Century and PhD supervisor of Rosen (Rashevsky 1960). Ludvig von Bertalanffy was one of the main proponents of General System Theory (von Bertalanffy 1969).

And, speaking about his research, concludes,

> What we shall do, in effect, is to begin by *abstracting away the structure* (that is, the physics and the chemistry) of the system, leaving behind only the functional organisation, which then can be characterized and studied abstractly. (Ibid) [emphasis in original]

'Structure' is one of those overused words that in interdisciplinary dialogues can cause no end of confusion. Although he is a mathematician, Rosen uses the word structure in very concrete biological terms, as in the shape, extent, and material composition of different cells. In mathematics, on the other hand, 'structure' can mean something much more abstract, as in 'algebraic structure' or 'smooth manifold structure'.

Our work in applied mathematics is very much based on using abstract mathematical structure to help and to guide our intuition for unravelling how biological processes might work. This mathematical structure can also be referred to as 'regularity' or, somewhat more loosely, as 'symmetry'. We are particularly interested in the structure of dynamical systems and processes, which almost sounds like a contradiction in terms if one uses the more concrete meaning of 'structure'. We must therefore be careful to pay attention to the context in which this term is used.

## 3.1 Conceptual Model of the Cell

We stress that what follows is mainly a *conceptual* discussion, even though as the paper progresses the language becomes increasingly mathematical. In this early stage of our research we are more interested in developing new perspectives in the conceptualisation of biological systems and of their possible 'homomorphisms' to computer science constructs than in achieving mathematical consistency and rigour. A more rigorous mathematical treatment will need to follow once the theoretical framework has come into better focus.

Rosen argued that since the lifetime of the cell by far exceeds the lifetime of any of its parts, the cell must implement a mechanism to repair itself (Rosen 1972a). As expressed in a much more recent article,

> According to Rosen's theory the fundamental characteristic of a living organism is its ability to conserve its integrity of organization in spite of changes in its environment and in spite of the finite lifespan of all of its components. This capacity for autoconservation raises a major theoretical problem, because in present-day organisms the degradation and resynthesis of components involve the action of a series of interdependent macromolecules, which depend in their turn on another series, and so on, so generating a problem of infinite regress. (Cornish-Bowden & Cardenas 2008)

This problem is addressed by Rosen in abstract mathematical terms, and by Cornish-Bowden and coworkers in a more broadly accessible way.

We notice that that the cell is made of two parts: the cytoplasm and the nucleus. We regard the cytoplasm as the site where the metabolic (in truth, also catabolic) activity takes place, whereas the nucleus is the site that implements (among other things) the repair mechanism. The cytoplasm and the nucleus can be seen as two separate but coupled input-output systems. In point of fact, this is not entirely correct. Whereas the metabolic system can indeed be regarded in this way, as shown in Figure 1, the repair system is a little different.

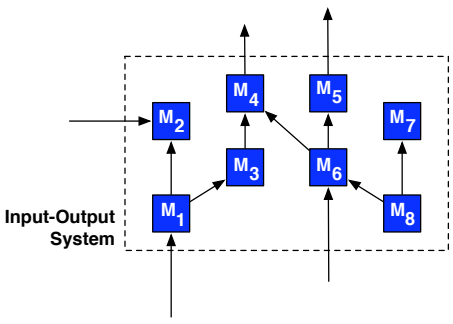


Figure 1: **Block diagram of an input-output system**

Figure 2 (Rosen 1972b) shows that the repair system consists of components (red circles) each of which is paired to a metabolic component. Of course in nature things are rather more complicated, but notice how even the simple block diagrams employed here can already express a fair amount of complexity.

For example, the signal that $M_6$ should be rebuilt does not come from $M_6$ itself, but from $M_5$. Such a signal does not have to follow from a 'call for help' from $M_6$, it could arise at some time after $M_6$ has stopped functioning. It could be caused indirectly by this fact, or it could simply be something $M_5$ does periodically. In any case, notice that in this particular model the $R$-components do not depend on each other, something that's different to the $M$-components. In other words, epigenetics, which is concerned with dependence between genes, does not appear to be modelled by this theory (might be a possible extension thereof).

