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# ON THE MODELING AND CONTROL OF BIOLOGICAL SIGNALING CHAINS

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**Abstract**

Sequential machines are proposed as mathematical models for biological signaling chains, a class of critical biochemical reactions that affect all live organisms. The problem of correcting malfunctions in such models is then addressed in the context of a model matching control problem. A solution is provided to the problem of model matching by static controllers. This note is a summary of HAMMER [1995].

## 1. Introduction

The development of biochemical techniques for the correction of malfunctioning biological processes *in vivo* has been an important theme in modern medical research. Such techniques could provide inroads into the alleviation or the cure of numerous impairments, including genetic defects, debilitative geriatric processes, effects of certain viral infections, and pre-cancerous or cancerous transformations of cells. Interventive biochemical techniques have achieved a number of successes over the years; Still, in many ways, the development of techniques to correct malfunctions of biological processes *in vivo* is largely in its infancy.

As is well known, biological cells are complex dynamical systems, driven by numerous inter-dependent biochemical processes that operate in a delicate balance. It is therefore natural to expect that corrections of cellular malfunctions would, in many cases, have to be performed in a dynamical manner: An orderly sequence of corrective actions would need to be applied, with each corrective action depending upon the conditions within the affected cells at that time. In other words, the correction would need to be executed by an artificial system, commonly called a *controller*.

In the present context, the controller will execute corrective actions so as to drive the malfunctioning cell toward desirable behavior; It will monitor the conditions within the cell, and provide a temporally variant supplementary population of molecules to correct deficiencies.

The application of control techniques to the study and correction of biochemical processes depends on a two-way interaction between the areas of control theory and molecular biology: First, data from molecular biology is used to construct a mathematical model of the relevant biochemical processes; then, based on the model, control theoretic techniques are used to derive a mathematical model of a controller that corrects the impairments; and, finally, tools of molecular biology are used to implement the corrective controller as a biochemical entity.

The present paper concentrates on some aspects of the application of control theory to biology. First, we discuss the mathematical modeling of biological signaling chains, which are critical biological processes that seem to be involved in a substantial class of adverse impairments of cell function. Then, we concentrate on the structure of static corrective controllers, since static controllers are among the simplest.

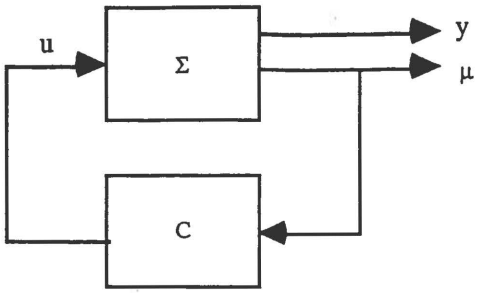
In basic terms, a biological signaling chain is a sequence of biochemical events that starts with the appearance of a signaling molecule, proceeds through several biochemical stages that involve the creation of other molecules, and ends with the creation of an operational molecule that affects the function of a gene, or of another entity within a cell. A signaling chain can be modeled as a sequential machine (see Section 2 for details).

An important concern in the development of control algorithms for biological systems is the fact that biological data is, to a substantial degree, incomplete and inaccurate. Our discussion concentrates therefore on the derivation of controllers for cases where there is a substantial uncertainty about the exact mathematical model of the controlled system.

Since an accurate model of the biological system is not available, we consider instead a family $M$ of $q$ *potential models* $\Sigma_1, ..., \Sigma_q$ of the system. Each one of the potential models is a sequential machine. The actual model of the system, which we call the *active model*, is one of $\Sigma_1, ..., \Sigma_q$, but it is not known apriori which one. The controller is designed so as to achieve the desired objective irrespective of which one of the potential models is the active model.

Each sequential machine model $\Sigma$ accepts a multivariable input sequence $u$, and generates two multivariable sequences: an *output sequence* $y$ that constitutes the response of the system; and a *monitoring sequence* $\mu$ that consists of quantities that are continuously monitored and measured, and can be used for feedback purposes. The monitoring sequence serves as the input of a sequential controller $C$ which, in turn, generates the input sequence of $\Sigma$.

