Linear Metabolism - Repair Systems

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Foreword

The Theory of Manufacturing study had as one of its objectives to explore alternative formal system structures for characterizing modern manufacturing processes. One such structure is based upon the metaphor of a living cell. In this Working Paper, the abstract mathematical structure of this metaphor is developed as a basis upon which to build a formal theory of cellular processes.

T.H. Lee
Director

Abstract

Metabolism-repair systems represent a formal mathematical framework for representating characteristic properties of living systems such as repair, replication, adaptation and so forth. In this paper, the concrete realization of such structures is developed in the case when the system "metabolism" is linear. Explicit results are given to show when system repair operations can counteract environmental and metabolic fluctuations. Additional results pertaining to the replication operation and the possibility for "Lamarckian" inheritance are also given, together with a formal demonstration of the increase in complexity as we proceed from the processes of metabolism to repair to replication. The paper concludes with a discussion of several application areas, together with a consideration of several conceptual and mathematical questions requiring attention for further development of this non-Newtonian systems paradigm.

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1. Extending the Newtonian Paradigm

A number of authors [1-3] have recently (and not so recently) pointed out a variety of deficiencies inherent in the classical Newtonian paradigm of mechanics regarding its utility in describing living systems, both biological as well as social and behavioral. In particular, it has been rather convincingly argued that the Newtonian world view, as exemplified in classical particle mechanics, say, has no role for any type of anticipatory behavior on the part of either observers or decisionmakers [4]. Furthermore, the crucial biological activities of repair and replication do not fit in any natural way into Newton's *"Weltanschauung,"* leading to the conclusion that an extension of the Newtonian paradigm, comparable in scope and impact to the extensions offered by both quantum mechanics and relativity theory in physics, is long overdue for mathematically capturing the essence of biological, social and behavioral phenomena.

About 30 years ago in a series of papers devoted to relational cell models [5-7], Rosen introduced the notion of a metabolism-repair (M,R)-network in an attempt to show formally how the features of repair and replication could be *naturally* induced solely from a cell's metabolic machinery. In subsequent work, it was also pointed out how anticipatory behavioral modes also followed in a straightforward manner from the (M,R)-formalism. Unfortunately, the formalism set up by Rosen and developed by others [8-9] was purely relational; i.e., it dealt with the *func-*

tional characteristics of the cell independent of its structural organization. In order to make contact with real material objects, it was necessary to explore means for *realizing* abstract (M,R)-systems in hardware, organic or otherwise. The initial attempts in this direction led to realizations of (M,R)-systems as automata, but with a highly non-canonical state-space [10-11]. Perhaps due to the discouraging results which followed from these somewhat unfortunate realizations, the topic seems to have disappeared from the literature and died, what in our opinion, is a very premature death.

In this paper, we attempt to resurrect the theory of (M,R)-systems making use of the vastly deeper understanding of the nature of canonical realizations acquired over the past decade or so, starting with the pioneering work of Kalman in the early '60s [12-14]. By making use of concepts and tools that were totally unknown at the time of Rosen's initial work, we put the (M,R)-set-up on firmer system-theoretic grounds, while at the same time answering a number of questions that were only in the realm of speculation at the time of the early papers [5-11]. While our results are presented only for the simplest case of *linear* (M,R)-systems, the extension to nonlinear situations follows along the same lines as the extensions from linear to nonlinear in system theory as given, for example, in [15,20]. It is our expectation that the framework set forth here will serve as a point of departure for the development of the kind of extension to the Newtonian paradigm that will serve the same role in biology and the social sciences that the Schrödinger equation and the Lorentz transformation served for physics.

The paper is organized according to the following scheme. Section 2 presents the basic ideas surrounding (M,R)-systems as originally developed by Rosen. In Section 3, a brief review of the realization problem and the construction of canonical state-space models is given for linear dynamical systems. The principal new results of the paper are presented in Sections 4 and 5, where we give explicit

characterizations of those linear systems which can be extended to linear (M,R)-systems, together with a discussion of how system complexity increases as we attempt to superimpose additional biological structure upon the basic metabolic machinery. Along the way, it is shown that Rosen's original scheme for the cell's replication mechanism can only be possible for a very limited class of (M,R)-processes. Finally, in Sections 6 and 7 we discuss the extensions of our results to nonlinear (M,R)-processes, as well as issues pertaining to a network of cells and the stability and control problems that such structures generate. The paper concludes with an indication of several application areas where (M,R)-systems should prove valuable in formalizing a variety of important practical questions.

2. Metabolism - Repair Networks

Consider a collection of N "cells", each of which accepts a variety of inputs and produces a spectrum of outputs. Assume that at least one cell accepts inputs from the "environment" and at least one cell produces outputs that are sent to the environment. Further, suppose that every cell accepts either environmental inputs or has as its inputs an output from at least one other cell; similarly, assume that each cell produces either an environmental output or has its output utilized as another cell's input. Such a network might look like Figure 1 (with N=5). Here we have the cells M_1 – M_5, together with the two environmental inputs ω_1 and ω_2, as well as the single environmental output γ_1. We call such a network a "metabolic" network.

It is reasonable to suppose that any cell in such a network will have a finite lifetime after which it will be removed from the system. When this happens, all cells whose input depends upon the output from the "dead" cell will also be affected, ultimately failing in their metabolic role, as well. In Figure 1, for instance, if

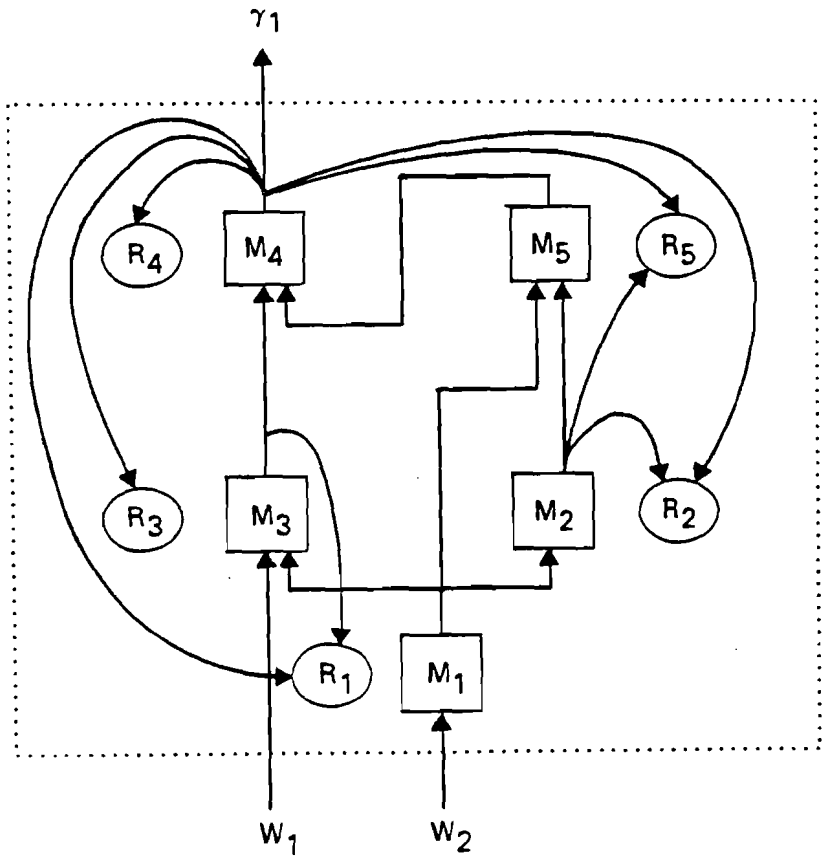


Figure 1. A Metabolism Network

the cell M_1 fails, then so will M_2, M_3, M_4 and M_5 all of whose inputs ultimately depend upon M_1's output. Any such cell whose failure results in the failure of the entire network is called a *central component* of the network.

Now let us suppose that we associate with each metabolic component M_i, a component R_i whose function is to *repair* M_i. In other words, when M_i fails the repair component R_i acts to build a copy of M_i back into the network. The R_i are constituted so that each R_i must receive at least one environmental output from the network and, in order to function, R_i must receive all of its inputs. Thus, in Figure 1 each R_i must receive the sole environmental output γ_1. Note also by the second

condition that any cell M_i, whose repair component R_i receives M_i's output as part of its input, cannot be built back into the network. We will call such a cell *non-reestablishable*. Thus, the cell M_2 is non-reestablishable, while cell M_5 is *reestablishable*.

