

Homeostatic adaptation to inversion of the visual field and other sensorimotor disruptions

Ezequiel A. Di Paolo

German National Research Center for Information Technology GMD

Schloss Birlinghoven,

Sankt Augustin, D-53754, Germany

Ezequiel.Di-Paolo@gmd.de

Abstract

Adaptation to inversion of the visual field is studied in a simple simulated model of phototactic behaviour. Inspired by recent findings in neuroscience, a novel neural architecture based on continuous dynamical neural networks is implemented. Individual cells behave homeostatically by facilitating local plasticity whenever their activity goes out of bounds. Robots are evolved to perform long-term phototaxis on a series of light sources while trying to keep neurons behaving homeostatically. Robots are then tested under the condition of left/right inversion of vision. Initially, their phototactic capability is lost, which in most cases causes neurons to lose homeostasis and trigger plastic changes. After long periods of maladaptation, robots adapt to the new sensorimotor situation, and phototactic behaviour is recovered. The introduction of other disruptions such as radical perturbations to motor and sensor gains also results in eventual adaptation. The model intends to bring Ashbyan ideas on the relation between adaptation and internal stability into the context of current research on evolutionary robotics. In spite of the promising preliminary results, a series of unsolved questions are raised by this model. Possible solutions are suggested.

1. Introduction

On putting on a pair of goggles that invert the visual field in the left/right direction, or in the up/down direction, or both, a normal human subject will act clumsily, stumbling upon furniture, walking straight into walls and reaching for non-existent objects in midair. However, as a result of prolonged periods of continuously wearing the distorting goggles, the frequency of errors diminishes, collisions are avoided, and the subject regains normal locomotion. After a few weeks, activities such as riding a bicycle, driving a car, skiing, and even fencing become possible. Subjects at this stage report that their visual

experience is no longer upsetting, and even that (most) things seem to them to be ‘the right way up’.

Adaptation to radical sensorimotor distortions constitutes a whole field of neuro-psychological investigation both in human and non-human animals. Studies go back to preliminary experiments in the 19th century by Helmholtz using prismatic lenses, passing through Stratton’s classic experiments (1896, 1897), to more modern versions (Welch, 1974, Spillman and Wooten, 1984). This method of research reached a peak of creativity in the 1940s and 1950s with the experiments of the Innsbruck school (Kohler, 1964)¹. Eventual adaptation in these cases is difficult to explain purely in terms of specific evolutionary pressures (which may safely be considered as nil for the case of visual inversion in humans), and so it probably arises as a consequence of more general principles of organismic operation.

In a series of extensions to the Innsbruck experiments, J. G. Taylor (1962) tried to bring his results within a theoretical framework largely based on W. Ross Ashby’s cybernetic views of adaptation as resulting from the ultrastable nature of organisms (Ashby, 1960). According to these ideas, adaptation to wearing distorting glasses comes about by the action of different internal systems of the organism which are challenged in their stability by the sensorimotor distortion. By seeking new forms of stability as a result of plastic changes, these systems are able to bring about new overall patterns of sensorimotor coordination that are themselves stable.

Ashby’s key idea that adaptation implies some sort of internal homeostasis of “essential variables” still lurks in the background of much work on adaptive behaviour, but has been disappointingly little explored directly in research contexts such as evolutionary robotics². Similarly, Taylor’s attempts to introduce Ashby’s theory into

¹Adaptation to visual distortion has also been observed in chicks, rats, the barn owl, and monkeys (Welch, 1974). Other forms of radical adaptation in nature include adaptation to loss of a limb or a sensor, see Goldstein (1995/1934) for a classical account.

²‘Homeostasis’ is here taken to mean activity which is bounded to stricter limits than those defined by extreme physiological conditions, rather than the narrower definition of activity that approaches a set-point.

a research program on adaptation to rearranged vision have met, despite strong experimental results, only with a lukewarm response (although this may be partly due to some of Taylor’s own interpretations).

To help reverse this situation in both these contexts, this paper will explore the usefulness of Ashby’s views in a model of adaptation to visual inversion in simple robots. It does not pretend to settle the question of how adaptation to perceptual distortions occurs in human beings and other animals. It should be construed as a preliminary and minimalistic model, which, nevertheless will be able to open a range of interesting questions. The model is built in the hope that it can illuminate, in a more or less abstract way, how radical adaptation occurs in natural cases. It may also have repercussions for robot design.

The following section discusses the connection between homeostatic internal mechanisms and adaptation in the context of Ashby’s theory. Section 3 briefly reviews recent neurobiological data and hypotheses regarding homeostasis in the dynamics of synaptic change, and in single cell activity. Section 4 describes a novel architecture for neural networks in which plasticity is triggered locally whenever neuron activity is out of bounds. This architecture is implemented on Khepera-like robots in a simulated phototaxis scenario using a genetic algorithm to find adequate parameters. Section 5 describes experiments on adaptation to inverted vision. Adaptation occurs despite homeostatic plasticity not being selected to act favourably under perturbed conditions. Finally, section 6 examines the consequences of this result and discusses some of the questions that remain open and possible ways of answering them.

