Artificial Homeostasis: Integrating Biologically Inspired Computing

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Abstract

We present a framework which integrates artificial neural networks, artificial immune systems and a novel artificial endocrine system. The natural counterparts of these three components are usually assumed to be the principal actors in maintaining homeostasis within biological systems. This paper proposes a system which promises to capitalise on the self-organizing properties of these artificial systems to yield artificially homeostatic systems. The components develop in a common environment and interact in ways which draw heavily on their biological counterparts for inspiration.

Key words: Homeostasis, wibble

1 Introduction

One of the most impressive abilities of many organisms is their ability to ensure a reasonably stable internal state despite wildly changing external environmental factors. This ability, often termed homeostasis, is a major feature of an organism's autonomy, highly prized by researchers of Computational Intelligence (CI) and robotics **?**.

The investigation of animal behaviour by biologists has taken many forms (cite behaviorists,control theorists), but the basic goal has been to understand the

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ways in which animals achieve useful behaviour which leads to on-going autonomy of the individual. Of these approaches, the one which most directly reflects the interest in on-going autonomy of a homeostatic nature is the dynamical systems approach. This considers the state of the individual in some state space which represents the state of the organism at any time. Homeostasis in such a state space is usually assumed to mean an orbit about some attractor which represents the "normal" condition for the organism. Clearly the presence of "attractive" values for particular variables will often lead to this type of cyclic path through state space, but should probably not be considered an immediate goal when constructing autonomous systems. This is for several reasons:

- The definition of such state spaces is fraught with problems such as: "What variables should be included?" and "How should behavioral attributes be represented?"
- The presence (or absence) of cyclical behaviour is often dependent on external factors such as the rising and setting of the sun. Thus, we need to define a set of circumstances under which a particular cyclic path will occur. Due to the unconstrained nature of the environment in which most workers wish their CI systems to operate, this is intractable.
- Once there are a significant number of interacting variables and control systems it is extremely hard to "design in" such cycles and to verify their presence and robustness. Thus, systems designed in such a way tend to be of limited complexity.

We thus propose that in the spirit of biologically inspired computing we take one more lesson from biological systems. This lesson is that the existence of complex homeostatic systems is due to a series of selective pressures and to self-regulating growth and development of each individual organism. Thus we wish to create a system which is capable of developing robust homeostasis in a self-organizing manner. To achieve this we must consider the mechanisms usually associated with maintenance of homeostasis. These are primarily the endocrine system, the immune system and the neural system. Thus we propose to consider the artificial counterparts of these three biological systems.

The use artificial neural networks (ANNs) as a means of mapping inputs to outputs is very common in CI [REFS]. For example, in a mobile robot the ANN might connect sensors and actuators [REFS]. Typically (after a learning period) the ANN defines a static, reactive response to a given input, but this is not the whole story in natural systems. In humans the endocrine system can affect the performance of the brain by means of various hormones, such as adrenaline Besendovsky and del Ray (1996). Hormonal signals are also used as controls for many other processes that help to achieve homeostasis, so their use would appear to have significant potential for their use in CI. We call such a system employing controlling hormones, an artificial endocrine system

(AES).

All animals have an immune system that is used to help keep them healthy. Artificial Immune Systems (AIS) have been researched for 15 years now and have been found to provide powerful and flexible tools for CI (de Castro and Timmis (2002a); Bradley and Tyrell (2002); Forrest et al. (1997)) to name a few. Initial work in AIS took inspiration from the basic functionality of the immune system (that being to be able to distinguish self cells from non-self cells) Forrest et al. (????). However, the view that the immune system operates in such a manner is being questioned and alternative views are proposed: two such views are the one of self assertion (Varela et al. (1988), Bersini (2002)) and that of the danger theory Matzinger (2002). With self assertion models, the system learns new senses of self over time, and will *assert* new cells to cover changes in the self. The danger model proposes that the immune system does not recognise self from non-self, but recognises danger from non-danger. Immunologists are activly debating this issue, as are practictioners in the field of AIS, of what implications self assertion models and danger models have for artificial immune systems (**?**Aickelin and Cayzer (2002)).

We propose a mechanism using a self assertion AIS approach, based on the immune network metaphor Neal (2002) which can be used in conjunction with an AES to control the self-organizing development of an ANN and AES by tracking the changes in self that occur during development of the system. Whilst this approach, for the time-being, ignores the problem of using the AIS for identification of infective agents and their distinction from novel components of self, it is not seen as an insoluble problem and other work is tackling this problem of tracking changes in self whilst continuing to identify infective agents Kim and Bentley (2002).

Thus the development of the target system is a self-organizing process which employs an artificial endocrine system and an artificial immune system to help shape and control an artificial neural network. The artificial neural network takes information from the perceptual channels and controls the state of any actuators that are present. The AIS and AES are essentially internal components, but in a similar way to its biological counterpart the AES can respond to changes in perceptual state.