Introduction of the repair components $\{R_i\}$ generates the following basic question: who repairs the repairers ? It would lead to a useless infinite regress to introduce another level of repair mechanisms, but what is the alternative ? Nature's solution to the problem is to make the repair components self-replicating. Before R_i dies, the replication mechanism built into R_i arranges to produce a *copy* of R_i, which then takes R_i's place in the network. Such networks are called (M,R)-systems.

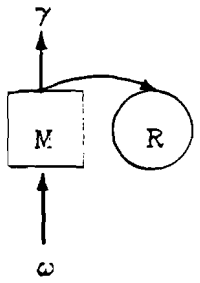
The elementary concepts introduced above already allow the following interesting results to be established [5,7]:

Theorem 1 (Rosen). Every finite (M,R) *- network contains at least one non-reestablishable component.*

Corollary. If an (M,R) *- network contains exactly one non-reestablishable component, then that component is central.*

Thus, we see that every (M,R)-network must contain some cells that cannot be built back into the system if they fail. Further, if there are a small number of such cells, then they are likely to be of prime importance to the overall functioning of the system. This last result has clear implications for policies devoted to keeping every component of a system alive (politicians and other social reformers: please note!). It may be much better to allow some cells to fail rather than run the risk of incurring a global system failure by trying to prop-up weak, non-competitive components which, by Theorem 1, can't all be saved in any case.

Let us now turn to an examination of the simplest possible (M,R) - system composed of a single component (N = 1). Diagrammatically, we have



or, more abstractly,

$$\Omega \xrightarrow{f} \Gamma \xrightarrow{P_f} H(\Omega, \Gamma) ,$$

where Ω = input set, Γ = output set, $f : \Omega \longrightarrow \Gamma$ (= metabolic map), $P_f : \Gamma \longrightarrow H(\Omega, \Gamma)$ (= repair map) with $H(\Omega, \Gamma)$ = set of all physically feasible metabolic maps. Here we subscript the repair map by f to indicate that the role of P_f is to produce the metabolism f when the metabolic part of the system receives its "correct" input $\omega \in \Omega$. We shall return to this point in detail in Sections 4 and 5.

The first point that arises is how to abstractly characterize the system replication map. Arguing biologically, the repair component P_f represents the system's *genetic* component and the job of the replication map is to use the system's metabolic machinery $(\Omega,\Gamma,H(\Omega, \Gamma))$ and process it into a copy of $P_f \in H(\Gamma, H(\Omega, \Gamma))$. Putting these remarks together, we see that the replication map, call it β_f, must act as

$$\beta_f : H(\Omega, \Gamma) \longrightarrow H(\Gamma,H(\Omega, \Gamma)) .$$

Thus, the abstract diagram characterizing the entire (M,R)-system is

$$\Omega \xrightarrow{f} \Gamma \qquad \xrightarrow{P_f} H(\Omega, \Gamma) \qquad \xrightarrow{\beta_f} H(\Gamma, H(\Omega, \Gamma))$$

{metabolism} {repair} {replication}

In what follows, we shall be concerned with putting concrete "meat" on the abstract "skeleton" of this diagram.

Before discussing some of the questions surrounding the behavior of such a system, two important points should be noted: 1) if we delete the repair and replication components of the diagram, we are left with the standard starting point of Newtonian mechanics and modern system theory , namely, pure metabolism; thus, the single-component (M,R)-network represents a genuine extension of the classical paradigm; 2) there is no set-theoretic difference between metabolism and repair: they both represent maps between abstract sets. Biologically, this suggests that there may be no intrinsic difference between a cell's metabolic and its genetic activity. We shall explore this point in more detail later on.

The important questions surrounding the repair aspects of the above type of (M,R)-system revolve about the degree to which the repair and replication components of the system can preserve the metabolic behavior in the face of fluctuations in the system's input ω or disturbances to its metabolism f.

Let's take a look at a few aspects of this question that we shall address in considerably more detail in Section 4.

Stable Metabolic Operations in Changing Environments - imagine the situation in which the cell's "usual" input ω is disturbed to a new input $\bar{\omega}$. The condition for stable operation of the cell is for the environment ω to be such that

$$P_f(f(\omega)) = f \ , \qquad (*)$$

i.e. the metabolic structure f is stable in the environment ω in the sense that the repair mechanism P_f always regenerates f when the environmental input is ω. We would say that all $\omega \in \Omega$ satisfying (*) form a stable environment for the cell.

Now suppose that the new environment $\bar{\omega} \neq \omega$. Then (*) will hold only if either

$$f(\omega) = f(\bar{\omega}) \quad \text{or} \quad P_f(f(\bar{\omega})) = f .$$

The first case is trivial in the sense that the observed products of the cell are invariant to the change of environmental inputs. If $f(\omega) \neq f(\bar{\omega})$ then the cell's outputs are not stable with respect to the change of environment and we must consider the repair mechanism to see whether or not the environmental alterations can be compensated for in the sense that

$$P_f(f(\bar{\omega})) = \bar{f} \neq f ,$$

with $\bar{f}(\bar{\omega}) = f(\omega)$, i.e., whether the genetic mechanism will produce a new metabolism $\bar{f}$ which duplicates the output of f, but with the input $\bar{\omega}$ rather than ω. In this case, the entire metabolic activity of the cell would be permanently altered if we had

$$P_f(\bar{f}(\bar{\omega})) = \bar{f} .$$

On the other hand, if we had $\bar{f}(\bar{\omega}) = f(\omega)$ or, more generally,

$$P_f(\bar{f}(\bar{\omega})) = f ,$$

then the cell's metabolism would only undergo periodic changes in time.

Finally we could have the situation in which

$$P_f(\bar{f}(\bar{\omega})) = \hat{f} \neq f , \bar{f}$$

and, iterating this process, we may see that an environmental change will cause the cell to wander about in the set $H(\Omega , \Gamma)$, changing its input/output behavior through a sequence of metabolic processes $f^{(1)} , f^{(2)} , f^{(3)} , \ldots$. This "hunting" process will terminate if either

(i) there exists an N such that

$$P_f (f^{(N)}(\bar{\omega})) = f^{(N)}$$

or

(ii) there exists an N such that

$$P_f\,(f^{(N)}(\bar{\omega})) \;=\; f^{(N-k)}\,, \quad k = 1,2,\ldots,N-1\ .$$

In case (i) the cell becomes stable in the new environment $\bar{\omega}$, while in case (ii) the cell undergoes periodic changes in its metabolic structure. If no such N exists, the cell is unstable and aperiodic. (Note: This last possibility can occur only if the set of possible metabolisms $H(\Omega\ ,\ \Gamma)$ is infinite).

A collection of related questions also arise in connection with the replication map β_f. For instance, we can ask whether or not Lamarckian changes are possible, i.e., can an environmental change $\omega \longrightarrow \bar{\omega}$ generate a permanent change in the genetic map P_f via the replication map β_f discussed above? In one particular construction of β_f due to Rosen [7], it can be shown that such changes are not possible. We shall show that Rosen's case is very special and that the general situation is far more complicated, even for linear maps.

Finally, we have a circle of issues relating to the complexity of (M,R)-systems. We can ask, for example, how complex P_f and β_f must be in order to repair a given metabolic map f, and the degree to which this requisite complexity can be generated within the bounds of biological and/or social constraints. We shall explore such considerations within the detailed confines of the linear framework developed in Section 4.

3. Input/Output Maps and Realizations

Beyond any doubt, it can safely be asserted that the fundamental problem of mathematical system theory is the construction of models from data: the Realization Problem. In general terms, we are given a system's external behavioral

description f (input/output behavior), and the task is to *construct* an internal state-space and dynamics so that the behavior of the resulting system Σ agrees with f, Σ being in some sense the "simplest" such system. The degree to which this construction can be carried out, either analytically or computationally, depends upon the character of f, as well as upon other problem boundary conditions (measurement error, constraints, input classes, etc.). Here we shall give a brief summary of the simplest and most well-understood case when f is linear. For a fuller account of these results, as well as their extensions to nonlinear f, we refer to the works [16-18].