2. Homeostatic adaptation

According to Ashby, a form of behaviour is adaptive if it conserves the “essential variables” of the organism within physiological limits (Ashby, 1960, p. 52). By essential variables, he refers to those physiological variables that must be kept within limits for the organism to survive. By implication, this means that Ashby’s definition of adaptive behaviour is a special case of other definitions conflating *adaptation* and *viability* (e.g., Maturana and Varela, 1980, pp. xx-xxi, 105). Such definitions state that an organism remains adapted if its relation to its environment is such that the conservation of its identity and its condition of being alive are not interrupted. There are some problems with this view which will not be discussed here. In particular, it allows no room for the idea of any *maladaptation* other than death itself. Ashby’s definition would seem to suffer from the same problem were it not for the fact that he is implicitly using a softer condition. Even though he says that essential variables must be kept within bounds for survival, in his implementation of an actual adaptive

device (the Homeostat), as well as in many examples throughout his (1960) book, homeostatic variables are not really ‘essential’ but are rather *tokens* or *proxies* for essential conditions. In some cases they are not even physiological variables at all (e.g., pp. 220-221).

Where does the link between internal stability and an adapted relation to the environment come from? It is certainly not a logical link, since it is possible to think of internally stable systems which do not exhibit stable behaviour, but are instead ‘driven’ by environmental changes possibly to their own destruction without losing their stability in the meantime (e.g., a rock in the extreme case). The connection is, in fact, hypothetical. If habitual patterns of agent/environment interaction are challenged, and they are observed to ‘recover’, then this must be because some sort of ‘ecological’ invariant is being maintained, and, if this is the case, it could well be because the ecological invariant is a reflection of internal physiological stability. In such cases, whenever internal stability is lost, adaptivity is also lost.

Ashby proposes a generalized architecture for an ultrastable system, i.e., a system that will tend to change its own configuration plastically whenever stability is lost, until it finds a new internal dynamics which will make the system stable under the new conditions. Plastic changes are triggered by internal variables which are caused to go beyond allowed limits by the new agent/environment relation. In other words, such variables will tend to behave homeostatically, and drive the whole system towards an adapted state in a non-teleological manner in cases in which this is possible.

It is important to emphasize the fact that the connection between internal ultrastability and adaptation is hypothetical, and will account at best for a sub-set of all possible instances of adaptation. It could well be that adaptation, interpreted as the recovery of an ecological invariant, occurs due to plastic changes ‘outside’ the organism, for instances in other organisms or in the physical environment itself, without the adapted organism having changed internally. These cases would require different explanations.

3. Brain plasticity and homeostasis

Activity-dependent, long-lasting modification to the number and strength of synapses in the nervous system has long been considered the basic mechanism by which neuronal circuits are refined by experience. How do individual nerve cells in particular, and the nervous system in general, manage to retain their basic properties in a more or less stable condition in the face of processes that are constantly inducing change? Only in recent years have neurobiologists begun to focus on how neurons maintain their electro-chemical and functional properties by means of regulatory processes leading to such stability, (Abbott and Marder, 1995, Abbott et

al., 1997, Davis and Goodman, 1998, Desai et al., 1999, Frégnac, 1998, Galarreta and Hestrin, 1998, LeMasson et al., 1993, Spitzer, 1999, Turrigiano et al., 1998, Turrigiano, 1999).

Regulatory processes that depend on neuronal activity have been found and postulated at different levels. For instance, Frégnac (1998) mentions processes that regulate total synaptic strength over long timescales so that a mean synaptic efficacy is maintained, or allowed to change only between certain bounds, by means of input-dependent regulation (Markram and Tsodyks, 1996, Abbott et al., 1997). In relation to this, Galarreta and Hestrin (1998) have shown that synaptic depression after prolonged presynaptic firing is much stronger at excitatory synapses than at inhibitory ones, thus inducing stability in the activity of cortical neurons.

A different class of homeostatic mechanisms regulate the structural and functional properties of individual cells. For instance, relatively constant firing properties are maintained by neurons over the long term in spite of variations in shape and size, protein turnover, loss and gain of synapses, etc. Theoretical and experimental work suggests that this is achieved by regulating the balance of ionic conductances (LeMasson et al., 1993, Abbott and Marder, 1995) with the result that average firing rates are not allowed to be too high or too low.

Recently a different form of synaptic plasticity which achieves a similar end has been described (Turrigiano et al., 1998, Turrigiano, 1999). Firing rates in cortical pyramidal neurons are kept within functional boundaries by a slow multiplicative scaling of all synaptic strengths. Synapses are scaled up when firing rate is low and down when firing rate is high. Interestingly, this mechanism conserves relative differences between individual inputs and may lead to competition between and elimination of synapses (Turrigiano, 1999).