(1.1)




The controller C employs no external prompting or reference, creating the necessary input sequence for $\Sigma$ all automatically. For this reason, we call C an *autonomous controller*. The closed loop system depicted in the diagram is denoted by $\Sigma_c$. Since $\Sigma_c$ has no external inputs, we refer to it as an *autonomous system*.

The purpose of the controller C is to drive the system $\Sigma$ so that the resulting behavior of the closed loop system $\Sigma_c$ is desirable. We concentrate on the following.

(1.2) The Model Matching Problem. Let $\Sigma$ be a system having the family $M = \{\Sigma_1, ..., \Sigma_q\}$ of potential models. With each potential model $\Sigma_i$, associate an autonomous system $\Delta_i$ called the *desired model*. Denote by $\Sigma_{ic}$ the system obtained by closing the loop around $\Sigma_i$ through a controller C.

(i) Determine whether or not there is a controller C that satisfies the conditions

(1.3) $\Sigma_{ic} = \Delta_i$, i = 1, ..., q,

i.e., whether or not there is a (single) controller C that transforms each one of the potential models into its corresponding desired model; and

(ii) Design a controller C that satisfies condition (1.3), if one exists. ♦

A solution to the model matching problem is provided in Section 3.

Sequential machines have been used for quite some time to model various biological entities (e.g., RASHEVSKY [1948], M. SUGITA [1963], von NEUMANN [1966], LINDENMAYER [1968], KAUFFMAN [1969], ROSENBERG and SALOMAA [1975], IEEE [1974], HAMMER [1993, 1994a and b], the references cited in these works, and many others). The material in the paper relates to the theory of sequential machines and discrete event systems, and thus relates to GINSBURG [1962] and [1966], EILENBERG [1974], HOARE [1976], MILNER [1980], ARNOLD and NIVAT [1980], RAMADGE and WONHAM [1987], the references listed in these papers, and many other excellent sources.

## 2. Basic Models

### 2.1. Signaling chains in biology.

Many critical processes in cell biology seem to involve signaling chains (e.g., ALBERTS, BRAY, LEWIS, RAFF, ROBERTS, and WATSON [1994, Ch. 9]). In general terms, a signaling chain is a cascade of biochemical events. It proceeds from the appearance of an activating molecule - through the generation of a number of intermediary molecules - to the creation of a final product. Signaling chains transfer biochemical information from one part of the cell (or of the organism) to another. Here is an outline of a signaling chain.

The signaling chain starts when a molecule of a signaling ligand becomes attached to a receptor protein and activates it. The activated receptor protein performs an enzymatic function, which leads to the activation of a number of G protein molecules. Each activated G protein molecule releases one of its subunits, which, upon encountering an adenylyl cyclase enzyme molecule, activates it. Each activated adenylyl cyclase molecule catalyses then the generation of a large number of cAMP molecules. In the next step, each cAMP molecule activates an A-kinase enzyme molecule upon encounter. The activated A-kinase enzyme molecule catalyzes the activation of a large number of molecules of another enzyme. Finally, each molecule of the last enzyme catalyzes the creation of a number of product molecules as the final step of the signaling chain. In brief, a signaling chain is an orderly sequence of biochemical events, proceeding in discrete well defined steps.

Signaling chains are at the heart of numerous biological functions, including muscle activation, vision, smell, wound healing, the cell growth and division cycle, and others. Impairments in the function of signaling chains are believed to cause many disorders, including pre-cancerous and cancerous transformations of cells (e.g., ALBERTS, BRAY, LEWIS, RAFF, ROBERTS, and WATSON [1994, Ch. 24]). The understanding of signaling chains is critical to the study and the potential cure of numerous disorders.

In many cases, the final product of a signaling chain is a gene regulatory protein -- a gene activator, a gene repressor, or a gene enhancer. These proteins regulate the function of genes by turning gene transcription on or off, or by affecting the rate of gene transcription. The regulation of gene transcription is done through the gene promoter, which is a segment of DNA near the gene to which regulatory proteins can bind. A gene promoter turns on the transcription of its gene at a certain rate when all appropriate gene activator and gene enhancer protein molecules become bound to it, while all relevant gene repressor protein molecules are not bound to it. In other words, the gene promoter activates gene transcription at a certain rate when a specific combinatorial condition is met, and whence the function of a gene promoter can be modeled by a combinatorial logic circuit (e.g., ALBERTS, BRAY, LEWIS, RAFF, ROBERTS, and WATSON [1994, Ch. 9]).