Let Ω be a set of admissable system inputs, with Γ being the corresponding set of outputs. We shall assume that the elements of Ω are *sequences* of vectors in R^m, while Γ consists of sequences of vectors in R^p, $m,p \geq 1$. The behavior map is specified by a time-invariant, linear map $f : \Omega \longrightarrow \Gamma$. Thus, a typical element $\omega \in \Omega$ has the form

$$\omega = (u_0, u_1, u_2, \ldots)' , \quad u_i \in R^m ,$$

while an element $\gamma \in \Gamma$ looks like

$$\gamma = (y_1, y_2, y_3, \ldots)' , \quad y_i \in R^p .$$

Notice that we assume that time is discrete with the input ω starting at time $t = 0$, while the first output appears one unit later at time $t = 1$. In view of the linearity assumption on f, we can assert the existence of a sequence of matrices

$$B = \{A_1, A_2, A_3, \ldots\} , \quad A_i \in R^{p \times m} ,$$

such that the action $\omega \longrightarrow f(\omega) = \gamma$ can be represented as

$$y_t = \sum_{i=0}^{t-1} A_{t-i} u_i , \quad t=1,2,3,\ldots .$$

We call the sequence B, the *behavior sequence*. For technical reasons, it turns out to be convenient later to express the above input/output relation in component

form as

$$y_t = \sum_{i=0}^{t-1} [A_t^{(1)}_{-i} \mid A_t^{(2)}_{-i} \mid \cdots \mid A_t^{(m)}_{-i}] S(u_i) ,$$

where $A_t^{(j)}_{-i}$ = j^{th} column of A_{t-i} and $S(u_i)$ = "stack" of the vector u_i, i.e. the vector formed by stacking the columns of u_i to form a column of scalars. In this situation, where u_i is already a vector, $S(u_i) = u_i$ and the operation "S" has no effect. Later it will be important when it is operating on matrices.

The structure of the above input/output relation can also be written using a block Toeplitz matrix F as $\gamma = F\omega$, or,

$$\begin{bmatrix} y_1 \\ y_2 \\ . \\ \vdots \end{bmatrix} = \begin{bmatrix} A_1 & 0 & 0 & \cdots \\ A_2 & A_1 & 0 & \cdots \\ A_3 & A_2 & A_1 & \cdots \\ \vdots & \vdots & \vdots & \vdots \end{bmatrix} \begin{bmatrix} u_0 \\ u_1 \\ u_2 \\ \vdots \end{bmatrix} \qquad (*)$$

In what follows, it will also be useful to re-arrange the behavior sequence B in the block Hankel form

$$H = \begin{bmatrix} A_1 & A_2 & A_3 & \cdots \\ A_2 & A_3 & A_4 & \cdots \\ A_3 & A_4 & . & \cdots \\ \vdots & \vdots & \vdots & \vdots \end{bmatrix} .$$

We can now formulate the Realization Problem as:

Given the behavior sequence B, find an integer n, a vector space X of dimension n, and matrices $F \in R^{n\times n}$, $G \in R^{n\times m}$, $H \in R^{p\times n}$ such that

(1) $A_i = H F^{i-1} G$, $i = 1,2,\ldots,$

(2) The pair (F,G) is completely reachable, i.e.

$$\text{rank } [\, G \mid FG \mid F^2G \mid \cdots \mid F^{n-1}G \,] = n;$$

(3) the pair (F,G) is completely observable, i.e.,

$$\text{rank}\,[\,H' \mid F'H' \mid F'^2H' \mid \cdots \mid F'^{n-1}H'\,] = n\,.$$

Dynamically, we can express the system $\Sigma = (F, G, H)$ as

$$x_{t+1} = Fx_t + Gu_t\,, \quad x_0\,, = 0 \quad x_t \in X, \tag{Σ}$$

$$y_t = Hx_t\,, \qquad t = 0,1,2,....$$

The condition (1) simply means that the behavior of Σ agrees with that of B while conditions (2) - (3) insure that Σ is the simplest possible linear system satisfying condition (1), in the sense that there is no system whose state-space X has smaller dimension and whose behavior agrees with B The problem is how to construct the space X and the system $\Sigma = (F, G, H)$ from B. The answer hinges critically upon whether we know *in advance* whether or not there exists *any* $n < \infty$ with the requisite properties. If yes, then we can invoke a number of algorithms for determining Σ; if not, we are in the realm of the so-called "partial realization" problem, some of the deepest waters in modern system theory. We shall refer to the references for a discussion of this case and consider here only the situation where n is *assumed* finite and known.

Assuming the dimension n is known for a system Σ satisfying conditions (1) - (3), the first, and still one of the simplest, procedures for actually constructing (F, G, H) is the Ho Realization Algorithm [14,16,19], developed by B.L. Ho in 1968. Let $n < \infty$ be given. It can be shown that the infinite Hankel array H is such that rank $H = n$. Thus, there exist matrices P and Q such that

$$PHQ = \begin{bmatrix} I_n & 0 \\ 0 & 0 \end{bmatrix},$$

where I_n = n×n identity matrix. Let $\sigma(H)$ denote the infinite array obtained from H by left-shifting each row, i.e.

$$\sigma(H) = \begin{bmatrix} A_2 & A_3 & A_4 & \cdots \\ A_3 & A_4 & A_5 & \cdots \\ A_4 & A_5 & \cdots & \cdots \\ \vdots & \vdots & \vdots & \vdots \end{bmatrix}$$

Further, let R_l and C^s be "editing" matrices having the following actions:

R_l (A) $\doteq$ "retain first l rows of A."

C^s (A) $\doteq$ "retain first s columns of A."

Then Ho's Algorithm shows that a canonical (minimal) realization of B is given by setting $X = R^n$ and taking $\Sigma = (F, G, H)$ to be

$$F = R_n P \sigma(H) Q C^n ,$$

$$G = R_n P H C^m ,$$

$$H = R_p H Q C^n .$$

Thus, aside from the trivial editing operations R and C, the only real computation involved in Ho's procedure is the calculation of the matrices P and Q reducing H to Hermite form. All this is under the assumption, of course, that the all-important dimension $X = n$ is known via other considerations (e.g., all $A_i = 0$ for $i > N$). In what follows, we shall often invoke the existence of this algorithm (or its many equivalents) as a means for constructively realizing different behavior sequences that we encounter.

4. Linear (M,R)-Systems: Repair

Now we return to the consideration of the metabolism-repair systems outlined in Section 2, with the additional assumption that the metabolism, repair and replication maps are linear. For the moment, let us focus attention only upon the metabolic and repair structures.

The metabolic map $f: \Omega \longrightarrow \Gamma$ is exactly the structure discussed in the preceding section, with Ω and Γ vector spaces of input and output sequences, respectively. The repair map $P_f: \Gamma \longrightarrow H(\Omega, \Gamma)$ must abstractly produce f, given the output $\gamma \in \Gamma$ produced by f from the input $\omega \in \Omega$. Since we have seen that the metabolic map f is equivalent to the behavior sequence B, i.e.,

$$B = \{A_1, A_2, A_3, \ldots\} \approx f ,$$

we conclude that the space

$$H(\Omega, \Gamma) = \{\text{all possible behaviors } B\} .$$

This is a vector space under the obvious rules for addition and scalar multiplication.

Since we have assumed the map P_f to be linear, we can represent its action as

$$w_\tau = \sum_{i=0}^{\tau-1} R_{\tau-i} v_i , \qquad \tau = 1,2,\ldots, \qquad (**)$$

where (w_i, v_i) are the output and input to the repair system, respectively, with the elements R_j being linear maps determined by γ and f. However, since the repair system, when it operates properly, must accept the input γ and produce the output f, we must have $w_\tau = A_\tau$ and $v_\tau = S(y_{\tau+1})$ where S = "stack" operator defined in the previous section. Note here that we have used a different time parameter τ for the repair system, as it will usually be the case that the time-scale of operation of the repair system is considerably slower than the metabolic operation. We return to this point again in connection with replication in the next section.