These mechanisms are functionally reminiscent of the BCM (Bienenstock et al., 1982) model of activity-dependent ('sliding') thresholds for Long Term Potentiation (LTP) which has been proposed for the visual cortex. According to the model, potentiation occurs only when postsynaptic activity is higher than a threshold value, which itself depends on the history of postsynaptic activity. The threshold slides towards higher values when the history of activity has been high on average, thus making further synaptic potentiation more difficult, and towards lower values when activity has been low, thus facilitating postsynaptic potentiation and, as a consequence, a likely increase in activity.

So far, the functional value of these and other mechanisms has mainly been the subject of speculation. Stemmler and Koch (1999) have recently proposed that cells may adjust their firing properties to the statistics of the input patterns, so that the information encoded in

the firing rate is maximized (roughly with each rate value corresponding to a distinct pattern). The model implies homeostasis in the firing rate, but the above homeostatic mechanisms do not necessarily imply this interpretation. Also it has been proposed that the modification of intrinsic structural properties (such as conductances) of neurons during homeostasis may also function as a form of memory mechanism (Marder et al., 1996) by driving neurons into different stable firing regimes.

4. Methods

Drawing inspiration from the above mechanisms, we propose to study homeostatic adaptation using a simple model of phototaxis aimed at testing whether internal changes driven by loss of homeostasis are able to induce the recovery of certain behavioural functions (in this case phototaxis) under radical sensorimotor disruptions (in this case visual inversion).

A population of simulated robots is evolved to perform normal phototaxis (without inversion of the visual field) on a series of light sources by presenting only one source at a time for a limited, but long period. Plastic neural networks are used to control the robots. Plasticity is triggered locally on the incoming synapses to a neuron if its activity becomes too high or too low. The rules of plastic change are encoded genetically, and need not lead to homeostasis in general. Rather, by selecting for long term stability of the phototactic behaviour, the stability of weight change is implicitly selected. Due to the computational costs involved in evaluating long term stability using hundreds of sources, homeostasis in the activity of each cell is slightly favoured during evolution. It is possible in this way to achieve long term stable behaviour using about 6 sources instead of hundreds. Robots evolved for this task are then tested for phototaxis after an inversion of their visual field (achieved by exchanging the positions of the sensors left and right), and other disruptions such as changing the sensor and motor gains for which they have not been evolved.

4.1 Robots

Robots are modelled as solid circular bodies of radius 4 with two diametrically opposed motors and two light sensors. The angle between sensors is 120 degrees, but there is an indeterminacy in the position of sensors with respect to the motors between evaluations. The mean angle between a sensor and the body midline is 60 degrees (Fig. 1) with a uniform variation of ± 5 degrees.

Motors can drive the robot backwards and forwards in a 2-D unlimited arena. Robots have a very small mass, so that the motor output is the tangential velocity at the point of the body where the motor is located. The translational movement of the whole robot is calculated using the velocity of its center of mass (the vectorial average

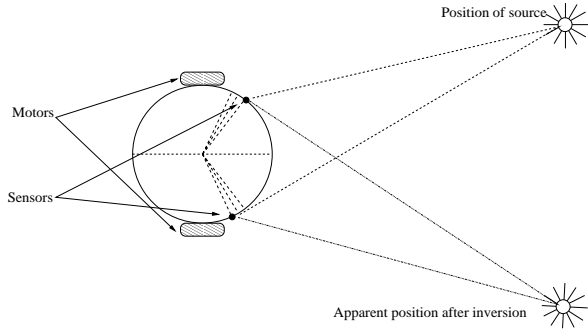


Figure 1: Position of sensors, motors, and apparent change in the position of source after sensory inversion.

of the motor velocities), and the rotational movement by calculating the angular speed (the difference of the tangential velocities divided by the body diameter). There is no inertial resistance to either form of movement.

Light from point sources impinges on sensors with a local intensity proportional to the source intensity and the inverse square of the distance from sensor to source. The model includes shadows on sensors produced when light is occluded by the body.

4.2 Plastic controller

A fully connected, 8-neuron, dynamic neural network is used as the robot's controller. All neurons are governed by:

$$\tau_i \dot{y}_i = -y_i + \sum_j w_{ji} z_j + I_i; \quad z_j = \frac{1}{1 + \exp[-(y_j + b_j)]}$$

where, using terms derived from an analogy with real neurons, y_i represents the cell potential, τ_i the decay constant (range [0.4,4]), b_i the bias (range [-3,3]), z_i the firing rate, w_{ij} the strength of synaptic connection from node i to node j (range [-8,8]), and I_i the degree of sensory perturbation on sensory nodes (modelled here as an incoming current) which is always 0 for the other neurons. There is one sensory neuron for each sensor and one effector neuron for controlling the activity of each motor.

The transduction from effector neuron activity to motor output is achieved by mapping the firing rate to the interval [-1,1], adding noise (mean = 0, range = 0.25) and multiplying the result by a gain (range [0.01,10]). Similarly, the incoming currents to sensory neurons are calculated by adding to the local light intensity a noise term (mean = 0.25, range = 0.25) and then multiplying by a gain factor taken from the same range as above. Left/right symmetry is enforced only for gain values.