This paper suggests a new method for combining ANN, AES and AIS in a single CI system, and develops a successful input-output model by means of positive and negative environmental cues. Work in Neal and Timmis (2003) details the first steps to realising the proposed system. It first describes the background to each of these areas, explains how they might be combined, and suggests the properties that such a system might have and its advantages over existing systems. As an example, a robotic implementation is set out in which a robot has to achieve homeostasis, in a changing environment, whilst

performing a task such as object avoidance, foraging, basking or exploration. An embodied agent application is more demanding than a simulated agent and thus provides a more exacting test which is less prone to over-specificity and provides a richer problem space for the agent to explore. Any plausible test of the system will require an embodied agent in a non-trivial environment performing several tasks, the successful completion of at least one of which is required for survival.

2 Biological Mechanisms for Homeostasis

Homeostasis is the ability of an organism to achieve a steady state of internal body function in a varying environment Besendovsky and del Ray (1996); Varela (1981); Vander et al. (1990). This is achieved via complex interactions between a number of processes and systems within organisms. This section explores the biology behind the concept of homeostasis. In order to understand this process it is necessary to examine three of the major systems within organisms; the nervous system, the endocrine system and the immune system. By examining these systems and their interactions, it is possible to understand how organisms can achieve this state. Work in this paper is concerned with attempting to create an artificial system that is capable of maintaining a state analogous to such homeostasis. Significant work has been done in extracting useful metaphors from the nervous system for the creation of artificial neural networks Haykin (1999). Work is now emerging in the field of artificial immune systems de Castro and Timmis (2002b), but little has been done on the creation on artificial endocrine systems. This section will discuss two of these computational intelligence approaches, namely ANN and AIS and postulate that through the unification of these approaches it may be possible to take the steps necessary to create an artificial homeostatic system. Work in de Castro and Timmis (2002b) describes some of these ideas, and the reader is directed to there for more detail on interactions of both the biological and artificial systems.

2.1 The Nervous System

The nervous system (NS) is central to an organism's ability to process and act upon stimuli that it receives from an external source. Organisms ranging from slugs to humans are endowed with a nervous system which ranges in size, ability and function. This system will then develop and improve over the lifetime of the organism, via processes such as learning and memory (although not exclusively these).

Organisms are constantly being exposed to a vast number of stimuli, to which it must react. Stated simply, the NS takes sensory input and generates effector output. The sensory parts of the NS take input from vision, taste etc., which are stimuli for effector elements such as muscles. The NS consists of two types of cells: neurons and neuroglia. Neurons are responsible for the firing of small electrical impulses in response to an input signal; neuroglia are cells which provide a type of support for neurons in the form of providing nutritional support, guiding development, the maintenance of the neuron environment and so on. Nearly half of the human nervous system is made from neurons, which are located in the brain. The basic components of a neuron can be considered as: (1) cell body and dendrites, (2) the axon and (3) axon terminals. Neurons will stimulate each other through the passing of an electrochemical signal from the axon of one neuron to the dendrite of another causing it to be stimulated. This in turn may cause the second neuron to send an activation (or possibly inhibitory) signal. This combined effect, may ultimately stimulate an effector.

2.2 The Endocrine System

Within an organism, chemicals known as hormones implement a regulatory mechanism acting directly at an individual cell level. This system, the endocrine system, is responsible for the production and storage of these chemicals Vander et al. (1990). Hormones are also produced by neurons and immune cells such as T-cells, but for the current purposes these mechanisms will be ignored. These hormones have a great deal of influence over a large number of bodily functions and are key actors in the maintenance of homeostasis. Hormones have many functions which affect behavior, assist growth, drive reproduction and so on. Typically, production of a hormone is in response to a change in state of the organism. Such changes are detected via the nervous system, immune system or by changes in other hormone or metabolite levels. Hormones are released into the blood or lymph system and are able to reach virtually all the tissues within the organism. It is quite possible (and normal) that there will be a number of different hormones present in the blood or lymph at any one time. However, not all cells will react to all hormones, as the response to hormones is highly specific: only certain cells are capable of responding to certain hormones. When a hormone locates its particular target cell, a binding takes place through specific receptors on the cells. Receptors on the target cell are usually located in one of two sites: within the cell nucleus (steroid hormone receptors) or in the plasma membrane (non-steroid hormone receptors, e.g., proteins, amines, and peptides). Non-steroid hormones decay and are ultimately removed from the organism at various rates. Built into the system is a mechanism by which hormones such as these will decay. This decay rate may well be a few minutes, but could potentially be a number of days.

When a hormone binds with a receptor on the cell membrane, it stimulates internal signals to the appropriate sites within the cell, which in turn alter the cell's activity.