It is an easy exercise to see that the elements R_j must have the form

$$R_j = [B_{j1} \mid B_{j2} \mid \cdots \mid B_{jp}], \quad B_{js} \in R^{p \times m}, \quad j = 1,2,\ldots .$$

So, in component form we can write (**) as

$$w_\tau = \sum_{i=0}^{\tau-1} [R_{\tau-i}^{(1)} \mid R_{\tau-i}^{(2)} \mid \cdots \mid R_{\tau-i}^{(p)}] S(v_i),$$

where we have written $R_j^{(s)} \doteq B_{js}$.

Just as the metabolism f was represented by the sequence $\{A_1, A_2, \cdots\}$, we can now see that the repair system P_f can be represented as

$$P_f = \{R_1, R_2, R_3, \cdots\}.$$

Similarly, we can also identify P_f with the Toeplitz matrix

$$P_f \approx \begin{bmatrix} R_1 & 0 & 0 & \cdots \\ R_2 & R_1 & 0 & \cdots \\ R_3 & R_2 & R_1 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix}.$$

Remarks:

(1) If we write each A_i as

$$A_i = \left[A_i^{(1)} \mid A_i^{(2)} \mid \cdots \mid A_i^{(m)}\right], \qquad A_i^{(j)} \in R^p,$$

the "complexity" of each component of the metabolic map f is $O(pm)$; the complexity of each element R_j of the repair map P_f is $O(p^2 m)$. Thus, already the often noted complexity increase associated with living systems begins to emerge through natural mathematical requirements.

(2) A straightforward calculation shows that the assumption dim $\Sigma = n < \infty$ implies that the set $\{A_1, A_2, \ldots, A_{2n}\}$ is linearly dependent (this follows from elementary properties of the Hankel array H). It is now easy to see that the condition

dim $\Sigma = n < \infty$, also implies that the canonical realization of the repair sequence $\{R_1, R_2, \ldots\}$ has dimension $n_p \leq n$. Thus, we can again employ Ho's Algorithm to produce a system $\Sigma_p = (F_p, G_p, H_p)$ realizing the repair sequence.

Example. . To fix the foregoing ideas, consider the situation in which the system's environmental input ω is

$$\omega = (1, 1, 0, 0, \ldots) ,$$

with the metabolic output $\gamma = f(\omega)$ being given by

$$\gamma = \{1,2,3,4,\ldots\} = \text{natural numbers.}$$

Since ω and γ are scalar sequences, we have $m = p = 1$. We easily obtain the behavior sequence

$$B = \{1,1,2,2,3,3,4,4,\ldots\} = \{A_1, A_2, A_3, \ldots\} .$$

It can be shown that this behavior sequence has a canonical realization $\Sigma = (F, G, H)$ of dimension $n = 3$, so an application of Ho's Algorithm yields the canonical system matrices

$$F = \begin{bmatrix} 0 & 1 & 0 \\ 1 & -1 & 1 \\ 1 & -2 & 2 \end{bmatrix} , \quad G = \begin{bmatrix} 1 \\ 1 \\ 2 \end{bmatrix} , \quad H = [\,1\,0\,0\,] .$$

The dynamics for the metabolic subsystem are

$$\Sigma: \quad x_{t+1} = \begin{bmatrix} 0 & 1 & 0 \\ 1 & -1 & 1 \\ 1 & -2 & 2 \end{bmatrix} x_t + \begin{bmatrix} 1 \\ 1 \\ 2 \end{bmatrix} u_t , \quad x_0 = 0 , \quad x_t \in R^3 ,$$

$$y_t = [\,1\,0\,0\,]\, x_t , \qquad t = 0,1,2,\ldots$$

Turning now to the repair component, we must have $P_f(\gamma) = f$ which leads to

$$R_i = \begin{cases} [1], & i \text{ odd} \\ [-1], & i \text{ even} \end{cases}$$

Thus, the Toeplitz operator for P_f is

$$P_f \approx \begin{bmatrix} 1 & 0 & 0 & 0 & \cdots \\ -1 & 1 & 0 & 0 & \cdots \\ 1 & -1 & 1 & 0 & \cdots \\ \vdots & \vdots & \vdots & \vdots & \cdots \end{bmatrix}$$

with the associated Hankel array

$$H_P = \begin{bmatrix} 1 & -1 & 1 & -1 & 1 & \cdots \\ -1 & 1 & -1 & 1 & -1 & \cdots \\ 1 & -1 & 1 & -1 & . & \cdots \\ \vdots & \vdots & \vdots & \vdots & \vdots & \cdots \end{bmatrix}$$

Since we know that the repair sequence has a finite-dimensional realization of dimension $n_P \leq n = 3$, experimenting a bit with Ho's Algorithm (or computing rank H_P) gives $n_P = 1$, with the resultant canonical repair realization $\Sigma_P = (F_P, G_P, H_P)$, where

$$F_P = [-1], \quad G_P = [1], \quad H_P = [1].$$

The repair dynamics are then

$$z_{\tau+1} = [-1]\, z_\tau + [1] v_\tau, \quad z_0 = 0, \quad \tau = 0,1,2,\ldots$$
$$w_\tau = [1]\, z_\tau .$$

From our earlier remarks, we connect this system with the metabolic map f via inputs and outputs as $w_\tau = A_\tau$, $v_\tau = y_{\tau+1}$.

Remarks

(1) At first glance, there appears to be a contradiction here to our earlier claim that the repair system is more "complex" than the metabolism. In this example, we see that dim $\Sigma_P = 1$ < dim $\Sigma = 3$, so if one measures complexity by state-space dimension, then Σ_P is actually never more complex than Σ. In fact, as we have already noted, this will ***always*** be the case. However, our earlier remark used a different notion of complexity, one involving the objects of the behavioral descriptions, the elements A_i and R_i. Unless $p = 1$, the objects $\{R_i\}$ always contain more elements than the $\{A_i\}$. Thus, by this measure of complexity, the repair sys-

tem is always at least as complex as the metabolism. Roughly speaking, it is more difficult to *describe* the behavior of the repair process than the metabolism, but is simpler to *realize* its dynamics. In engineering terms, there are fewer "integrators", but of a more complicated type.

Now let us return to a consideration of the main function of the repair mechanism: to restore the correct input/output behavior (ω, γ) in the face of changes in either the environmental input ω or the metabolic machinery f. There are several cases and subcases to examine:

Case I. Fixed environment ω^* and a fixed genetic machinery P_{f^*} with variable metabolism f.

In this case, we are concerned with changes in the metabolic machinery from some nominal, or basal, metabolism f^*. In other words, we consider those metabolisms f such that $P_{f^*}(f(\omega^*)) = f$ or f^*. In the first case, the repair machinery P_f^* stabilizes the system at the new metabolism f; in the second case, P_f^* acts to restore the nominal metabolism f^*.

To study this situation, it is useful to consider the map

$$\Phi_{\omega^*,f^*} : H(\Omega,\Gamma) \longrightarrow H(\Omega,\Gamma)$$
$$f \longmapsto P_{f^*}(f(\omega^*))$$

The case in which the repair system stabilizes the system at the new metabolism f corresponds to finding the fixed points of the map Ψ_{ω^*,f^*}, i.e., those metabolisms f such that

$$\Psi_{\omega^*,f^*}(f) = f.$$

The situation in which the repair system restores the design metabolism f^* by "repairing" the perturbation $f^* \longrightarrow f$, corresponds to finding those perturbations f such that

$$\Psi_{\omega^*,f^*}(f) = f^* .$$

Note that by construction we must have

$$\Psi_{\omega^*,f^*}(f^*) = f^* ,$$

i.e., f^* is a trivial fixed point of Ψ_{ω^*,f^*} as is the null metabolism f=0, by virtue of the fact that Ψ_{ω^*,f^*} is linear, being induced from the linear map P_{f^*}.