In some cases, the number of regulatory proteins that affect a gene is quite large, and some regulatory proteins are products of signaling chains. Thus, the expression of

a mammal gene may depend on a large number of signaling chains, the final products of which all converge onto the same gene promoter. Furthermore, sometimes the activation of a gene forms a link within a larger signaling chain. In such case, a ligand molecule leads to the activation of the first part of a signaling chain, which leads to the activation of the gene; the gene product initiates then the continuation of the signaling chain, which may ultimately lead to the activation of a second gene, and so on. Thus, it is possible for an important biological function to be regulated through an interaction between a number of signaling chains and gene promoters (ALBERTS, BRAY, LEWIS, RAFF, ROBERTS, and WATSON [1994] and the references listed there).

### 2.2. Mathematical models.

Signaling chains and combinations of signaling chains are natural candidates for modeling by sequential machines. Consider a biochemical system $\Sigma$ that operates within a medium. Let $\sigma^1, ..., \sigma^m$ denote the species of molecules that may appear within the medium, and that significantly affect the operation of the system $\Sigma$. Then, the state of the molecular population at an instant $k$ of time can be characterized by a vector $\pi_k = (\pi_k^1, \pi_k^2, ..., \pi_k^m)$ of integers, where $\pi_k^i$ is the population (or is nearly proportional to the population) of molecules of the species $\sigma^i$ present within the medium at the instant $k$. We write $\pi_k \in Z^m$, where $Z^m$ is the set of all $m$-dimensional vectors with integer components.

In addition to the molecular species present within the medium, the state of the biochemical system $\Sigma$ may also be affected by other quantities, including the temperature of the medium, or the irradiation intensity within a specified narrow spectral range. Usually, discretized measures of these quantities can be used to describe their level. For instance, consider the temperature. In biochemical experiments, the temperature is usually varied only between two discrete levels, a 'high' temperature and a 'normal' temperature; this is done to activate markers, or to elicit a desired selection process.

Let $\tau_k^1, ..., \tau_k^d$ be integers that represent discretized measures (at the time $k$) of quantities other than molecular populations that affect the biochemical system $\Sigma$. We then create the augmented vector $s_k := (\pi_k, \tau_k^1, ..., \tau_k^d) \in Z^n$, where $n := m + d$; This vector represents all the significant information pertaining to the state of the biochemical system $\Sigma$ at the time $k$. In this way, the (discretized) state of the biochemical system at the time $k$ becomes represented by a vector of integers.

Consider now the case where $\Sigma$ is a biochemical signaling chain, i.e., a sequential biochemical process that proceeds in discrete steps. As before, the state of the signaling chain at a step $k$ is represented by a vector of integers $s_k \in Z^n$. At each step, the biochemical conditions within the reaction system can be altered by externally injecting into its medium additional molecules, or by changing the temperature or the irradiation level. These alterations in the biochemical conditions can be represented by an additive input vector $u_k \in Z^n$; each component of $u_k$ represents the number of molecules of the respective species that were injected into the medium, or the increment in temperature, or the increment in irradiation intensity, or the increment in another additive variable. After the alteration process, the state of the biochemical system becomes $(s_k + u_k)$.

Thus, immediately preceding step $k+1$ of the signaling chain, the state of the biochemical system is given by $(s_k + u_k)$. This state determines the outcome of reaction step $k+1$, which is described by the state $s_{k+1}$. Consequently, there is a function $f : Z^n \to Z^n$ such that

(2.2.1) $\quad s_{k+1} = f(s_k+u_k), k = 0, 1, 2, ...$

The initial condition $s_0$ of the reaction system at the start of the signaling chain has to be provided. The function $f$ is called the *recursion function* of the reaction, and it is determined from empirical data.

Of course, to be precise, one needs to use probabilistic models to describe the results of a reaction step. The deterministic model (2.2.1) may be regarded as the average outcome of a reaction step.