2.3 The Immune System

The immune system is a remarkable, but complex, natural defence mechanism, which responds to foreign invaders called pathogens. Organisms typically have two lines of immunity, innate (inherited at birth) and adaptive (also known as acquired) which develops over the lifetime of the organism. However this is not the case for all organisms, such as the shark, which has a very powerful innate immune system and no acquired immune system. The innate immune system has first contact with any pathogenic substance and in a large amount of cases, this is all that is needed to remove the pathogenic material from the organism. However, there many times the innate immune system is insufficient and can not remove the infection. If this is the case, then the pathogen is passed over to the adaptive immune system.

The adaptive immune system primarily consists of B- and T-lymphocytes (cells). Through receptors on the cell, they are capable of binding with pathogenic material (antigens). Binding will occur between the receptors (paratopes) and antigen receptors (epitopes) if the affinity between the two is above a certain threshold. If a T-cell successfully binds an antigen this will cause the T-cell to stimulate B-cells through the emission of lymphokines. Additionally, B-cells can also bind with antigens, and therefore a notion of antigenic affinity is created. The B-cells receive stimulation from this interaction with the antigen. Through the combination of these two interactions (antigens and T-cells) a B-cell then becomes stimulated and reaches a threshold at which it transforms into a *blast cell*. These blast cells then produce large amounts of clones (in proportion to antigenic affinity: the higher the affinity, the larger the number of clones produced) and also a large number of free antibodies, which undergo somatic hypermutation (cite) to increase the diversity of the immune response. This whole process is known as affinity maturation and is part of the *clonal selection theory* (Burnet, 1959), which is the term used to identify the process described above. These antibodies (with the assistance of killer T-cells) will remove the antigen from the system. The immune system maintains a so called *immune memory* of cells, so that when exposed to the same (or slightly different) antigen, a quicker secondary response can be elicited, which results in quicker removal of the infection.

The immune system remembers encounters with antigenic material Tizzard (1988). There are a number of theories on how the immune system remembers encounters with antigenic material, with the most favoured view being that

one of clonal selection and memory cells Burnet (????). However, a theory first proposed in Jerne (1979) and suggested an idiotypic network and the immune network theory. Although not widely accepted, this theory is interesting especially for computer scientists, this will be explored in greater depth later. The idiotypic network was devised to explain the stimulation of B-cells in the absence of antigens. This is achieved by stimulation and suppression between cells via a network communicating via idiotypes on paratopes. The network acts as a self-organising and self-regulatory mechanism that captures antigenic information. Notable work in Farmer et al. (1986) further explored the immune network theory and created a simple model of the idiotypic network, which was further extended by Perelson (1989). It can be noted that such a self-regulated system is akin to a homeostatic system, i.e. is capable of maintaining its own internal steady state.

2.4 Interactions between Biological Systems

So far, attention has been given to three systems within an organism: the nervous system, endocrine system and immune system. These systems do not act independently but as one large and complex system.

First, immune, neural and endocrine cells can express receptors for each other. This allows for interaction and communication between cells and molecules all three ways. Secondly, it appears that products from immune and neural systems can exist in lymphoid, endocrine and neural tissue at the same time. This indicates that there is a bi-directional link between the nervous system and immune system. Third, it would seem that both endocrine and neural systems could affect the immune system. There is evidence to suggest that by stimulating areas of the brain it is possible to affect certain immune responses, and also that stress (which is regulated by the endocrine system) can suppress immune responses: this is also reciprocal in that immune cells can affect endocrine and neural systems. The action of various endocrine products on the neural system is accepted to be an important stimulus of a wide variety of behaviors. These range from behaviours such as flight and sexual activity to sleeping and eating.

With the immune system, the primary function is to defend the body against forign invaders and malfunctioning cells that may cause infection. There are a wide variety of components that are used to achoive this, ranging from the bone marrow to lymph nodes. The immune system displays a number of interactions with other biological systems, whcih can be summarised as follows. Different immune cell populations have receptor profiles for modulators such as neurotransmitters and endocrine hormones. Immune products are also known to coexist in neuroendocrine tissue for modulators such as the neuro-

transmitters and endocrine hormones. (de Castro and Timmis (2002b)). The nervous systems function is the reception of stimuli, with the transmission of nerve impulses and activation of muscle (or effector) mechanisms. The nervous system has a number of interactions, which can be summarised as follows. Neural cells express receptors for cytokines, hormones and neuro-transmitters. The brain can stimulate defense mechanisms against infection, thus engaging the immune system. The hypothalamus within the brain, controls the pituitary and other endocrine glands and it is known that neural products coexist in immune and endocrine tissues (de Castro and Timmis (2002b)). Finally, the endocrine systems function is to secrete hormones into the blood and other body fluids, with the aim being to regulate metabolism, growth etc. There are again, a number of components that make up this system, ranging from the pineal gland, thyroid to the thymus. Again, there are a number of interactions that the endocrine system is involved with, which can be summarised as follows. Endocrine cells express receptors for cytokines, hormones, and neuro-transmitters. Hormones provide feedback to the brain that affect neural processing, with the reproductive hormones affecting the development of the nervous system. Again, endocrine products also coexist in both immune and nervous tissue (de Castro and Timmis (2002a)). There are a number of examples of the interactions between the three systems. One example is that of the hypothalamus.