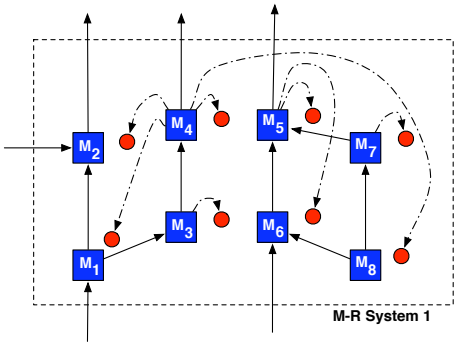


Figure 2: **Block diagram of a metabolism-repair system**

The repair function relies on the continual synthesis of basic units of metabolic processing (enzymes), in response to inputs provided by the metabolic activity itself. The inputs are shown as curved 'dot-dash' arrows in Figure 2, which in our extremely simplified model could be seen to carry a 'stress signal' from a damaged $M$-component to its $R$-component, or as we said more generally from any $M$-component to any $R$-component whose job is to replace its $M$-component with a new version.

Figure 3 shows how this extremely simple model reflects the 'bare bones' of internal cellular organisation. The metabolic input-output system is embedded in the larger $M$-$R$ system (the cell), which includes also the repair system (the nucleus).

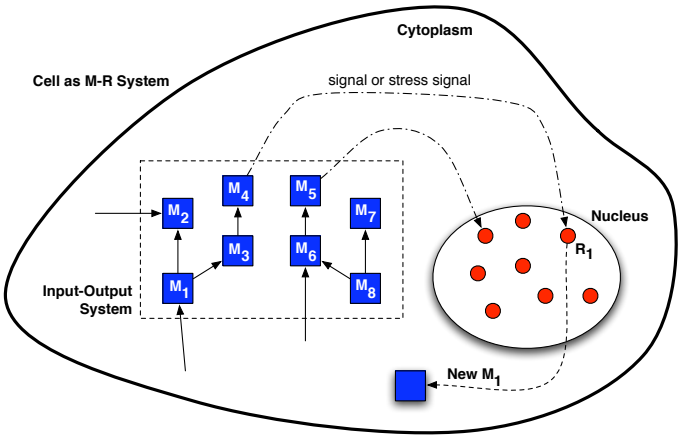


Figure 3: **Metabolic input-output system and repair system as part of $M$-$R$ system**

## 3.2 Dependency Structure of $M$-$R$ Systems

There are some additional definitions and theorems that we should mention before we switch to a more formal treatment (Rosen 1972b). The discussion deals with the structural (in an abstract sense) characteristics and requirements of systems whose components exhibit some level of interdependence.

Let's start by looking at component $M_1$ in Figure 1. If we take out this component, $M_2$, $M_3$, and $M_4$ will cease to function. However, the rest of the system will continue to work unhindered. On the other hand, if we remove the same component from the $M$-$R$ system of Figure 2 we notice that $M_4$ will fail to send a signal to $R_8$, thereby preventing $M_8$ from being replaced. As a consequence, the whole system will eventually stop functioning. In the $M$-$R$ system, $M_1$ is called a **central component**, i.e. a

component upon which the whole system depends. $M$-$R$ systems are typically more vulnerable to this kind of cascaded or domino effect.

If we now take out $M_2$ from Figure 2, something else happens. The rest of the system continues to function and eventually $M_2$ will get replaced when an appropriate signal from $M_4$ reaches $R_2$. Components like $M_2$ are called **reestablishable**, whereas components like $M_1$ are called **non-reestablishable**. We can now state an interesting theorem (Rosen 1972a):