Since each $f \in H(\Omega,\Gamma)$ has the form $f = \{A_1,A_2,A_3,\ldots\}$, we can represent Ψ_{ω^*,f^*} by the infinite matrix

$$\Psi_{\omega^*,f^*} = \begin{bmatrix} \Psi^*_{11} & \Psi^*_{12} & \Psi^*_{13} & \cdots \\ \Psi^*_{21} & \Psi^*_{22} & \cdot & \cdots \\ \vdots & \vdots & \vdots & \vdots \end{bmatrix}$$

where $\Psi^*_{ij} \in R^{p\times p}$, i,j=1,2,... Since Ψ_{ω^*,f^*} is induced from the repair map P_{f^*}, the elements Ψ^*_{ij} will be determined by the elements $\{R^*_1,R^*_2,R^*_3,\ldots\}$ and $\omega^* = \{u^*_0,u^*_1,\ldots\}$ determining P_{f^*} . It should be noted that in general, as with the choice of the matrices $\{A^*_i\}$ defining f^*, there is some level of arbitrariness in the elements $\{R^*_i\}$. Unless the input ω^* has special structure, there will be p(m−1) degrees-of-freedom in the choice of each A^*_i; similarly, each R^*_i will have pm(p−1) degrees-of-freedom in its elements, non-uniqueness that is inherited by the elements Ψ^*_{ij} comprising Ψ_{ω^*,f^*}

We can now make the following observations about the properties of Ψ_{ω^*,f^*} and the behavior of the repair system P_{f^*} in the form of

Theorem 2. (1) The requirement that $f^* = \{A^*_1,A^*_2,A^*_3,\ldots\}$ *be a fixed point of* Ψ_{ω^*,f^*} *means that the vector* $[A^*_1,A^*_2,A^*_3,\ldots]'$ *is a characteristic vector of* Ψ_{ω^*,f^*} *with associated characteristic value 1.*

The elements Ψ^*_{ij} *are restricted only in that they must be selected to satisfy this condition;*

(2) the perturbation metabolism $f = \{A_1,A_2,A_3,\ldots\}$ *will be a fixed point of*

Ψ_{ω^*,f^*} *if and only if the vector* $[A_1,A_2,A_3,\ldots]'$ *is a characteristic vector of* Ψ_{ω^*,f^*} *with associated characteristic value 1;*

(3) the perturbation f *will be "repaired", i.e.,* $\Psi_{\omega^*,f^*}(f) = f^*$ *if and only if* f *has the form* $f = f^* + \ker \Psi_{\omega^*,f^*}$. *In other words, for repair we must have the vector* $[A_1-A_1^*,A_2-A_2^*,A_3-A_3^*,\ldots]' \in \ker \Psi_{\omega^*,f^*}$.

The last two points have deep implications for the ability of the repair system to function effectively in that they are diametrically opposed: if we want to be able to repair many different types of perturbation f, then by (3) we need to have $\ker\Psi_{\omega^*,f^*}$ "large"; if $\ker\Psi_{\omega^*,f^*}$ is large, then there are relatively "few" characteristic vectors with associated characteristic values 1 implying that there are only a "small" number of perturbations f that will be stabilized by the repair system. The sum total is that we can either arrange to have $\ker \Psi_{\omega^*,f^*}$ "large" and *repair* many disturbances, or we can have $\ker \Psi_{\omega^*,f^*}$ "small" and be able to *stabilize* many metabolic disturbances, but not both! The amount of flexibility we have in choosing the $\ker \Psi_{\omega^*,f^*}$ is dictated by the degrees-of-freedom we have in determining P_f which, as noted above, is proportional to the quantity $pm(p-1)$, where p and m are the number of metabolic outputs and inputs, respectively. (It should be noted that this is the number of degrees-of-freedom *after* satisfying the condition in part (1) of Theorem 2).

It is impossible to speak any more precisely about the repair mechanism in the absence of more specific details about the structure of Ψ_{ω^*,f^*}. So, let us examine the process determining Ψ_{ω^*,f^*} in greater detail.

From the component representation of (**), we can see that

$$A_\tau = \sum_{j=0}^{i} \sum_{i=0}^{\tau-1} [R_{\tau-i}^{*(1)} \mid \cdots \mid R_{\tau-i}^{*(p)}] (A_{i-j+1}\, u_j^*), \qquad (**')$$

This is clearly a triangular (in fact, Toeplitz) representation as A_τ depends only upon the elements $A_1, A_2, \ldots, A_\tau$ in a linear, Toeplitz manner. As long as all p components of $A_{i+j-1}\, u_j^*$ are not zero, we can always find a solution to this equation in the components of the matrices $\{R_{\tau-1}^*\}$ and the elements $\{u_j^*\}$, i=0,1,2,...,$\tau-1$; j=1,2,...,m. In fact, generically there is a pm(p-1)-parameter ***family*** of such solutions, after we have selected some of the entries of the R's in order to satisfy the requirement that

$$A_\tau^* = \sum_{j=0}^{i} \sum_{i=0}^{\tau-1} [R_{\tau-1}^{*(1)} \mid \cdots \mid R_{\tau-1}^{*(p)}]\, A_{i-j+1}^*\, u_j^*), \quad \tau=1,2,\ldots .$$

where the elements R^* denote the parametrized family of solutions satisfying this relation.

On the other hand, the induced relation Ψ_{ω^*,Γ^*} says that we must have

$$\begin{bmatrix} \Psi_{11}^* & \Psi_{12}^* & \Psi_{13}^* & \cdots \\ \Psi_{21}^* & \Psi_{22}^* & . & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix} \begin{bmatrix} A_1^* \\ A_2^* \\ \vdots \end{bmatrix} = \begin{bmatrix} A_1^* \\ A_2^* \\ \vdots \end{bmatrix},$$

for some ***triangular*** choice of Ψ_{ij}^*. In particular, this means that $\Psi_{ij}^*=0$, $j > i$ and we have

$$A_\tau^* = \Psi_{\tau 1}^* A_1^* + \Psi_{\tau 2}^* A_2^* + \cdots$$

But, we also have the expression for A_τ^* from above involving the elements $\{R_{\tau-1}^*\}$. Setting these two expressions equal, we obtain

$$\Psi_{\tau 1}^* A_1^* + \Psi_{\tau 2}^* A_2^* + \cdots = \sum_{j=0}^{i} \sum_{i=0}^{\tau-1} [R_{\tau-1}^{*(1)} \mid \cdots \mid R_{\tau-1}^{*(p)}]\, (A_{i-j+1}^*\, u_j^*), \quad \tau=1,2,\ldots \quad (\dagger)$$

The relation (†) then enables us to pin down some of the elements $\{\Psi_{\tau k}\}$, k=1,2,.... The arbitrary elements in $\{\Psi_{\tau k}^*\}$ will usually then be dictated by the arbitrary elements in the $\{R_{t-1}^*\}$ in order to make the ker Ψ_{ω^*,Γ^*} "large" or "small", as the case may be.

One case in which we can be very specific about the structure of Ψ_{ω^*,f^*} is when m=p=1. In this case we can easily solve the relation (**') for the elements Ψ^*_{ij}, obtaining the triangular Toeplitz array

$$\Psi_{\omega^*,f^*} = \begin{bmatrix} R_1^* u_o^* & 0 & 0 & \cdots \\ R_2^* u_o^* + R_1^* u_1^* & R_1^* u_o^* & 0 & \cdots \\ R_3^* u_o^* + R_2^* u_1^* + & R_2^* u_o^* + R_1^* u_1^* & R_1^* u_o^* & \cdots \\ R_1^* u_2^* & . & . & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix}$$

Here there are no degrees-of-freedom in the $\{R_i^*\}$, so the spectral structure of Ψ_{ω^*,f^*} is fixed.

Example (continued)

We can make use of the above scalar input/output case to examine the repair mechanism for our earlier sample problem. Before we had

$$\omega^* = \{1,1,0,0,\ldots\} = \{u_o^*, u_1^*, u_2^*, \ldots\},$$
$$f^* = \{1,1,2,2,3,3,\ldots\} = \{A_1^*, A_2^*, A_3^*, \ldots\},$$
$$P_f = \{1,-1,1,-1,\ldots\} = \{R_1^*, R_2^*, R_3^*, \ldots\}.$$

Let us suppose that the metabolism f^* is perturbed to the new metabolism

$$f = \{1,2,2,2,3,3,4,4,\ldots\} = \{A_1, A_2, A_3, \ldots\},$$

i.e., there is a change only in the 2nd element. The system output under f is now

$$\gamma = f(\omega^*) = \{1,3,4,4,5,6,7,\ldots\}.$$

Thus, the metabolic change results in a change of output from γ^* = natural numbers to the closely related sequence γ, which differs from γ^* only in the 2nd and 3rd entries. The question is what effect this seemingly minor change has upon the repair mechanism.