> *The hypothalamus is an excellent example of the interactions between these three major physiologic systems of the human body. Anatomically, the hypothalamus is part of the brain; it is located beneath the thalamus in the diencephalon. Signals from the limbic system are the primary neural trigger for the hypothalamus. Electrochemical signals from the hypothalamus trigger the auto-nomic nervous system as well as the pituitary. Nevertheless, the hypothalamus also produces a variety of hormones that are conveyed through a group of blood vessels to the pituitary, triggering the release or inhibition of the corresponding pituitary hormones. Furthermore the hypothalamus is an integral part of a series of feedback loops which not only regulate many systemic physiologic processes, but also adjust those processes to deal with environmental or internal changes and/or threats to the organism. As part of this feedback system, the hypothalamus senses the amount of certain hormones in the blood, the amount of neural stimulation in the limbic system, and the amount of certain thymic hormones. This information is then processed by the hypothalamus and adjustments in both neural and hormonal secretions are accomplished. The adjustment can be either to restore homeostasis or to move in either direction from it, depending upon the result of the combined information processed by the hypothalamus.* de Castro and Timmis (2002b).

	ANN	**AES**	**AIS**
(1)	Neuron	Endocrine gland	Lymphocyte
(2)	Network topology	Hormone interactions	Affinity measures
(3)	Learning algorithms	Hormone structure update	Immune algorithms

Table 1
ANN, AES and AIS in a simple framework, see section 3 for (1), (2) and (3)

3 A Framework for Artificial Homeostasis

This section proposes a potential *framework* for creating artificial homeostasis. The concept of a framework for CI is not new, and indeed work in de Castro and Timmis (2002b) argued that a framework would consist of (1) a representation of the components of the system (2) mechanisms by which to evaluate interactions of these components and (3) procedures for adaptation. Under such a conceptualisation, it is easier to discuss how such systems may be combined to form a more complex system. Table 1 captures the salient features of this argument with the addition of AES and will be expanded on in the following sections. It is proposed that combinations of these components, will be useful in the construction of systems capable of artificial homeostasis.

3.1 Artificial Counterparts of the Biological Systems

Significant work has been done in extracting useful metaphors from the nervous system for the creation of artificial neural networks Haykin (1999). Work is now emerging in the field of artificial immune systems de Castro and Timmis (2002b), but little has been done on artificial endocrine systems (AES). This section will discuss ANN and AIS and postulate that through the combination of these approaches and an AES it may be possible to create an artificially homeostatic system. Work in de Castro and Timmis (2002b) describes some of these ideas, and the reader is directed to there for further detail on interactions of both the biological and artificial systems.

3.1.1 Neural Networks

A substantial body of research has been undertaken in extracting useful metaphors from the neural systems. Artificial Neural Networks (ANN) are parallel distributed processing systems that are constructed via the connection of simple processing known as artificial neurons Haykin (1999). ANN have been applied to a vast array of problem areas such as machine vision Sandini et al. (1989)and robot control Oyama et al. (2001). Figure 1 is a graphical depiction of a simple artificial neuron. In order to be of any practical use, individual neurons are connected together to form artificial neural networks. These networks are *trained* in order to be able to classify input patterns (x) through the constant adjusting of the weights (w_i) until the ANN can recognise the pattern. The weights are adjusted via a number of possible *learning algorithms* e.g. backpropogation. An artificial neuron can be represented mathematically as shown in Equation 1. Once the summing of the inputs has taken place, the neuron will fire, depending on the activation function $f(u)$, in this case of work in this paper a standard sigmoidal activation function has been employed, as shown in equation 2.

Fig. 1. A simple artificial neuron

$$u = \sum_{i=0}^{nx} w_i \cdot x_i \tag{1}$$

$$f(u) = \frac{1}{1 + e^{-u}} \tag{2}$$

Work in this paper proposes to augment this basic artificial neuron, with interactions from an artificial endocrine system. For the purposes of this work, the weights within the ANN are constant, although for future work, this will not be the case.