> **Theorem**: Every $M$-$R$ system must contain a non-reestablishable component.
>
> **Proof**: Note that we are relying on the silent but obvious assumption that the $M$-$R$ system in question is finite, i.e. it has a finite number of components. Choose a reestablishable component and call it $M_1$. By hypothesis its corresponding repair component $R_1$ is not dependent on $M_1$. Thus $R_1$ must receive inputs from some component of the system. Therefore, there must be a component $M_2$ the output of which is an input to $R_1$. If $M_2$ is non-reestablishable we are done; otherwise, $R_2$ receives an input from another component $M_3$ distinct from $M_1$ and $M_2$. Its corresponding repair component $R_3$ must, if $M_3$ is reestablishable, receive an input from a component $M_4$ distinct from $M_1$, $M_2$, and $M_3$. Continuing in this manner we must eventually reach a non-reestablishable component, because otherwise this sequence of dependencies would go on to infinity, but we have started with the assumption that our system was finite. QED
>
> **Corollary**: If an $M$-$R$ system contains a single non-reestablishable component, then such a component is central.

The reason is fairly clear on an intuitive level, from the above construction, but we can prove it by saying that if we were to remove that single non-reestablishable component and be left with a working $M$-$R$ system, such a system would contradict the theorem we just proved because it would be composed of only reestablishable components.

### 3.3 Significance and Interpretation

We can say a few interesting things about the above results (Rosen 1972b). If we regard a cell as an $M$-$R$ system, there must be one or more metabolic components whose loss or injury is not repairable by the system.

One may think that minimising the number of non-reestablishable components would be desirable, i.e. down to 1. However, the smaller the number of non-reestablishable components the more central they become, i.e. their loss causes greater damage to the whole system. Thus, it should be interesting to see whether evolution has selected for a relatively large number of non-reestablishable components, such that the loss of each of these might not cause too great damage to the cell.

It is also worth asking how these considerations might relate to the execution graphs and to the composition of web services. Is there an optimum configuration of the execution graphs from the point of view of their robustness in case of the loss of one component? This seems relevant especially in the BIONETS context where a node that, for instance, hosts one of the service components ('service cell') may drift out of range, leaving the execution graph with the job of replacing it in order to complete the execution of the service. So these considerations relate to the balance between maximum repairability and minimum centrality in execution graphs and complex transaction models.

## 4 The Mathematics of $M$-$R$ Systems

The article on relational models in cell biology that Rosen wrote in 1972 (Rosen 1972b) is actually based on a series of articles from his PhD work in the late 50s (Rosen 1958a) (Rosen 1958b) (Rosen 1959), which are somewhat easier to read since they are less condensed. We will summarise here only a few of the main ideas in order to give a flavour of the category theory approach. Our motivation is that there is a close correspondence between categories and transformation semigroups, which represent our main analytical tools for understanding the algebraic structure of metabolic pathways. In addition, category theory can also be used to provide a common formal foundation with logic. Hence it is worth recounting some of Rosen's development in order to familiarise ourselves with the concepts also in a biological context. We stress that the following development is only the starting point of an approach that we hope will converge in our future work with automata theory and logic.

Figure 4 shows an atomic interaction of the above system, and how it can be translated into mathematical language. Each metabolic $M$-component receives inputs and generates outputs. Thus, it can be

seen as a function (or map, or mapping) from a set $A$ of inputs to a set $B$ of outputs, where the subscript $i$ refers to a particular component:

$$f_i : A_i \to B_i. \tag{1}$$

This is standard notation and is part of how a function is defined. A function can be further specified as being injective, surjective, or bijective. It is read, "$f_i$ is a function that maps the elements of set $A_i$ to the elements of set $B_i$". Another way to say this is

$$f_i \in H(A_i, B_i), \tag{2}$$

which reads, "$f_i$ belongs to the set of mappings from the set $A_i$ to the set $B_i$". $H$ denotes such a set of mappings.