To address this issue, we compute the matrix Ψ_{ω^*,f^*} which, using the requirement that f^* must be a fixed point, gives

$$\Psi_{\omega^*,f^*} = \begin{bmatrix} 1 & 0 & 0 & . & . \\ 0 & 1 & 0 & . & . \\ 0 & 0 & 1 & . & . \\ \vdots & \vdots & \vdots & \vdots & \vdots \end{bmatrix} = \text{identity.}$$

Consequently, appealing to Theorem 2 we find that the metabolism f is also a fixed point of Ψ_{ω^*,f^*} with characteristic value 1; hence, the repair mechanism will process γ into f and thus stabilize the system at the new metabolism f. In fact, this will be true for *any* metabolic perturbation f of this system: the repair process will immediately "lock-on" to the new metabolism f and stabilize the system there. Thus, for this system there is no "repair" but only an immediate stabilization at the new metabolism.

Another important point to note about this scalar case is that we must have the product $R_1^* u_0^* = 1$ or 0 for the possibility of *either* repair or "locking-on" to a new metabolism. Otherwise, we cannot ever exactly restore f* or exactly lock-on to a new metabolism, but only obtain a scalar multiple of f* or f. Technically, of course, this is not an important distinction; in practice, it may or may not be significant.

Case II. A fluctuating environment ω with fixed nominal metabolism f and fixed genetic machinery P_{f^*}.*

In this situation, we have a change of environment $\omega^* \longrightarrow \omega$, and want to find all those environments ω such that

$$P_{f^*}(f^*(\omega^*)) = P_{f^*}(f^*(\omega)) \quad (= f^*)$$

implies

$$f^*(\omega^*) = f^*(\omega)\,.$$

In other words, we want to know when P_{f^*} is 1-1.

But the matrix representation of P_{f^*} is

$$P_{f^*} = \begin{bmatrix} R_1^* & 0 & 0 & \cdots \\ R_2^* & R_1^* & 0 & \cdots \\ R_3^* & R_2^* & R_1^* & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix}, \qquad R_i^* \in R^{p \times pm}\,,$$

implying that P_{f^*} is 1-1 if and only if ker $R_1^* = \{0\}$. This will be the case if and only if $m = 1$ and rank $R_1^* = p$.

Here we only consider the situation when $P_{f^*}(f^*(\omega^*)) = P_{f^*}(f^*(\omega))$, since if this is not the case, then we are back in Case I, i.e., that of a metabolic change. We can now conclude

Theorem 3. If $m = 1$ *and rank* $R_1^* = p$, *all environments* ω *such that* $f^*(\omega) = f^*(\omega^*)$ *are given by* $\omega = \omega^* + \ker f^*$;

On the other hand, if $m > 1$ *and/or rank* $R_1^* = r < p$, *then any environmental change of the form* $\omega = x + \omega^*$, *where* x *is any solution of the equation* $f^*(x) = \hat{\gamma}$, $\hat{\gamma} \in \ker R_1^*$, *will be repaired by* P_{f^*}.

Proof. Let $m = 1$ and rank $R_1^* = p$. Then the operator P_{f^*} is 1 - 1 and all the environments ω such that $P_{f^*}(f^*(\omega)) = P_{f^*}(f^*(\omega^*))$ implies $f^*(\omega) = f^*(\omega^*)$ consist of those ω satisfying $\omega = \omega^* + \ker f^*$.

Now let $m > 1$ and/or rank $R_1^* = r < p$, i.e. ker R_1^* is non-empty. Let $\hat{\gamma} \in \ker R_1^*$ and let x be a solution of the equation $f^*(x) = \hat{\gamma}$. Then any environmental change of the form $\omega^* \longrightarrow \omega = x + \omega^*$ will be repaired by the genetic mechanism P_{f^*} since

$$\begin{aligned} P_{f^*}(f^*(\omega)) &= P_{f^*}(f^*(x) + f^*(\omega^*)) \\ &= P_{f^*}(\hat{\gamma}) + P_{f^*}(f^*(\omega^*)) \\ &= 0 + f^* = f^* \end{aligned}$$

Theorems 2 and 3 characterize all those metabolic and environmental changes that can be "repaired" by a fixed genetic machinery P_{f^*}. Let us now consider the ways in which this genetic apparatus itself can change by means of replication.

5. Linear (M,R)-Systems : Replication

The system replication map

$$\beta_f \ : \ H(\Omega,\Gamma) \longrightarrow H\,(\Gamma,\, H(\Omega,\Gamma))$$

can be formally considered in much the same fashion as just discussed for the repair mechanism P_f. However, since the functional role of β_f is quite different from that of P_f, a number of interesting questions arise that are absent in the case of repair, questions involving mutation, adaptation, Lamarckian inheritance and so forth. We shall consider these matters in more detail in a moment, but first let us look at the formal realization of β_f.

Since β_f is a linear map accepting inputs of the form $f = \{A_1, A_2, A_3, \ldots\}$ and producing outputs $P_f = \{R_1, R_2, \ldots\}$, we must have a representation of the action of β_f as

$$c_\sigma = \sum_{i=0}^{\sigma-1} U_{\sigma-i}\ e_i \ ,$$

for an appropriate set of matrices $\{U_j\}$, where the input $e_i = S(A_i)$ and the output $c_i = R_i$. Arguing just as for the repair map , we conclude that U_j must have the form

$$U_j = [C_{j1} \mid C_{j2} \mid \cdots \mid C_{j,mp}] \ , \quad j = 1,2,\ldots \ ,$$

where each $C_{jr} \in R^{p \times mp}$. In what follows, we shall write $U_j^{(r)} \doteq C_{jr}$. So, just as with f and P_f, we have the representation of β_f as

$$\beta_f = \{U_1, U_2, \cdots\} \ ,$$

together with the associated Toeplitz identification

$$\beta_f \approx \begin{bmatrix} U_1 & 0 & 0 & \cdots \\ U_2 & U_1 & 0 & \cdots \\ U_3 & U_2 & U_1 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix} \ .$$

and the associated Hankel array

$$H_\beta \approx \begin{bmatrix} U_1 & U_2 & U_3 & \cdots \\ U_2 & U_3 & U_4 & \cdots \\ U_3 & U_4 & U_5 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix} .$$

Note that in the above set-up, since the inputs for the replication system must correspond to the metabolism f, while the outputs must be the associated repair map P_f, we have the relations

$$e_\sigma = S(A_{\sigma+1}) , \quad c_\sigma = R_\sigma ,$$

with S being the "stacking" operator. These relations are expressed in the time-scale σ of the replicator system. Here we have introduced still a third time-frame σ to distinguish between the scale t for metabolism and τ for repair. Usually, we will have $\Delta t \leq \Delta\tau \leq \Delta\sigma$.

Using the same arguments as for P_f, it can be established that if f has a finite-dimensional realization, so does β_f and the dim $\beta_f \leq$ dim f. So, in connection with the example given in the last section, we find that if

$$f = \{1,1,2,2,3,3,4,4,\ldots\} ,$$
$$P_f = \{1,-1,1,-1,1,-1,\ldots\} ,$$

then, after a bit of algebra,

$$\beta_f = \{1,-2,1,0,0,\ldots\} .$$

Thus, only the terms U_1, U_2 and U_3 are non-zero. Note the apparent decrease in complexity of the sequences f, P_f and β_f as we pass from metabolism to repair to replication. We will return to this point below.

Applying Ho's Algorithm to β_f yields the realization of the replication map β_f as

$$q_{\sigma+1} = \begin{bmatrix} 0 & 0 & 0 \\ 1 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} q_\sigma + \begin{bmatrix} 1 \\ 0 \\ 0 \end{bmatrix} e_\sigma , \quad q_0 = 0 , \; q_\sigma \in R^3 ,$$

$$c_\sigma = (1 \; -2 \; 1)\, q_\sigma , \qquad \sigma = 0,1,2,\ldots .$$

The machinery outlined above provides a systematic procedure for generation of a canonical replication system via Ho's Algorithm (and a repair mechanism, too) for any metabolism, provided only that the metabolism possesses *some* finite-dimensional realization; this is the *only* condition needed for the existence of a finite-dimensional repair and replication process constructible directly from the metabolic components Ω, Γ and $H(\Omega, \Gamma)$ via "natural" mathematical operations. In the paper [7], Rosen suggests another construction for the replication system, one which imposes no assumptions on the metabolism but which entails some severe conditions of another nature order to make the scheme work. Since Rosen's construction brings forth many of the aspects of replication we want to examine, and is of some interest in its own right, we briefly summarize his argument.