3.1.2 Artificial Endocrine Systems

This paper proposes a new biologically inspired technique known as an Artificial Endocrine System (AES). The role of the AES is to provide a long term regulatory control mechanism for the behaviour of the system. The AES proposed consists of *gland cells* which secrete *hormones* in response to external stimuli, to the value r_g for one gland g. This is shown in equation 3 where α_g is the rate at which hormones are released for a particular gland g.

$$r_g = \alpha_g \sum_{1=0}^{nx} x_i \tag{3}$$

The level of hormone is subject to geometric decay, as shown in equation 4 where $c(t)_g$ is the hormone concentration at a time t for a gland g and β is the decay constant.

$$c(t+1)_g = c(t)_g \cdot \beta \tag{4}$$

Membrane receptors located on artificial neurons are sensitive to hormones, thus providing a mechanism for the regulation of the ANN by the AES. Gland cells secrete and record the concentration of hormones present in the system. Each gland cell secretes a specific hormone, represented by a simple string of bits. Within the integrated AES-ANN the hormone sensitive membranes of neurons simply have a list of hormone receptors (again, represented as bit patterns) to which hormones are matched and a neuron-specific action associated with each receptor. At present, perfect matches of hormone to receptor are considered (though this is not necessarily required: imperfect matches should generate lesser reactions). In the natural endocrine system, hormones are transported throughout the body: the same effect is achieved in the artificial endocrine system through the matching of each hormone secreted to the receptors on each cell's membrane in turn. A record of the current concentration of a hormone is maintained in the gland cell which secretes the hormone, and is then used to moderate the strength of reaction.

True to the analogy with the biological endocrine system, different cells types react to particular hormones, in different ways. The actions which are triggered in individual cells can vary according to four factors: the hormone which is detected, its concentration, the type of receiving cell and the individual cell's make-up. The former two of these factors are explained above, but the latter require further explanation. The type of cell receiving the hormone signal will clearly dictate what actions it is capable of performing. For example, a neural cell may lower (or raise) its threshold value or increase (or decrease) its sensitivity to one or many of its inputs; and a gland cell may increase (or decrease) secretion rate of a hormone. The precise make-up of cells is fixed when they are added to the system. This may include variations in membrane characteristics (abilities to receive hormone signals), the effects that those signals have within the cell and other cell-type-specific characteristics such as connectivity pattern of a neuron etc.

In order to allow for the AES-ANN interactions, the hormone levels have to be able to affect the input weights in the ANN. Figure 2 provides a simple graphical representation of how this is achieved. Here the recorded hormone level affects each input weigh on a particular neuron. It is easier to see this when these interactions are described mathematically, as in equation 5, where in this case x_i and w_i are the same as equation 1 and ng is the number of glands in the system, C is the concentration of hormone, S is the sensitivity of the connection for receptor i to hormone j and M is the match between the

receptor i and hormone j and is defined in equation 6, where dis is a distance measure function.

Fig. 2. The affect of endocrine interaction on the artificial neuron

$$u = \sum_{i=0}^{nx} w_i \cdot x_i \cdot \prod_{j=0}^{ng} C_j \cdot S_{ij} \cdot M_{ij} \tag{5}$$

$$M = \frac{1}{1 + dis(i,j)} \tag{6}$$

It is now possible to compare equation 1 with equation 5. It should be noted that the new equation for the AES-ANN interaction is simple augmentation of the original equation, with the iterative application of hormone levels applied to each input weight in the neuron. It should also be noted that this new AES Neuron bares a passing resemblance to the *Sigma Pi Neurons* McClelland (1986), it is fundamentally different upon further examination.

3.1.3 Artificial Immune Systems

AIS is very much an emerging area of biologically inspired computation. This insight into the immune system has led to an ever increasing body of research in a wide variety of domains such as machine learning Timmis and Neal (2001), immunised fault tolerance Bradley and Tyrell (2002) and computer security Forrest et al. (1997) to name a few. Recently, an attempt has been made to bring together what at times seemed a disparate area of research, in a general AIS framework which describes basic AIS components, interactions and algorithms de Castro and Timmis (2002b). Here the authors argued that AIS could be seen as a novel soft computing paradigm that has great potential to be hybridised with a variety of other soft computing approaches and computational intelligence paradigms. Here the authors proposed that a number of already established AIS algorithms can be used as basic algorithms outside of their originally intended domain. Four basic immune process were incorporated into a proposed framework, those being bone marrow models, thymus models, clonal selection models and immune network models.

True to the analogy with the immune system, we propose to utilise the immune system metaphor in two ways. The first way is for the AIS to act (in part) as a growth regulator for cells within the artificial system. Within our system, cells correspond to neurons, endocrine glands and connections between these. As it is proposed that this system will develop over time, some mechanism for controlling this growth is required. The role of AIS will be to remove cells and or connections that have a detrimental impact on the functioning of the system. The other role of the AIS will be to act in the more traditional manner

of responding to pathogenic agents (which could be environmental changes, inputs from sensors, malfunctioning parts of the robot) and affect the ANN and AES accordingly. It is expected that the role of the AIS within the target system will be implemented via an immune network algorithm.