Under normal conditions, only certain combinations of state values can arise in a biochemical reaction system. Let $D$ be the subset of $Z^n$ that corresponds to all possible state values that can appear in the reaction system being considered. Then, the recursion function $f$ has to satisfy the requirement $f : D \to D$, i.e., the function $f$ must map each state in $D$ into a state in $D$. We refer to $D$ as the *state domain* of the system. Similarly, there are restrictions on the external inputs that can be applied to the system. To represent such restrictions, we let $D_{in} \subset Z^n$ be the set of all permissible input vectors. We impose the requirement $u_k \in D_{in}$, $k = 0, 1, 2, ...$ The set $D_{in}$ is called the *input domain.*

A comment on negative input values is in order now. For components of the input vector $u_k$ that represent temperature or irradiation intensity, negative values simply indicate a reduction of the temperature or of the irradiation intensity, as the case may be. For a component of $u_k$ that corresponds to a molecular species, negative values represent the injection of molecules that destroy the activity of that molecular species. For instance, for regions of RNA or DNA molecules, activity can be destroyed by the injection of anti-sense molecules. For components of the input vector $u_k$ or of the state vector $s_k$ that are prohibited from attaining negative values, the appropriate components of the input domain $D_{in}$ or of the state domain $D$, respectively, are restricted to non-negative integers.

In order to create a feedback controller for the biochemical system $\Sigma$, it is necessary to monitor the state of $\Sigma$.

Let $\mu_k$ be the result returned by the monitoring equipment at step k. Then, since $\mu_k$ is determined by the state $s_k$ of $\Sigma$, we write

(2.2.2) $\mu_k = \psi(s_k)$, k = 0, 1, 2, ...

Here, $\psi$ is called the *monitoring function;* It describes the data provided by the measuring equipment. For the sake of uniformity, we shall also represent the outcome of the monitoring function as a vector of integers, so that $\psi : D \to Z^r$; The integer r indicates the number of values returned by the monitoring process at each step. In case the monitoring equipment measures the actual state of the system, $\psi$ becomes the identity function and $\mu_k = s_k$. The monitoring function $\psi$ is designed as part of the control design process, to provide an adequate feedback signal that facilitates the operation of the controller.

The system $\Sigma$ also generates an output signal y that serves to activate or influence other reaction systems within the cell, within the organism, or within the environment. The value $y_k$ of the output signal at the step k is determined, again, by the state $s_k$, and is given by

(2.2.3) $y_k = h(s_k)$, k = 0, 1, 2, ...,

where h is called the *output function*. To summarize, our biochemical system $\Sigma$ is described by a sequential machine over the integers, given by (2.2.1), (2.2.2), and (2.2.3).

### 2.3. Static Controllers.

A controller is a device that guides the controlled system toward the achievement of a prescribed objective. Referring to (1.1), a static controller is described by

(2.3.1) $u_k = h_c(\mu_k)$, k = 0, 1, 2, ...

where $h_c$ is a function called a *static output feedback function;* Denoting by $D_\mu \subset Z^r$ the domain of monitoring values for the system $\Sigma$, we require that $h_c : D_\mu \to D_{in}$.

Considering the system $\Sigma$ of (2.2.1) and (2.2.2), we obtain

(2.3.2) $u_k = h_c\psi(s_k)$, k = 0, 1, 2, ...,

where $\psi : D \to D_\mu$ is the monitoring function of $\Sigma$. In particular, when the entire state $s_k$ of $\Sigma$ is measured, $\psi$ is the identity function, and (2.3.2) takes the form $u_k = h_c(s_k)$, k = 0, 1, 2, ... This case represents a *static state feedback controller*.

The basic objective of the present paper is to derive the set of all pairs of functions $(\psi,h_c)$ for which the closed loop system (1.1) has prescribed characteristics. The function $\psi$ represents then the necessary measurements that need to be taken in real time, whereas the function $h_c$ represents the controller.

Current recombinant DNA technology provides a foundation for the implementation of biochemical intracellular controllers. Since static controllers represent combinatorial logic circuits, they can, in principle, be implemented by mimicking the natural mechanisms of gene promoters.