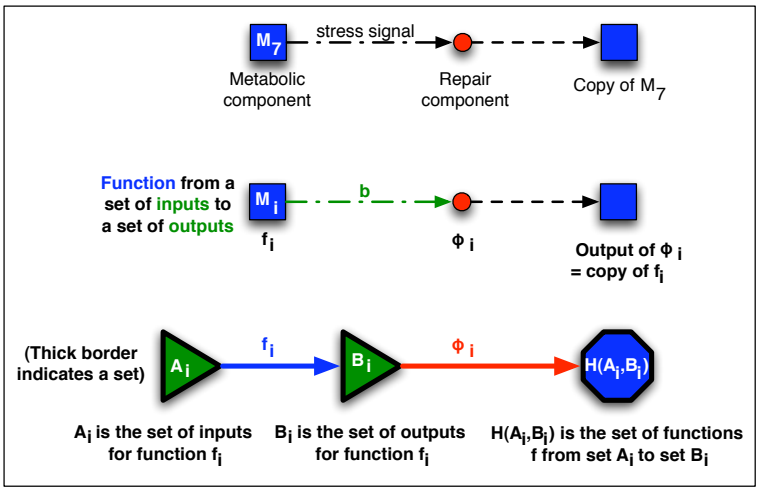


Figure 4: **Translating block diagrams into mathematical language**

Each repair $R$-component receives a signal from an $M$-component, and generates a new copy of this component. Hence it can be seen as a function from a signal $b \in B_i$ to another function, where $B_i$ is the set of signals associated with the output of metabolic component $i$:

$$\phi_i : [\text{set of signals}] \to [\text{set of functions}] \tag{3}$$

or,

$$\phi_i : B_i \to H(A_i, B_i). \tag{4}$$

Using the more compact notation,

$$\phi_i \in H(B_i, H(A_i, B_i)). \tag{5}$$

As shown in Figure 4, we have effectively swapped the blocks with the arrows in going from the block diagram view of the system to the functional view of the system. Although this is only a change in notation, it represents a subtle shift from a view of the system that emphasises its structure to a view that emphasises its functions and relations. This is precisely what category theory does, and is compatible with Rashevsky's idea of relational biology. The simplest $M$-$R$ system that captures these relationships is composed of a single metabolic and a single repair component, which can be written as follows:

$$(A \xrightarrow{f} B) \xrightarrow{\phi_f} H(A, B) \tag{6}$$

Whereas $M$-components are being replaced by $R$-components, we have not addressed the replacement of $R$-components. As mentioned above, this risks to lead us to an infinite regress of repair components that repair other repair components, ad infinitum. Rosen makes the very bold claim that the solution to this riddle is that category theory embodies all the necessary 'machinery' to enable the self-replication of the $R$-components. The 'self' in 'self-replication', however, refers to the whole $M$-$R$ system rather than to an individual $R$-component replicating itself. This means that the $M$-$R$ system is able to perform this replication. However, Rosen does not provide a mechanism to do so. What he does is to prove the mathematical feasibility of this fact, in two steps:

- first he recasts the repair map (Eq. 4), which is a map from the set $B$ to the set $H(A, B)$, as a map from the set of all possible repair maps to $H(A, B)$, which we can loosely write as $\phi \rightarrow f$
- then he shows that this map is invertible: $f \rightarrow \phi$

Thus, Rosen proved that $M$-$R$ systems are self-contained, or metabolically closed using the Cornish-Bowden terminology, or operationally closed using the Maturana and Varela terminology, by proving that $\phi$ and $f$ can generate each other, as shown at the bottom of Figure 6.

As shown in Figure 5, we use the familiar concept of the graph of a function to give an intuitive geometrical proof. We imagine several repair functions $\phi_i$'s, each of which can accept several inputs from its own set $B_i$ or also from other sets $B_i$'s to generate different $f_i$'s. This is more general than what we showed in the figures so far. For example, $\phi_5$ generates $f_{15}$, $f_{25}$, and $f_{35}$ upon receipt of $b_1$, $b_2$, and $b_3$, respectively. The possibility that a function $\phi_i$ can accept inputs from multiple $B_i$'s is represented as a Cartesian product for the domain of the $\phi_i$'s: $B = (B_1 \times B_2 \times ...)$. The curve linking the three points associated with $\phi_1$ is only meant to aid the visualisation: $\phi_1$ is certainly a discrete function, given that the number of $f_i$'s is finite.