All parameters are genetically encoded, with the exception of sensory gains which may either be encoded genetically or regulated by an additional effector neuron

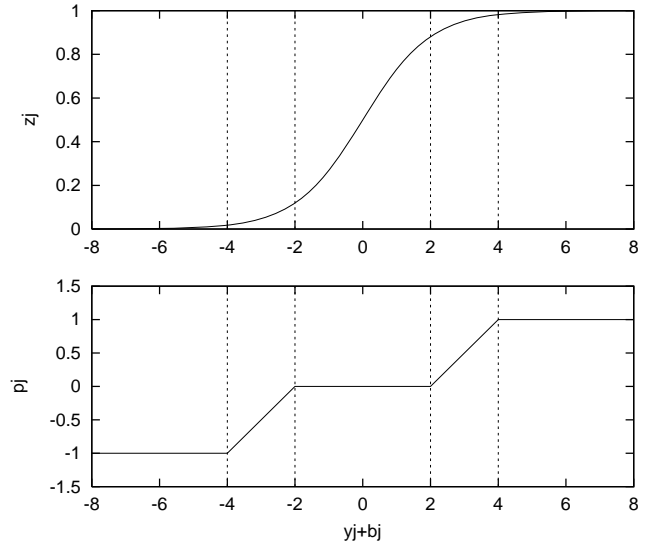


Figure 2: Facilitation of local plasticity as a function of 'cell potential'. Top: neuron activity ('firing rate'). Bottom: strength and sign of local plastic facilitation.

(but only one choice is allowed during each run).

Each connection between neurons has two additional parameters: an integer in the range [0,3] indicating the plastic rule that affects the connections, and a rate of change η_{ij} (range [-0.9,0.9]) expressing the strength and direction of change. The rules are:

$$\begin{aligned} R0 : \Delta w_{ij} &= \delta \eta_{ij} p_j z_i z_j, \\ R1 : \Delta w_{ij} &= \delta \eta_{ij} p_j (z_i - z_{ij}^o) z_j, \\ R2 : \Delta w_{ij} &= \delta \eta_{ij} p_j z_i (z_j - z_{ij}^o), \\ R3 : \Delta w_{ij} &= 0, \end{aligned}$$

where Δw_{ij} is the change per unit of time to w_{ij} , δ is a linear damping factor that constrains change within allowed weight values, and p_j is the degree of local plastic facilitation, explained below. Rule 0 expresses Hebbian/anti-Hebbian change (depending on the signs of p_j and η_{ij}). Rules 1 and 2 potentiate or depress the strength of the connection depending on how the activity of the presynaptic and postsynaptic neurons (respectively) relates to a threshold value z_{ij}^o . This parameter depends linearly on the current weight w_{ij} , so that it takes a value of 0 when the weight is at its minimum (-8) and 1 when the weight is at its maximum (8). For the average case, rule 2 gives an analogous behaviour to the one proposed by the BCM theory mentioned in the previous section, making potentiation or depression harder when the current synaptic efficacy is respectively too high or too low (or the opposite behaviour depending on the overall sign). Rule 1 behaves analogously but the threshold is applied to the presynaptic neuron. Similar rules have been successfully used for evolving plastic con-

trollers for real robots (Floreano and Mondada, 1998).

The factor p_j (range [-1,1]) indicates the degree and direction of facilitation to plastic change. Its value depends on the activity of the postsynaptic neuron as indicated in Fig. 2. No plastic change occurs when the firing rate is between the bounds shown in the figure by the inner vertical lines (corresponding approximately to $z_j = 0.119$ and $z_j = 0.881$). If the firing rate becomes too high the neuron facilitates plastic change ‘positively’ by linearly increasing p_j until it reaches a value of 1. It remains there for higher rates. For low firing rates the same happens with the difference that facilitation is in the opposite direction. The actual form of change depends also on the sign of η_{ij} which is genetically specified. In this way, each individual neuron acts locally by facilitating plasticity under certain conditions and in certain directions. Facilitation does not reflect any *a priori* measure of value for the whole robot as is often the case in architectures that make use of ‘value systems’ (e.g., Friston et al., 1994, Reeke Jr. et al., 1990, Scheier and Pfeifer, 1995, Sporns and Edelman, 1993).

The network is updated using an Euler integration method with a time step of 0.2 (typical runs have been tested against an order-4 Runge-Kutta resolution for stability).

4.3 Genetic Algorithm

A population of 60 robots is evolved using a steady-state, rank-based genetic algorithm with elitism. All real-valued parameters are encoded in a real-valued vector (size = 146 for 8 neurons), each component encoding a single parameter in the neural network. These components belong to the range [0,1], and are linearly scaled to the range corresponding to the parameter encoded (with the exception of gain values which are exponentially scaled). A global mutation operator is used (Beer, 1996, Di Paolo, 2000) which adds a small random vector to the real-valued genotype each time a mutation occurs. Individual components are not changed if the new value is not within [0,1]. The rules of plastic change are encoded in an integer genotype of length 64 with three possible alleles per loci corresponding to each rule (point mutation is used). The overall mutation rate is chosen so as to have a probability of no mutation of about 70%. Uniform recombination is also used.