4 Proposed Mechanisms for Artificial Homeostasis

The control mechanism proposed unites in a single framework the techniques presented earlier. A system that allows the low-level interaction of (computational) neural, immune and endocrine systems provides much scope for the construction and self-organizing development of highly functional computational intelligences. The structure proposes to follow to some degree the biological realities of cell proliferation, differentiation and apoptosis. Cells that are added to the system are differentiated on a very restrictive epigenetic landscape [REF developmental biology]. Components added will become one of: neurons, synapses, B cells, hormone producers or connections between hormones. These types have been chosen as they are usually viewed as being fundamental to the control of behaviour and maintenance of homeostasis.

4.1 The Grand View

The various components of the system will be implemented using relatively standard machine learning components for the neural and immune systems. This is not possible for the endocrine system as there is not an established literature concerning the implementation of artificial endocrine systems.

The functions performed by the natural endocrine system are both diverse and pervasive throughout the organism. The release of hormones into the bodily fluids and blood-stream permits their rapid transport throughout the body. An artificial endocrine system (AES) must be capable of such global activity. The natural endocrine system plays a pivotal role in initiation and regulation of a huge variety of homeostatic functions. The artificial system must be capable of such interactions. The innate endocrine components outlined in section 4.2.2 (control of growth, epigenesis and apoptosis) are only examples of what is a much more general mechanism. The growth of the artificial endocrine system is an on-going process which allows for the development (via cell reproduction and mutation) of new hormones and new hormone release mechanisms. The hormones released by the AES will be globally applied to the cells in the system which react dependent upon their type and sensitivity. Novel reactions to hormones may be generated by the component cells of the different parts of the system (also via cell reproduction and mutation). The

sensitivity to hormones of all cells in the system will be defined by a membrane definition associated with each cell. Membranes provide various binding sites for hormones generated and are capable of inhibition and excitation of the cell in question. Binding sites on the membranes are defined as bit strings which are related to hormones in circulation by checking the hamming distance between the binding site pattern and the hormone molecule pattern. Concentrations of hormones are increased by endocrine cells which release their particular hormone molecule into the system. The concentrations are decreased by the continuous breakdown of the molecules in circulation. This breakdown rate varies between hormones. The release of a particular hormone molecule by a particular gland cell may be triggered or suppressed by several mechanisms. These are as follows: (1) A particular pattern of activity on a perceptual channel (or channels); (2) A particular pattern of activity in the artificial neural system; (3) A particular pattern of activity in the artificial immune system, and (4) The presence of a hormone (or hormones) at a particular concentration. Thus a gland cell must be allowed to monitor any of several state indicators internal to the system. In order to achieve this it must be permitted to "connect" in various ways. These must include: (1) Connections directly to perceptual channels; (2) Connections to the outputs of artificial neurons, and (3) Connections to monitor the stimulation level of artificial immune system components. In addition to these connections, gland cells must have a membrane definition as described above. The combination of these sources of stimulation provides a realistic complexity of stimuli for the gland cells and provides scope for the evolution of useful and interesting functionality in the AES.

A large degree of commonality exists between the various cell types of the system. The functionality of the various cell types can be summarised as follows. The addition of cells to the neural system is essentially stochastic in nature and occurs at a rate determined by a combination of the growth hormone and epigenetic hormone concentrations (section 4.2.1). At any given time the state of the neural system (NS) will be monitored by the AIS (section 4.2.3), and thus there will be knowledge of "self" encoded which represents the current NS state. Clearly when a neuron is added to the NS this state will alter and the AIS will identify the new neuron as "non-self". This will result in the AIS beginning the process of killing the cell at a particular rate. The rate of this process is however controlled (as are most other processes in the system) by the concentrations of various hormone levels. Of these various hormones, the fitness hormone (the generation of which is inhibited and stimulated by negative and positive reinforcement stimuli respectively) is very important. If the fitness hormone is being secreted in large quantities then the time taken for the new cell to elicit a full reaction from the AIS is drastically increased. This allows sufficient time for the AIS to extend its definition of self to include the new neuron. As the processes of cloning and mutation are continuously ongoing in the AIS, this may occur and prevent the culling of the cell without

Cell Type	Hormone Sensitive Membrane	Connectivity			Comments
		Perceptual Channels	Neural Outputs	Actuators	
Endocrine	Yes	Input	Input	None	Produce hormones to regulate internal processes
Neural	Yes	Input	Input	Output	Read perceptual channels and activate actuators
Immune	Yes	None	None	None	Monitor internal state and cull cells that inhibit performance

Table 2
Summary of Cell Type Functionality

any further intervention from other mechanisms. If however the fitness hormone concentration is low then the process will proceed apace, and the cell will be destroyed quite rapidly. Thus if a neuron is added to the NS which increases the fitness it is likely to survive due to its influence in increasing the fitness hormone concentration. Neurons which decrease the fitness will have the opposite effect and are thus more likely to be culled rapidly.