## 3. Model Matching and Static Controllers

We start with the case where the model of the system is known.

### 3.1. Model matching for a known system.

Consider a system $\Sigma$ whose model is known precisely, so that the family of potential models of $\Sigma$ consists of a single member. Let

(3.1.1) $s_{k+1} = f(s_k+u_k)$, k = 0, 1, 2, ...,

be the recursive representation of the model, let

(3.1.2) $\mu_k = \psi(s_k)$

be its monitoring sequence, let $D_{in}$ be its input domain, and let D be its state domain. A static controller for $\Sigma$ is given by

(3.1.3) $u_k = h_c(\mu_k) = h_c\psi(s_k)$,

where $h_c$ is the controller function. Referring to the model matching problem (1.2), let $\Delta$ be the autonomous model that needs to be matched, and let

(3.1.4) $s_{k+1} = g(s_k)$, k = 0, 1, 2, ...,

be a recursive representation of $\Delta$. In this subsection we assume that $\Sigma$ and $\Delta$ have the same state dimension and the same state domain. The recursive representation of the closed loop system $\Sigma_c$ of (1.1) is then given by

(3.1.5) $s_{k+1} = f(s_k + h_c\psi(s_k))$, k = 0, 1, 2, ...

Allowing the input domain $D_{in}$ of the controlled system $\Sigma$ to equal its state domain D, we obtain the following simple necessary and sufficient condition for model matching by a static controller.

(3.1.6) THEOREM. Let $\Sigma$ and $\Delta$ be recursive systems described by (3.1.1) and (3.1.4), with the recursion functions f and g, respectively. Assume $\Sigma$ and $\Delta$ have the same state domain D, and that the input domain $D_{in}$ of $\Sigma$ equals D. Then, (i) and (ii) are equivalent.

(i) There are a monitoring function $\psi$ and a static controller C that transform $\Sigma$ into $\Delta$ via the closed loop (1.1).

(ii) Im g $\subset$ Im f.

As we can see from the Theorem, the solution to the present model matching problem is quite simple. One has to compare the set of all possible values of the recursion function f of $\Sigma$ with the set of all possible values of the recursion function g of $\Delta$. A static controller that achieves the required model matching exists then if and only if the former contains the latter. Furthermore, when condition (ii) of the Theorem holds, there is a function $\phi$ satisfying $g = f\phi$; Then, for each choice of $\phi$, one can obtain a controller by setting

(3.1.7) $\begin{cases} \psi := \phi - I, \\ h_c := I. \end{cases}$

(see HAMMER [1995]) for details).

### 3.2. Model matching in different dimensions.

We consider now the case where the state space dimension of the model $\Delta$ that needs to be matched is different from the state space dimension of the system $\Sigma$ being controlled. Let the recursive representation of $\Delta$ be

(3.2.1) $\quad x_{k+1} = g(x_k),\ k = 0, 1, 2, ...,$

where $x_k \in Z^\gamma$ for all integers $k \geq 0$, and $\gamma$ is the number of components of the state of $\Delta$. As before, n denotes the number of components of the state vector s of $\Sigma$. We distinguish between two cases: $\gamma < n$ and $\gamma > n$ (the case $\gamma = n$ was discussed earlier).

First, the case $\gamma < n$. Recall that our models were built so that each component of the state vector represents a concrete physical quantity, like the population of a certain molecular species, or the temperature or irradiation intensity. This physical interpretation of the state components has to be preserved, of course, when comparing the state components of the system $\Sigma$ to the state components of $\Delta$. Each component of the state x of $\Delta$ corresponds to a specific component of the state s of $\Sigma$. For the sake of notational simplicity, assume that component i of x corresponds to component i of s, i = 1, ..., $\gamma < n$.