Each individual robot is run for a number of independent evaluations (typically 5). Its fitness is calculated by averaging the fitness obtained in each evaluation minus 20% of the standard deviation to favour low variability. Each evaluation consists of the serial presentation of 6 distant light sources which the robot must approach in turn and remain close to. Only one source is presented at a time for a relatively long period T_S chosen randomly for each source from the interval $[0.75T, 1.25T]$, with $T = 400$ (2000 time steps) or $T = 800$ (4000 time

steps). After T_S the source is extinguished and another one appears at a random distance, [50,100], and angle, $[0, 2\pi)$. The intensity of each source is randomly chosen from the interval [500,1500], so that, on average, the intensity of light at the initial stages is of the same order of magnitude as the background noise on the sensors, thus encouraging the use of active seeking strategies.

Fitness is calculated by adding three terms. F_D corresponds to the proportion of reduction between the final and initial distance to a source, $1 - D_f/D_i$, (D_f : final distance to source, D_i : initial distance to source). This term is taken as 0 if $D_f > D_i$, and it is calculated for each source and then averaged for the whole evaluation. F_P indicates the proportion of time (over the whole evaluation) that the robot spends within a distance of 4 body radii of a source, and F_H indicates the time-average of the proportion of neurons that have behaved homeostatically (that is, without inducing plasticity). These factors (all between 0 and 1) can be differently weighted. Weights always sum 1. It was found that assigning too high a weight to F_H results in robots which are homeostatic, but do not move. Typical weights for F_H are between 0.15 and 0.2. Of the other two components, permanence near the source is favoured with typical weights between 0.64 and 0.68.

The search algorithm is run for between 1000 and 2000 generations, taking usually a few hundreds to achieve a considerably high level of average fitness. The capability of the evolved robots to adapt to a series of sensorimotor disruptions is then tested individually.

5. Results

5.1 Long term stability

Robots evolved for normal phototaxis are tested for long-term stability of their behaviour. In order to do this a robot is run for a much longer evaluation consisting of the presentation of a large number (between 100 and 400) of sources. If the robot is observed to perform phototaxis during all this time it is considered stable. This is done to test whether normal phototaxis is stable in the long term given that slow internal changes could be going on. Typically, about half of the evolutionary searches performed so far (about 10) have resulted in populations with a majority of stable individuals. Populations without long-term stability are considered as failures since they do not conform to the hypothesis we want to test.

5.2 Adaptation to left/right inversion of vision

In the current setting, the closest situation to actual experiments on the inversion of the visual field consists in exchanging the position of the sensors. It is for this reason that sensors have been placed relatively far apart from each other, so that an exchange of positions does

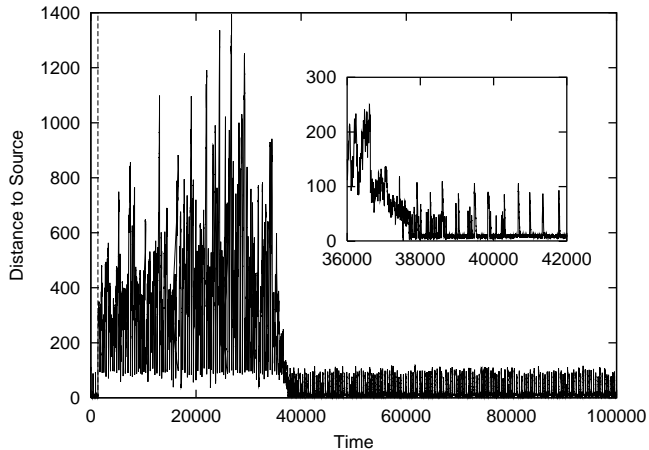


Figure 3: Distance from robot to sources. Each source lasts for about 400 time steps. The vertical dashed line shows onset of inversion of vision. Inset: detail at the time of adaptation.

not result in a trivial modification³.

Following a short initial period of normal phototaxis, an individual robot is tested in an inverted visual field by exchanging the position of the sensors just after one source has been extinguished and another one appears. The robot is at first unable to approach the source and turns in the opposite direction. Environmental stimulation would eventually fall below noise level if another distant source were not presented to the robot⁴ after a time T_S . After a long period, about half of the robots tested eventually adapt to the new sensorimotor situation and perform perfect phototaxis.

This result is shown for two different individuals in Figs. 3 and 4 where the distance to the current source is plotted as a function of time. Fig. 3 corresponds to an individual from a population evolved using $T = 400$, a fitness weight for homeostasis of 0.15, and for permanence near the source of 0.64 (2000 generations, but similar results having been obtained after 1000 generations). The individual corresponding to Fig. 4 has been evolved using $T = 800$, a fitness weight for homeostasis of 0.2, and for permanence near the source of 0.68 (1000 generations). The vertical dashed line shows the moment of inversion.