The continuous addition and deletion of neurons requires that the neural structures which are generated are capable of supporting incremental growth and culling in a suitable fashion. There are various ways to achieve this but we propose the use of the encoding techniques presented in **?**tanley and Miikkulainen. This will allow for the efficient storage transfer and alteration of the topologies produced without generating unusable neural systems.

The system as a whole thus has the ability to add cells to each of its three internal components in ways suitable to each. The three components have the ability to interact in complex networks and to develop feedback control in an ongoing way under pressure of positive and negative reinforcement. Over time the system adds cells to its three components and develops complex control strategies in a self-organizing fashion. The overall system is expected to display stability of external and internal behaviour that can be described as emergent homeostasis. Table 2 summarises the basic functionality of the three main cells contained within the system.

There are four innate components at the beginning of development of the system. These are engineered to ensure a suitable starting point for development to begin. There are three sets of cells that constitute embryonic starting points for the development of the three components described above, namely: the neural system, the immune system and the endocrine system. In addition to these there is the external boundary of the system (skin?) which provides access to sensory apparatus and actuators.

4.2.1 The Innate Neural System and its Development

At the outset, a minimal neural network is constructed linking some of the perceptual units and action generators which are available to the system. This minimal system may be of arbitrary design, but it is to be expected that the initial network will be hard-wired to generate some reflexive innate behaviour. The neural system will fulfil the same role as that played by the natural neural system in that it will connect to both the sensory apparatus and the actuators (effectors) of the system. This will allow reactive behaviour to be elicited. Cells which differentiate to become neurons will be inserted into the network using an intelligent insertion mechanism (**?**hat stuff) and will be evaluated with respect to the overall usefulness of the system behaviour. If there is a sustained drop in the usefulness of the overall system behaviour after the addition of a particular neuron to the network then that node may be culled. This culling process will be triggered by one of the modes of operation of the AIS that is built into the system. Neurons that exhibit other dangerous behaviour, such as firing continuously or for a very large proportion of the time, may also be culled by the AIS. Clearly neural cells may monitor perceptual channels and stimulate external activity directly. This neural activity may be moderated by hormones released by the artificial endocrine system, but only in a relatively homogeneous fashion. That is to say that functionally identical neurons will be affected identically by hormones at any one time. The neurons will be simple summation and threshold units as are typically used in many ANN systems.

4.2.2 The Innate Artificial Endocrine System

The initial population of gland cells will be quite small and consist of cells that secrete a few hormones that are key to the development and control of the initial cell population in the system. Three fundamental factors are required. These are growth hormone, fitness hormone and an epigenetic control hormone. The growth hormone will be secreted by a gland cell which monitors the size of the current population of cells in the system and via a negative

feedback mechanism (more cells implies less hormone release) achieve a steady state size of cell population. The fitness hormone will be secreted each time an improvement in performance is detected in order to suppress apoptosis of any new cells which have recently been added to the system. This should encourage retention of novel cells which improve performance. The epigenetic control hormone will be released by a cell which monitors the relative sizes of the populations in each of the parts of the system, and via a negative feedback system maintain a dynamic equilibrium between the components. Clearly these hormones must act directly on reproducing cells and affect the cells which they produce. Cells in the artificial endocrine system can monitor the internal state of the system as well as the perceptual channels to regulate the release of their individual hormones. The hormones can only indirectly (by affecting the AIS and neural system) affect the external behaviour of the system.

4.2.3 The Innate Artificial Immune System

The initial immune system will be based on an idiotypic network model, and will contain a small number of components which recognise the cells that are present within the innate parts of the other components of the system (the endocrine cells and the neural cells) as self. Thus any new cells generated that vary significantly in their properties from the initial sets may be recognises as non-self and as such will possibly elicit a destructive response from the AIS. In order to select those cells that are possibly of benefit to the overall performance of the system, when an improvement in performance is detected a suppression of the immune system's destructive power will be required accompanied by an increase in its ability to expand its definition of self, via the mechanisms of self assertion inherent within the immune network model. This suppression will be elicited by the release of a hormone from a cell in the innate artificial endocrine system which is sensitive to improvements in performance. In general, cells in the AIS will be sensitive to hormone concentration and will be capable of being suppressed or stimulated by them. The cells in the AIS will only be capable of detecting and destroying cells within the system that are persistently classified as non-self or dangerous. This labelling is however continuously modified as the idiotypic network of the AIS evolves. The cells in the AIS will not directly affect the external behaviour of the system via its actuators, or monitor directly the perceptual channels.