Now, augment the vector x to obtain a vector of dimension n by adding components $x^{\gamma+1}, ..., x^n$, and create the augmented system $\Delta_a$

$$(3.2.2) \qquad \begin{pmatrix} x_{k+1} \\ x^{\gamma+1}_{k+1} \\ \cdots \\ x^{n}_{k+1} \end{pmatrix} = \begin{pmatrix} g(x_k) \\ 0 \\ \cdots \\ 0 \end{pmatrix} \quad k = 0, 1, 2, ...$$

The new components $x^{\gamma+1}, ..., x^n$ correspond to the physical quantities represented by the components $s^{\gamma+1}, ..., s^n$ of the state vector s of $\Sigma$. The initial conditions for the new components are zero, i.e., $x^{\gamma+1}_0 = 0, ..., x^n_0 = 0$, so that, in view of the recursion (3.2.2), $x^{\gamma+1}_k = 0, ..., x^n_k = 0$ for all integers $k \geq 0$. Consequently, the system $\Delta_a$ of (3.2.2) is physically the same as the system $\Delta$ represented by (3.2.1); The added components $x^{\gamma+1}, ..., x^n$ of the sate vector are not populated at any step.

Now, $\Delta_a$ and $\Sigma$ have the same number of state components, and whence Theorem (3.1.6) directly applies. In this way, the case $\gamma < n$ is reduced to the case discussed in Subsection 3.1.

Consider next the case $\gamma > n$. Here, we augment the state vector of $\Sigma$ so as to increase the dimension from n to $\gamma$. To simplify the notation, assume that the first n components of the state vector x of $\Delta$ represent the same biochemical quantities as the components of the state vector s of the system $\Sigma$; The $(\gamma - n)$ components by which the state vector of $\Sigma$ is augmented represent the same quantities as the last $(\gamma - n)$ components of the state vector x of $\Delta$. We use an augmenting system $\Phi$ to augment the system $\Sigma$. Let $\Phi$ be a system with the recursive representation

(3.2.3) $\quad \sigma_{k+1} = \varphi(\sigma_k + v_k),\ k = 0, 1, 2, ...,$

where $\sigma \in Z^{\gamma-n}$ is the state vector and $v \in Z^{\gamma-n}$ is the input vector. In a biological context, $\Phi$ represents a new biochemical reaction system that is injected into the same medium within which $\Sigma$ operates. Of course, the two systems may interact, and the recursive representation of the augmented system takes the form

$$(3.2.4) \qquad \begin{pmatrix} s_{k+1} \\ \sigma_{k+1} \end{pmatrix} = \begin{pmatrix} f(s_k + \chi(\sigma_k) + u_k) \\ \varphi(\sigma_k + \xi(s_k) + v_k) \end{pmatrix}, \quad k = 0, 1, 2, ...$$

Here, the functions $\chi : Z^{\gamma-n} \to Z^n$ and $\xi : Z^n \to Z^{\gamma-n}$ represent the interactions between the two systems. The interactions are additive, since they represent changes in molecular populations. The combined system has the input vector $(u_k, v_k)$. Let $(\Sigma,\Phi)$ be the augmented system of (3.2.4), and denote

$$(3.2.5) \qquad f_e(s,\sigma,u,v) := \begin{pmatrix} f(s+\chi(\sigma)+u) \\ \varphi(\sigma+\xi(s)+v) \end{pmatrix}.$$

For the sake of simplicity, we assume that there are no restrictions on the selection of the recursion function $\varphi : Z^{\gamma-n} \to Z^{\gamma-n}$ of $\Phi$, and that the input domain of $\Phi$ is equal to its state domain.

Let $\psi : Z^\gamma \to Z^\gamma : (s,\sigma) \mapsto \psi(s,\sigma) =: \mu$ be a monitoring function for the system (3.2.4), and let $h_c : Z^\gamma \to Z^\gamma : \mu \mapsto (h^u_c(\mu), h^v_c(\mu)) = (u,v)$ be a static controller function. When the static controller is combined with the system (3.2.4) through the monitoring function $\psi$, we obtain the autonomous system

$$(3.2.6) \qquad \begin{pmatrix} s_{k+1} \\ \sigma_{k+1} \end{pmatrix} = \begin{pmatrix} f(s_k + \chi(\sigma_k) + h^u_c\psi(s_k,\sigma_k)) \\ \varphi(\sigma_k + \xi(s_k) + h^v_c\psi(s_k,\sigma_k)) \end{pmatrix},$$

$k = 0, 1, 2, ...$ In order to match the model $\Delta$, it is then necessary to find a recursion function $\varphi$, a static controller function $h_c$, and a monitoring function $\psi$, so that the recursion function of (3.2.6) becomes identical to the recursion function g of $\Delta$. To this end, let $\pi^n : Z^\gamma \to Z^n : (z_1, ..., z_\gamma) \mapsto (z_1, ..., z_n)$ be the standard projection onto the first n coordinates. Then, the following is true.