³At a preliminary stage, some experiments were performed in which non-plastic robots were simultaneously evolved to perform under both conditions (normal and inverted vision) to test how difficult this task was. It was found that, if the angle of the sensors was smaller than 60 degrees, then the task was rather trivial. For larger angles, in most cases fitness remains low for an important part of the evolutionary search, until it hits upon the obvious solution of using only one sensor and ignoring the other.

⁴This is the main reason why the multi-source scheme has been used. For adaptation to occur, it is essential, that the robot be able to act within the environment, and that such actions be the source of stimulations, and possible internal changes. An alternative would be to use a finite number of fixed sources within a limited arena.

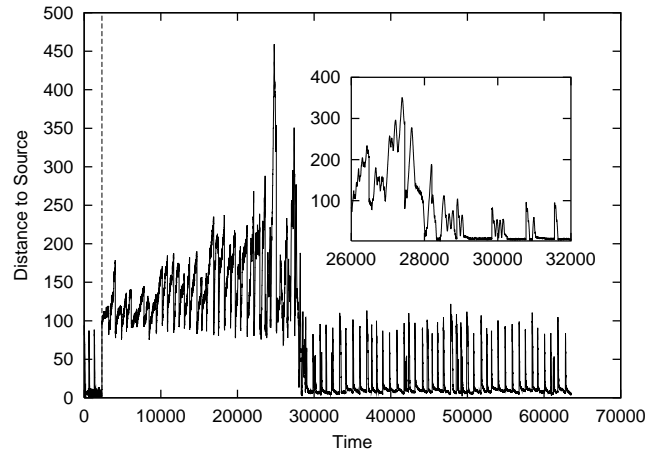


Figure 4: Same as in Fig. 3 for a differently evolved individual. Each source lasts for about 800 time steps.

Before inversion, phototaxis is evident from the spikes in distance (indicating the extinction of one source and the appearance of another) followed by a sharp decrease (indicating fast approach and nearby permanence). After inversion this behaviour disappears, and the distance from the source takes very high values (indicating ‘negative phototaxis’). In both cases it is clear that, after a relatively long period, the robot adapts (insets show the moment of adaptation in more detail). The resulting behaviour is again stable phototaxis.

Interestingly, the time to adaptation is an increasing function of the time of normal ‘development’ before inversion, Fig. 5, thus indicating that the capacity for plastic change is ‘hardened’, and that there is a critical period for adaptation in these robots. This reproduces known features of animal development and adaptation.

Figs. 6 and 7 show respectively the phototactic behaviour of the same individual before visual inversion and after adaptation to visual inversion. It is clear that the strategies are quite different. Normal phototaxis implies a slight angular movement first in one direction and then in the other so as to ‘centre’ the source in the visual field, and then the robot proceeds towards it. In contrast, the strategy after adaptation to inversion involves fully cycloidal movement, i.e., monotonic angular increase.

Fig. 8 shows, for the same run as Figs. 3, 6 and 7, the change in activity in both motor neurons at the time of adaptation together with incoming synaptic efficacies that have changed considerably for the corresponding effector neurons. Weights that present little or no change are not shown. The transition to a different mode of phototaxis can clearly be seen in this figure. The activity of the right motor changes from a more interesting participation in the production of movement before adaptation (Fig. 6) to quite low values representing con-

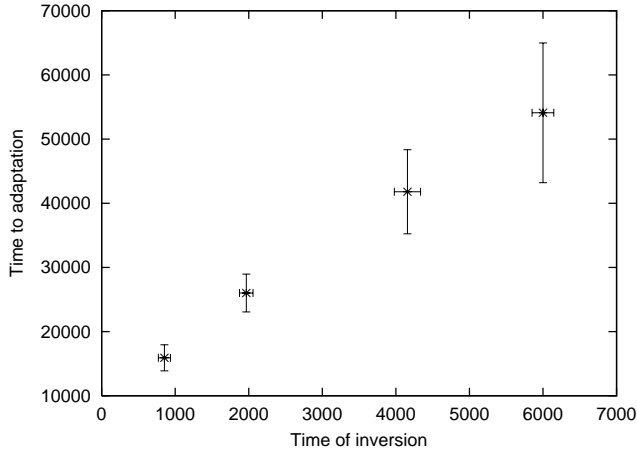


Figure 5: Time to adaptation as a function of time of inversion of vision for one individual (same as shown in Fig. 3). Each point is the average of 5 runs. Error bars indicate standard deviation

tinuous backward motion and therefore overall cycloidal movement (Fig. 7). Other neurons also change in their regimes, but not so sharply. Notice the long term stability of the weights after adaptation. This is interesting because some neurons (for instance the right effector neuron) are not behaving homeostatically. In spite of this, the actual rules and rates of change are such that weight change becomes stable and not necessarily due to saturation.