Recall that for replication we need a map $\beta_f : H(\Omega, \Gamma) \rightarrow H(\Gamma, H(\Omega, \Gamma))$ possessing the property that $\beta_f(f) = P_f$. Let X and Y be arbitrary sets. Then there is a naturally defined map

$$\hat{x} : H(X, Y) \rightarrow Y ,$$

given by

$$\hat{x}(f) = f(x) ,$$

for all $x \in X$. This is the so-called "evaluation map" on $H(X, Y)$. *Assume* that $\hat{x}$ is 1-1. Then there exists a map $\hat{x}^{-1}$ such that

$$\hat{x}^{-1} : Y \rightarrow H(X, Y) .$$

Now we need only set $X = \Gamma$, $Y = H(\Omega, \Gamma)$ to obtain the desired replication map, call it $\hat{\gamma}^{-1}$:

$$\hat{\gamma}^{-1} : H(\Omega, \Gamma) \rightarrow H(\Gamma, H(\Omega, \Gamma)) .$$

This is Rosen's construction, which mirrors the usual procedure for construction of the dual space of Γ. Note, however, that the success of this procedure for producing a replication map hinges entirely upon the map $\hat{x}$ being 1-1. Rosen

argues that this is a mathematical expression of the celebrated "one-gene, one-enzyme" hypothesis from molecular genetics, and uses this interpretation as supporting evidence for his construction. Let us examine this argument in light of the linear structures introduced above.

In our terminology, Rosen's construction involves the injectivity of the map

$$\begin{aligned} \hat{\gamma} \; : \; H\,(\Gamma,\, H(\Omega,\, \Gamma)) &\longrightarrow H(\Omega,\, \Gamma) \\ P_f &\longmapsto f \; . \end{aligned}$$

If $\hat{\gamma}$ *is* 1-1, then we have a map

$$\begin{aligned} \hat{\gamma}^{-1} \; : \; H(\Omega,\, \Gamma) &\longrightarrow H(\Gamma,\, H(\Omega,\, \Gamma)) \\ f &\longmapsto P_f \\ \{A_1,\, A_2, \ldots\} &\longmapsto \{R_1,\, R_2, \ldots\} \; . \end{aligned}$$

But, this means that $\hat{\gamma}^{-1}$ is equivalent to the matrix

$$\hat{\gamma}^{-1} \;=\; \beta_f \;\approx\; \begin{bmatrix} U_1 & 0 & 0 & \cdots \\ U_2 & U_1 & 0 & \cdots \\ U_3 & U_2 & U_1 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix} \; .$$

Thus, such a map $\hat{\gamma}$ exists if and only if the matrix β_f is invertible. But, since each $U_i \in R^{p \times p^2 m^2}$, β_f can be invertible if and only if: 1) $p = p^2 m^2$, i.e. $p = m = 1$ and 2) $U_1 \neq 0$. Consequently, we see that Rosen's scheme can work only in the case of a single-input/single-output metabolism, and even then only if $U_1 \neq 0$; this is a very severe restriction.

In summary, the construction we have given for the replication operation works for all finitely realizable metabolisms. The construction due to Rosen will work for any metabolism, provided that there is only a single-input and a single-output (assuming $U_1 \neq 0$). We shall see the implications of these different situations momentarily.

Within the context of replication, there are two basic questions of interest:

1) When can environmental changes $\omega \longrightarrow \omega'$ result in changes in the replication map β_f ?

2) If external disturbances modify β_f, what kinds of changes in f can result ?

The first of these is the question of Lamarckian inheritance, while the second addresses problems of mutation. We consider only the Lamarckian question here, deferring a treatment of the second, vastly more complicated question to a future paper.

From the diagram

$$\Omega \xrightarrow{f} \Gamma \xrightarrow{P_f} H(\Omega, \Gamma) \xrightarrow{\beta_f} H(\Gamma, H(\Omega, \Gamma)) ,$$

it is evident that

$$P_f (f(\omega)) = f = [\beta_f (f)] (f(\omega))$$

Suppose we have a change of environment $\omega \longrightarrow \omega'$. This results in a change $\gamma = f(\omega) \longrightarrow f(\omega') = \gamma'$. *Assume* that

$$P_f (\gamma) = P_f (\gamma') = f ,$$

i.e. the repair mechanism is capable of correcting for the environmental change. Then we have

$$(\beta_f \circ P_f) (\gamma) = (\beta_f \circ P_f) (\gamma') = P_f ,$$

implying that the replication operation is unaffected by the environmental change. That is, Lamarckian-type changes in β_f cannot occur under any type of environmental change that can be corrected by the repair operation P_f. Theorem 3 characterizes just what sorts of changes fall into this category.

Under Rosen's scheme, it is shown in [7] that *no* environmental change of any sort can lead to Lamarckian changes in β_f, a vastly stronger result but, as noted, under extremely restrictive hypotheses.

6. Linear (M,R)-Systems : a Summary

Our development of the realization theory for linear (M,R)-systems has been somewhat lengthy, in order to allow considerable commentary on the basic set-up and properties of these objects. Here we summarize the entire development in the following diagram.

(Metabolism): $\Omega \xrightarrow{f} \Gamma$

$$\omega = \{u_0, u_1, \ldots\} \overset{\{A_1, A_2, \ldots\}}{\underset{A_i \in R^{p \times m}}{\longmapsto}} \gamma = (y_1, y_2, \ldots), \quad u_i \in R^m$$

$$y_t = \sum_{i=0}^{t-1} [A_{t-i}^{(1)} \mid A_{t-i}^{(2)} \mid A_{t-i}^{(m)}] \, S(u_i) \qquad t = 1,2,\ldots$$

$$f = \{A_1, A_2, \ldots\} \approx \begin{bmatrix} A_1 & 0 & 0 & \cdots \\ A_2 & A_1 & 0 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix} \approx \begin{bmatrix} A_1 & A_2 & A_3 & \cdots \\ A_2 & A_3 & A_4 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix}$$

(Repair): $\Gamma \xrightarrow{P_f} H(\Omega, \Gamma)$

$$\gamma = (y_1, y_2, \ldots) \overset{\{R_1, R_2, \cdots\}}{\underset{R_i \in R^{p \times pm}}{\longmapsto}} f \approx \{A_1, A_2, \ldots\}$$

$$A_\tau = \sum_{i=0}^{\tau-1} [R_{\tau-i}^{(1)} \mid R_{\tau-i}^{(2)} \mid \cdots \mid R_{\tau-i}^{(p)}] \, S(y_i), \quad \tau = 1,2,\ldots$$

$$P_f = \{R_1, R_2, \cdots\} \approx \begin{bmatrix} R_1 & 0 & 0 & \cdots \\ R_2 & R_1 & 0 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix} \approx \begin{bmatrix} R_1 & R_2 & R_3 & \cdots \\ R_2 & R_3 & R_4 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix}$$

(Replication): $H(\Omega, \Gamma) \xrightarrow{\beta_f} H(\Gamma, H(\Omega, \Gamma))$

$$f = \{A_1, A_2, \cdots\} \overset{\{U_1, U_2, \ldots\}}{\underset{U_i \in R^{p \times p^2 m^2}}{\longmapsto}} P_f = \{R_1, R_2, \ldots\}$$

$$R_\sigma = \sum_{i=0}^{\sigma-1} [U_{\sigma-i}^{(1)} \mid U_{\sigma-i}^{(2)} \mid \cdots \mid U_{\sigma-i}^{(pm)}] \, S(A_i), \quad \sigma = 1,2,\ldots$$

$$\beta_f = \{U_1, U_2, \ldots\} \approx \begin{bmatrix} U_1 & 0 & 0 & \cdots \\ U_2 & U_1 & 0 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix} \approx \begin{bmatrix} U_1 & U_2 & U_3 & \cdots \\ U_2 & U_3 & U_4 & \cdots \\ \vdots & \vdots & \vdots & \cdots \end{bmatrix}$$

Assuming that the metabolic component has a finite-dimensioned realization, so do the repair and replication components, and these canonical realizations can all be computed by means of Ho's Algorithm. Furthermore, the dimensions of the realizations for the repair and replication systems will be no larger than that of the metabolic subsystem. Thus, ***any*** finitely realizable metabolism can be a metabolism-repair system using the constructions detailed here.