4.3 External Boundary

The external boundary of the system provides an interface to the world that receives sensory input via perceptual channels which in the first instance will

be considered to simply be streams of real-valued numbers. These perceptual channels can only be monitored by connections to cells in the neural system and endocrine system. In addition to the perceptual channels the boundary provides actuators which can be activated with real-valued numbers by neural outputs only.

5 Conclusions

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References

Aickelin, U., Cayzer, S., 2002. The Danger Theory and its Application to Artificial Immune Systems. In: Timmis, J., Bentley, P. (Eds.), Proceedings of the First International Conference on Artificial Immune SYstems (ICARIS). pp. 141–148.

Bersini, H., 2002. Self Assertion versus Self Recognition: A Tribute to Francisco Varela. In: Timmis, J., Bentley, P. (Eds.), Proceedings of the First International Conference on Artificial Immune SYstems (ICARIS). pp. 107–112.

Besendovsky, H. O., del Ray, A., 1996. Immune-Neuro-Endocrine Interactions: Facts and Hypotheses. Nature 249, 356–358.

Bradley, D., Tyrell, A. M., 2002. A Hardware Immune System for Benchmark State Machine Error Detection. In: Congress on Evolutionary Computation. PArt of the World Congress on Computational Intelligence. IEEE, Honolulu, HI., pp. 813–818.

Burnet, F., ???? The Clonal Selection Theory of Acquired Immunity. Cambridge University Press.

de Castro, L. N., Timmis, J., 2002a. An Artificial Immune Network for Multi Modal Optimisation. In: Proceedings of the World Congress on Computational Intelligence (WCCI). Honolulu, HI., pp. 699–704.

de Castro, L. N., Timmis, J., 2002b. Artificial Immune Systems: A New Computational Intelligence Approach. Springer-Verlag.

Farmer, J. D., Packard, N. H., Perelson, A. S., 1986. The Immune System, Adaptation, and Machine Learning. Physica D (22), 187–204.

Forrest, S., Hofmeyr, S., Somayaji, A., 1997. Computer Immunology. Communications of the ACM 40 (10), 88–96.

Forrest, S., Perelson, A., Allen, L., Cherukuri, R., ???? Self-Nonself Discrimination in a Computer, pages =.

Haykin, S., 1999. Neural Networks - A Comprehensive Foundation. Prentice-Hall.

Jerne, N. K., 1979. Towards a Network Theory of the Immune System. Ann. Immunol. (Inst. Pasteur) C (125), 373–389.

Kim, J., Bentley, P., 2002. Immune Memory in the Dynamic Clonal Selection Algorithm. In: Timmis, J., Bentley, P. (Eds.), Proceedings of the First International Conference on Artificial Immune SYstems (ICARIS). pp. 59–67.

Matzinger, P., 2002. The Danger Model: A Renewed Sense of Self. Science 296.

McClelland, D. E. R. J., 1986. Parallel Distributed Processing: Explorations in the Microstructure of Cognition. MIT Press, Cambridge, Massachussets.

Neal, M., 2002. An Artificial Immune System for Continuous Analysis of Time-Varying Data. In: Timmis, J., Bentley, P. (Eds.), Proceedings of the First International Conference on Artificial Immune SYstems (ICARIS). UKC, Canterbury, UK, pp. 76–85.

Neal, M., Timmis, J., 2003. A Neuro-Endocrine Controller Robotic Controller: Towards Artificial Homeostasis. In: Review.

Oyama, E., Chong, N. Y., Agah, A., Maeda, T., Tachi, S., 2001. Inverse Kinematics Learning by Modular Architecture Neural Networks with Performance Prediction Networks. In: IEEE International Conference on Robotics and Automation. pp. 1006 –1012.

Perelson, A. S., 1989. Immune Network Theory. Imm. Rev. (110), 5–36.

Sandini, G., Bosero, F., Bottino, F., Ceccherini, C., 1989. The Use of Anthropomorphic Visual Sensor for Motion Estimation and Object Tracking. Proc. OSA Topical Meeting on Image Understanding and Machine Vision. IEEE Transactions on Neural Networks 1 (1), 28–43.

Timmis, J., Neal, M., 2001. A Resource Limited Artificial Immune System. Knowledge Based Systems 14 (3/4), 121–130.

Tizzard, I., 1988. Immunology : An Introduction, 2nd Edition. Saunders College Publishing, Ch. The Response of B Cells to Antigen, pp. 199–223.

Vander, A. J., Sherman, J., Luciano, D., 1990. Human Physiology: The Mechanisms of Body Function, 5th Edition. McGraw-Hill.

Varela, F., Countinho, A., Dupire, B., Vaz, N., 1988. Cognative Networks: Immune, Neural and Otherwise. Theoretical Immunology 2, 359–375.

Varela, F. J., 1981. Self Organising Systems. New York Campus Press, Ch. Autonomy and Autopoiesis, pp. 14–23.