5.3 Other disruptions

If the homeostatic mechanisms governing plasticity which work during adaptation to visual inversion have not been evolved specifically to this end, then they should also be expected to lead to adaptation in other circumstances. Using an analogous method, adaptation to other sensorimotor disruptions has been tested. For instance, it is possible to simulate sensory or motor lesions by sharply changing the transduction gain of sensor and effector neurons (Shergold, 1997). We have observed that perturbing the gain of the right motor ($\pm 50\%$), the left motor ($\pm 50\%$), the right sensor (-25% , $+50\%$), or the left sensor (-75% , $+100\%$) results in a maladaptive period followed by adaptation. Results are less convincing for the left motor or right sensor because in both cases permanence near the source is either shorter in time or at a greater distance than the normal case. Adaptation in these cases is typically faster than adaptation to inversion of vision (with times to adaptation shorter than 10000). Fig. 9 shows adaptation to a reduction of 40% in the right motor gain.

Modifications to other body parameters and internal lesions (removal of connections or modifications to the

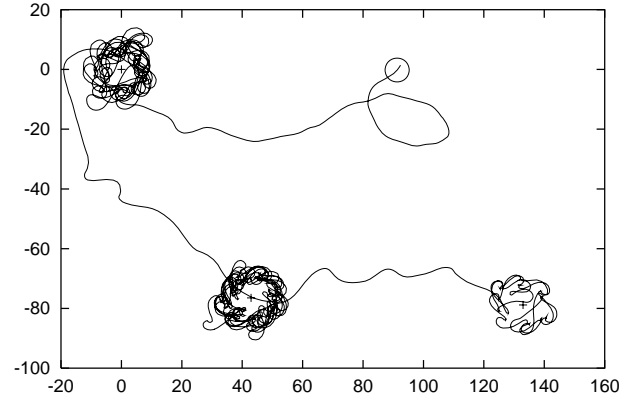


Figure 6: Phototaxis before inversion of vision. The circle indicates the initial position of the robot; sources are indicated with (+).

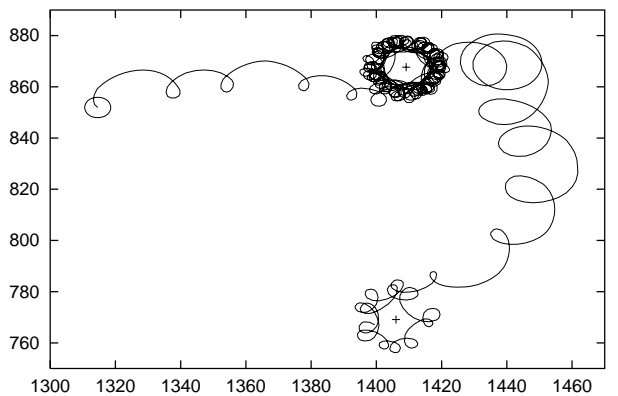


Figure 7: Phototaxis after adaptation to inversion of vision. The circle indicates the initial position of the robot; sources are indicated with (+).

behaviour of single cells, Beer, 1990) have not been explored so far.

6. Conclusions and open questions

Robots evolved to perform simple phototaxis while trying to maintain a certain degree of stability in the plastic structure of their controllers are able to adapt to nontrivial sensorimotor disruptions for which they have not been evolved. Why does this happen? The method seems to work because it selects implicitly for structural stability *and* the desired behaviour as one and the same evolutionary end. By doing so, we believe that we are shaping the space of weight change so that there is a stable attractor when a certain pattern of sensorimotor activity is present (of course, there may be other attractors in the space of weight change, and the method cannot exclude them). In this way, whenever the pattern of sensorimotor activ-

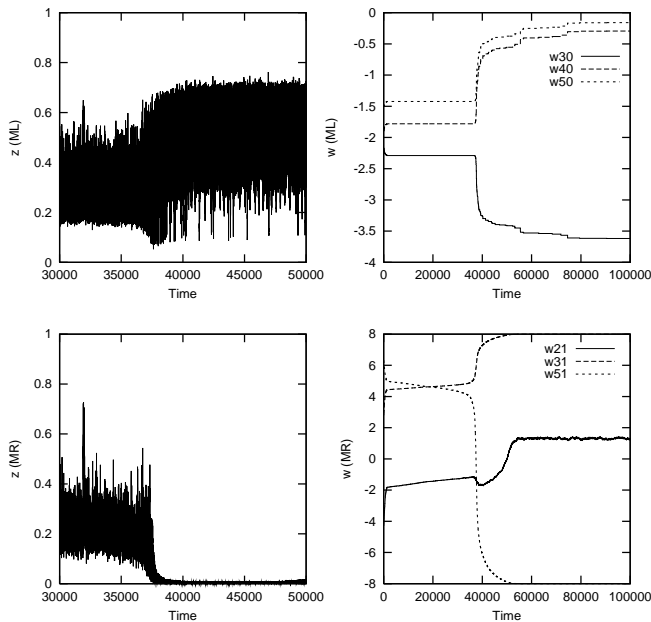


Figure 8: Change in activity (left) and incoming synaptic efficacy (right) for both motor neurons (top: left motor, bottom: right motor) corresponding to the same run shown in Fig. 3.

ity is present (in this case, being near a light source), weights will stop changing (see Figs. 3 and 8). In other cases they will most probably keep on changing. This constitutes an Ashbyan interpretation of why adaptation happens in this case, without the plastic rules being selected to achieve a given behaviour (in other words, without the use of an *a priori* value system).