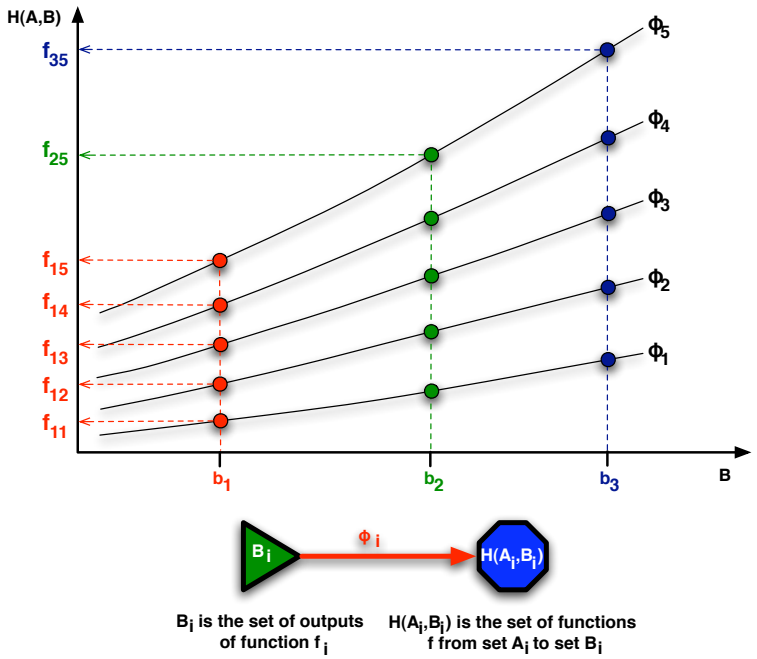


Figure 5: **Functional representation of repair components (inputs not shown in Cartesian product form)**

If each $\phi_i$ generates a single $f_i$ regardless of the input, Rosen calls that a constant map (for obvious reasons, since its graph would look like a horizontal line in Figure 5). If this argument is applied to Eqs. 1 or 2 then we are talking about a metabolic system whose behaviour does not change in the presence of environmental variations. If we are instead talking about Eqs. 3 or 4 (and Figure 5), that would indicate a system in which mutations never take place. Clearly some variability is desirable and present in the corresponding biological processes. Hence the greater generality of the graphs.

The first step in the proof is abstract and relies on the concept of the dual of a vector space $V$, denoted by $V^*$, and of the dual of the dual, or second dual, denoted by $V^{**}$. We recall that a vector space $V$ is defined on a field (finite or not) of scalars, which we could denote $F$.[3] A finite-dimensional vector space where each axis is defined over a finite field is necessarily discrete. If such a space were 3-dimensional, for example, it would have a total of $2^3 = 8$ points in the case of $\mathbb{Z}_2$ or $7^3 = 343$ points in the case of $\mathbb{Z}_7$. It is perhaps difficult to appreciate that also such topological objects, containing only a handful of points, can be vector spaces. They are however commonly encountered in applications such as network coding. Although the proof applies to vector fields in general we focus on the discrete case to highlight

[3]By a finite field we refer to a set composed of a prime number $p$ of consecutive positive integers, including 0, where addition and multiplication are performed modulo $p$. Such an object satisfies the field axioms. Familar examples of finite field are $\mathbb{Z}_2$, or $\mathbb{Z}_7$, and of infinite fields are the rational numbers $\mathbb{Q}$, the real numbers $\mathbb{R}$, and the complex numbers $\mathbb{C}$.

the fact that we can say something mathematically meaningful even for the extremely simplified model we are discussing here.