7. Discussion

The formalism given here for linear (M,R)-systems generates a long list of questions, problems and extensions of the classical "metabolism-only" Newtonian framework meriting further study. High on this list are problems concerned with networks, mutation and nonlinearity. Here we only touch upon a few of the major issues.

A. Networks - we began in Section 2 with a discussion of (M,R)- ***networks***, emphasizing the role of the repair mechanism as an object whose inputs generally

come from other cells in the network. In particular, we noted that a repair component needed to receive *all* of its inputs in order to function, so that if one of the inputs was from its own associated metabolism, then the removal of that metabolism would also incapacitate the repair subsystem. We then immediately shifted attention away from networks and considered only a single (M,R)-unit. This clearly involves a different interpretation of how the repair and replication components interface with the metabolism. As we have noted above, instead of imagining the metabolism to be removed, we consider what happens when there is an environmental change or when the metabolic machinery acts, but imperfectly. These considerations bring us up against the question of just how to interpret the action of the serially-connected metabolism-repair-replication subsystems.

Naively, we could imagine that the time-scales of operation of the subsystems are so disparate that the systems operate non-concurrently. In other words, the metabolic subsystem first processes ω into γ. When this operation is *complete*, the output γ is processed by the repair system and, finally, when the repair operation terminates, the replication process begins. Of course, real cells never operate in this fashion and this simple scheme can only be thought of as a convenient approximation when the time-scales are such that $\Delta t \ll \Delta\tau \ll \Delta\sigma$.

More realistically, the three subsystems operate concurrently with the differences in time-scales introducing time-lags into the repair and replication operations, relative to metabolic time. In this case, we must drop the mathematical fiction of infinitely long input and/or output sequences and assume that ω is of finite duration, with the metabolic output $\gamma = f(\omega)$ also of finite length. In these situations, the mathematical formalism requires the full machinery of the so-called Partial Realization Problem and its attendant version of Ho's Algorithm [14,16]. Since this is a matter of some delicacy, we defer its treatment to a later paper.

Returning to the problem of (M,R)-networks, as soon as we couple several (M,R)-units as in Section 2, we immediately encounter a new set of mathematical questions surrounding the operation and behavior of the network. For example, each unit of the network has its own characteristic time-scales for its metabolic, repair and replication operations. How do these time-scales interact to produce the global network behavior ? Also, there may be transport delays in passage of input materials from one cell to where it's needed for another's repair system. If this delay is too great, the receiving repair system may fail to operate. How can we build this type of delay into the mathematical formalism ? Finally, we encounter questions about the overall stability of the network. The principal questions of concern involve the "viability" or "resilience" of the network to various types of local perturbations. One such local disturbance might involve the breakdown of the metabolic-repair-replication sequence of a group of cells. Another class of disturbances would arise when we consider the dynamical behavior of an individual cell. We know from Theorem 1 that there must exist cells that cannot be repaired and that if there are only a "small" number of such cells, the removal of the non-repairable cells will result in the collapse of the entire network. How small is "small" ? It's at least 1, but can it be larger ? The answer seems to involve the connective pattern in the network. Also, how can we identify the non-reestablishable elements ? And how resilient is the network to mutations, in which the metabolism just changes, rather than dies ? These are typical questions of the type we can only begin to address if we have a good mathematical formalism at hand for characterizing the (M,R)-networks.

B. Mutations and Selection - we have already noted that a change in the replication map β_f corresponds directly to what in biology is termed a "mutation". It is at this point that by incorporating a selection mechanism into our set-up, we can use the (M,R)-framework to study the evolutionary behavior of either a single

(M,R)-cell or, more appropriately, a network of such cells. The identity

$$\text{adaptation} \equiv \text{mutation} + \text{selection}$$

allows us to talk about various types of goal-oriented behaviors (via directed mutations and/or modified selection processes), thereby incorporating anticipatory behavior into our set-up in a mathematically and physically natural fashion.

From a technical perspective, the problems of mutation and selection pose several challenges. The first just involves tracing out the effects on the metabolism of any particular change in the replication process $\beta_f \longrightarrow \beta_f'$. Since β_f does not act directly upon f, but only upon the genetic repair map P_f, there is an added level of difficulty involved in ascertaining the precise relationship between β_f and f. In Nature it is usually assumed that mutations arise from random events impinging upon the system from the outside; viewing the (M,R)-set-up as a metaphor for social and behavioral phenomena, this assumption is usually not tenable. Very likely, we will need to consider ***directed mutations*** arising either within the system itself (by feedback, say), or imposed upon the system by an outside controller. In the first case, we have the problem of incorporating the relevant feedback loops into the mathematical formalism; the second case is formally equivalent to the case of naturally-induced mutations, but emphasizes the importance of determining a direct path from β_f to f. This represents a new type of control process, not yet dealt with in the literature.

The second half of the above "adaptive identity" necessitates the superposition of a criterion of "goodness" upon the behavioral output of our (M,R)-network. Formally this problem is a typical one faced in control theory: determination of the system objective function. However, for (M,R)-networks we have a very different situation insofar as the interaction between the controls and the system behavior is concerned. First of all, there may be many different types of controls acting simultaneously (environmental changes in ω, mutations in β_f, changes in metabolism

f, etc.). In addition, the primary goal of living systems is not really optimality, but rather viability. Somehow, the selection process has to be developed to serve two conflicting needs at once: the need to specialize to exploit a particular eco-niche, and the need to generalize in order to remain viable under a variety of unknown, and probably unknowable, environmental disturbances and random mutations. The framework given above provides us with a vehicle for the detailed exploration of such questions.

C. Nonlinearity - our treatment has focused upon linear metabolic, repair and replication maps. It's fair to ask to what degree the results and conclusions we have drawn rely upon this obvious physical fiction. The answer: it depends. At the level of *abstract* input/output maps and their *abstract* realization by canonical dynamical systems, there is no problem. Relatively recent results in nonlinear system theory assert the existence of such objects under very weak hypotheses on the input/output behavior. However, at the level of the actual *construction* of the relevant state-space and dynamics (the Ho Algorithm level), much depends upon narrowing down the term "nonlinear". For large classes of nonlinear maps (multilinear, polynomial, linear-analytic, piecewise-linear,...), various extensions of Ho-type algorithms are possible; however, a general nonlinear f is just *too* general for any kind of specific construction. So, the degree to which we can actually carry out the operations outlined in Section 3 for nonlinear behaviors depends upon the degree to which we can precisely specify the type of nonlinearity involved and the degree to which that nonlinearity deviates from a linear structure. A reasonably up-to-date account of these matters is found in [15,20].

D. Applications - we should not fail to mention some of the applications to which a decent theory of (M,R)-systems can be directed. Leaving aside the obvious biological questions which motivated Rosen's original introduction of the (M,R)-

concept, there are numerous social and behavioral settings that appear to fit nicely into the overall (M,R)-scheme. For example, in [21] there is a treatment of technological development within a network of industrial firms using the (M,R)-ideas. While this work is preliminary, it appears to hold promise for shedding light on a number of issues currently of interest in the general area of flexible manufacturing systems. In another direction, the biologically-based arguments we have presented seem to be completely in line with recent trends in economics, in which an evolutionary view of economic processes has been promoted by Boulding [22], Nelson and Winter [23] and others as a means of breaking out of the Newtonian-based equilibrium-centered economic paradigm. Finally, there are the various approaches to an evolutionary view of social organizations, starting with Spencer and the Social Darwinists, and continuing on through Spengler and Toynbee and on down to the present-day work of Jantsch [24], Weidlich and Haag [25], Axelrod [26] and others. The (M,R)-paradigm holds out the promise of offering a formal structure within which to state and address many of the most pressing questions standing in the path of a deeper understanding of these areas.

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