(3.2.7) THEOREM. Let $\Sigma$ and $\Delta$ be the recursive systems of (3.1.1) and (3.2.1), having the recursion functions f and g, respectively, where the state dimension of $\Delta$ exceeds that of $\Sigma$. Assume that the input domain $D_{in}$ of $\Sigma$ equals D, and that there are no restrictions on the selection of the augmenting system $\Phi$ of (3.2.3). Then the following are equivalent.

(i) There are an augmenting system $\Phi$, a monitoring function $\psi$, and a static controller C for which the augmented closed loop system $(\Sigma,\Phi)_c$ is equal to $\Delta$.

(ii) $\mathrm{Im}\ \pi^n g \subset \mathrm{Im}\ f$.

We comment that the combination of an augmenting system and a static controller is equivalent to the use of a

dynamic controller. Thus, the results of the present subsection extend our discussion to the general case of dynamic controllers.

### 3.3. Model matching for families of systems.

We consider now the case where an exact model of the system $\Sigma$ is not known, and $\Sigma$ is represented by a family $M = \{\Sigma_1, ..., \Sigma_q\}$ of potential models. Each model $\Sigma_i$, $i = 1, ..., q$, has a recursive representation of the form

(3.3.1) $s_{k+1} = f_i(s_k+u_k)$, $k = 0, 1, 2, 3, ...$

with the initial condition $s_0$. Referring to the model matching problem (1.2), we denote by $\Delta_i$ the desired model corresponding to $\Sigma_i$, $i = 1, ..., q$. The model $\Delta_i$ has the recursive representation

(3.3.2) $s_{k+1} = g_i(s_k)$, $k = 0, 1, 2, ...$;

we assume at first that the state components of $\Delta_i$ correspond exactly to the state components of $\Sigma_i$. The objective is to find a monitoring function $\psi$ and a static controller $C$ so that the condition $\Sigma_{ic} = \Delta_i$ holds for all $i = 1, ..., q$. Recall that only one monitoring function and a single controller are used for all members of the family $M$, since it is not known in advance which member is active.

The case of multiple potential models can be reduced to the single model problem discussed in Subsections 3.1 and 3.2. To this end, use the recursion functions $f_1, ..., f_q$ of $\Sigma_1, ..., \Sigma_q$, and the recursion functions $g_1, ..., g_q$ of $\Delta_1, ..., \Delta_q$ to define the two vector functions

(3.3.3) $$f := \begin{pmatrix} f_1 \\ ... \\ f_q \end{pmatrix}, \quad g := \begin{pmatrix} g_1 \\ ... \\ g_q \end{pmatrix}.$$

Then, the following analog of Theorem (3.1.6) is true.

(3.3.4) THEOREM. Let $M = \{\Sigma_1, ..., \Sigma_q\}$ be the family of potential models of $\Sigma$, and let $\Delta_i$ be the desired model associated with the potential model $\Sigma_i$, $i = 1, ..., q$. Assume that all the systems $\Sigma_1, ..., \Sigma_q, \Delta_1, ..., \Delta_q$ have the same state set $D$, and that $\Sigma_1, ..., \Sigma_q$ have an input set equal to their state set. Then, the following are equivalent.

(i) There are a monitoring function $\psi$ and a static controller $C$ that solve the model matching problem for the family $M$ of potential models of $\Sigma$.

(ii) Im $g \subset$ Im $f$, where $f$ and $g$ are given by (3.3.3).

Thus, we see that the solution of the model matching problem for a family of potential models is similar to the solution for a single model.

The case where $\Sigma_i$ and $\Delta_i$ differ in their state dimensions can be treated by applying the discussion of Subsection 3.3 to the vectors $f$ and $g$ of (3.3.3). For further details see HAMMER [1995].

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