The dual space $V^*$ of $V$ is the space of all linear functionals from $V$ to its field of scalars. Linear functionals can be continuous or discrete. Keeping to the discrete, finite-dimensional case, there is an easy way to understand linear functionals. Since the elements of $V$ are vectors, one way to operate on $x \in V$ in such a way as to obtain a scalar is to perform the familiar dot product with another vector:

$$v \cdot x = a, \qquad x \in V, v \in V^*, a \in F \tag{7}$$

One way to make the simple dot product resemble a function is to fix $v$ and allow $x$ to vary. In other words we can view '$v\cdot$' as an operator acting on $x$, and we can write it as $h(x)$. In this manner we obtain one linear functional.[4] To obtain another, we pick a different vector, say $w$, and repeat the process to form $g(x)$, for example. The set of all such vectors forms the dual space $V^*$. Clearly $V$ and $V^*$ must be of the same dimension. It is easy to check that linearity holds:

$$h(x+y) = h(x) + h(y) \qquad \forall x, y \in V \tag{8}$$

$$h(ax) = ah(x) \qquad \forall a \in F. \tag{9}$$

In our analogy, the set $B$ in Figure 5 plays the role of the primary vector space, the set of functions $H(A,B)$ is the field of scalars, and the set of maps $\phi_i \in H(B, H(A,B))$ is the dual space.

Now to take the dual of $V^*$ we seek another vector space, $V^{**}$, such that its elements dotted with the elements of $V^*$ will again give a scalar. In the case where the starting vector space is infinite-dimensional, $V^{**}$ is larger than $V$ and the process of taking the second dual is called an 'embedding' of $V$ into $V^{**}$. If we are talking about finite-dimensional spaces, on the other hand, it turns out that $V$ and $V^{**}$ are isomorphic.[5]

This is useful to us because the process of taking the second dual in our functional analogy involves looking for a set of functions $\psi_b(\phi_i) \in H(H(B, H(A,B)), H(A,B))$ that will yield the same output as before, namely the functions in $H(A,B)$. As shown in Figure 5, this is achieved by fixing a particular value for $b \in B$ and letting the $\phi_i$'s vary over the set $H(B, H(A,B))$. By so doing, we construct an embedding of $B$ into $H(H(B, H(A,B)), H(A,B))$, as shown in Figure 6. Hence,

$$\psi_b(\phi_i) = \phi_i(b), \tag{10}$$

which is the analogue of the familiar relationship between a vector space and its second dual.

The second step in the proof relies on showing that the map $\phi \to f$ so constructed is 1-1 and onto, since any such map is necessarily invertible. The onto part of the proof does not seem too difficult since we are dealing with finite sets. Proving that the map is 1-1 requires two additional sub-steps. First, we need to require the map shown in Eq. 4 to be 1-1, as Figure 5 attempts to show graphically for each $\phi_i$. This ensures that no two $\psi$'s can ever overlap. In fact, as long as $\phi(b)$ acquires different values for different values of $b$, it will be impossible to construct two identical $\psi_b$'s for two different values of $b$. Second, the graphs of the functions $\phi_i$ in Figure 5 cannot cross because, if they did, the corresponding function $\psi_b$ for that value of $b$ would assume the same value for two different values of the abscissa $\phi$, and hence would not be 1-1. This second constraint means that two different repair functions are not allowed to generate the same metabolic component in response to the same input $b$, which is quite reasonable from a biological point of view. As long as these constraints are satisfied, the maps $\psi_b$'s will be 1-1, thereby proving the invertibility of the repair function.

The fact that DNA is indeed routinely repaired by the cell gives us pause. We already knew this and did not need category theory to tell us as much. However, the result of Rosen has a unique depth of significance because it highlights how the two repair systems, which we had no special reason to believe were related, may actually be very closely linked. How closely depends on how we interpret the meaning of 'invertible'. But there is no question that this result introduces an important aspect of the conceptualisation of the cell and of its possible mathematical modelling. Could this be an example of how the organisation of the metabolic activity mirrors in some way the structure of DNA? Could this be an example of what 'information contained in the environment' means?

The possibility that the same cell metabolism has an 'outward-facing' function to carry out the job of the cell and an inward-facing function to repair DNA brings us to the concept of multi-functionality.

[4] That a linear functional can be defined in this manner is discussed e.g. in Hall ((Hall 2003): 299).

[5] There is more to this story. As pointed out by one of the referees, Hilbert spaces are infinite-dimensional but also isomorphic to their second dual. In Halmos's terminology, they are also reflexive. We are only interested in the finite case here, and all finite-dimensional vector spaces are reflexive ((Halmos 1958): 24).

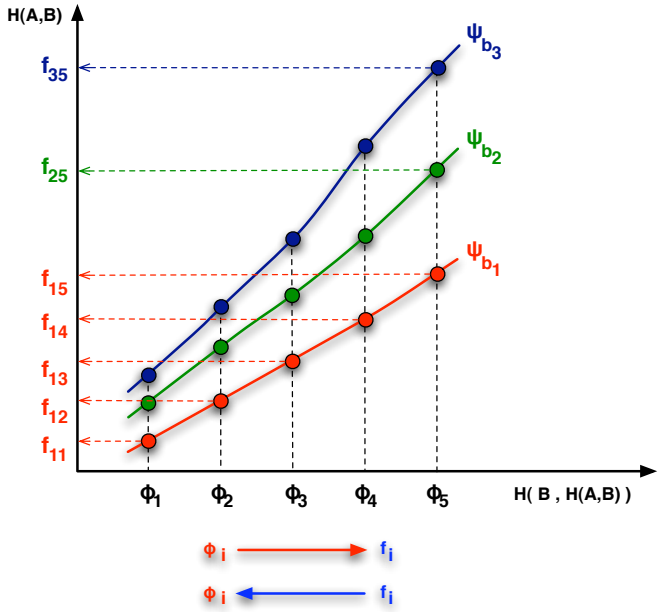


Figure 6: **Graph of the embedding of $B_1 \times B_2 \times ...$ into $H(H(B_i, H(A_i, B_i)), H(A_i, B_i))$.**

# 5 Multi-functionality

Cornish-Bowden and Cardenas share our view of the importance of the multi-functionality of the components of self-organising systems:

> A major principle that has emerged from studies of the circular organization of metabolism is that the circle of efficient causation can only be closed if some (and in reality probably many) of the catalysts used by organisms fulfil multiple functions. Multifunctionality, or 'moonlighting', is increasingly observed, but it is much more than just an interesting observation about living organisms, because it is essential to their survival. (Cornish-Bowden & Cardenas 2008)

Multi-functionality could, for example, enable an $M$-component to double up its function to map elements of the set of inputs $A$ to the set of outputs $B$ with the function to generate its $\phi$ – the same $\phi$ that will then generate $f$ when it receives the right signal, as discussed.

Nomura captures the analogous and nested functional structure of the $M$-$R$ system very succinctly (Nomura 2007): having defined the inverse of our $\psi_b$ as $\Phi_f$, the simplest $M$-$R$ system can be written as

$$\left\{ \left[ A \xrightarrow{f} B \right] \xrightarrow{\phi_f} H(A,B) \right\} \xrightarrow{\Phi_f} H(B, H(A,B)), \tag{11}$$

which shows how the $M$-$R$ system we have been talking about so far, on the left and in curly brackets, can itself be considered analogous to a 'metabolic component' of a larger $M$-$R$ system which repairs the $R$-components. This highlights Rosen's point that there is an "equivalence between the metabolic and genetic activities" (Rosen 1972b).

# 6 Conclusion

In this paper we have only scratched the surface of an important area of mathematics for the modelling of cellular processes, category theory. We have tried to make Rosen's mathematical argument for what can be called in the language of autopoiesis 'operational closure' more accessible to a wider audience. We have also hinted at the need to model the computational structure of the cell in more detail. If the former is motivated by 'essential' or ontological concerns that reach down to the nature of the very mathematical language used to codify these ideas, the latter is motivated by 'existential' or epistemological concerns that aim at formalising the scope and the character of the interaction between structure and function in cellular processes. In conclusion, much interesting work remains to be done.

## Acknowledgements

This work was supported mainly by the BIONETS Project (FP6 IST-027748) and in part by the OPAALS Project (FP6 IST-034824). I am indebted to Prof Alastair Munro for the Cornish-Bowden and Cardenas reference and to Dott. Miriam Pezzuto for the Nomura references. Many thanks to the referees who provided several valuable suggestions and corrections.



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