This is only a tentative explanation and more research is needed. One problem with this explanation is that weight change seems to be rather slow during the period of maladaptation (Fig. 8), and then varies abruptly near the point of adaptation. The explanation is not invalidated by this fact, but one would expect a more ‘exploratory’ dynamics in weight space during maladaptation (something which in Ashby’s Homeostat was achieved by random step changes in parameters). This fact may perhaps be pointing to a less internalistic interpretation of adaptation since it may well be that abrupt modifications are the result of internal mechanisms *and* the presence of an adequate environmental ‘opportunity’ for change. In the Homeostat, the new environmental situation was often held constant. In our case, it is only *statistically* without variations. Sources are randomly placed but individual sources appear in specific locations and have specific intensities. It may be that adaptation happens after the joint occurrence of a serendipitous environmental and internal situation.

Another problem with the proposed explanation is

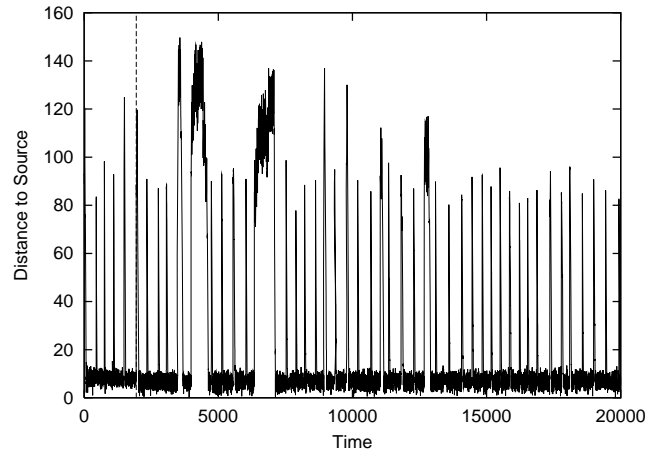


Figure 9: Distance from robot to sources for a 40% reduction in gain in the right motor. The vertical line shows the moment the disruption is introduced

that the very plastic changes that occur can re-shape the dynamics of weight change. If there was initially a stable fixed-point attractor associated with being near a source, plastic changes could eventually modify the characteristics of this attractor so that the resulting stable behaviour is no longer phototaxis.

What these problems suggest is that the method should be refined, particularly since the evolutionary search is not being used as in more ‘classic’ cases in which all constraints are generally placed only on the behaviour of the robot, and not on its behaviour *and* its ‘physiology’. Therefore, the current stage should be taken as a promising, but preliminary step into an expansion of the use of evolutionary methods for robotic design.

Other open questions have to do with the choice of admissible plastic mechanisms. One may readily think of ways of refining the rules of plastic change used in this paper. Simple depression/potentialiation could be included. A covariance rule (which in different circumstances reduces to the other three) given by:

$$R4: \Delta w_{ij} = \delta \eta_{ij} p_j (z_i - \langle z_i \rangle)(z_j - \langle z_j \rangle),$$

where $\langle z \rangle$ is the average activity over a certain time window, could be a more interesting choice. How relevant the included mechanisms are can only be tested with more experiments. But it is possible to hypothesize that good results could be obtained by using rules, or combinations of rules, that are able to introduce variety more easily while they are active than the rules used so far. In other words, rules that tend not to get the system stuck in a small region of possible configurations (cf., Ashby’s Law of Requisite Variety, Ashby, 1956, Ch 11). This could be achieved by implementing both postsynaptic and presynaptic regulation of plasticity and adding

noise. Other possibilities of activity-dependent plasticity include modulatory synapses and ‘volume’ modulation in which the release of diffusible chemicals can affect the properties of an ensemble of local neurons (e.g., Zoli and Agnati, 1996). The latter has recently been successfully implemented in an evolutionary robotics context (Husbands et al., 1998), but it remains an open question how one would define homeostatic internal dynamics in such cases. Probably one should include some global measure of activity of neuromodulators and neurons. There is no reason why the method of selecting for stable structures in the long term should not work for these cases as well, even if the homeostatic variables are not so clearly defined beforehand. It may, however, be computationally expensive to test this idea. In our case, single cell homeostasis has been explicitly (though mildly) favoured during evolution because robots cannot be evolved for ‘a whole lifetime’ (that is, the same order of time that it takes a robot to adapt to an inversion of vision, i.e., a series of about 100 sources per evaluation instead of the 6 we have used). Presumably, if we could, some form of homeostasis would be the evolved result in the case of diffusible modulation, since it will tend to induce stability.

A question of practical interest involves the transition to real robots. Lots of indeterminacies have been included in the model so that the results should be relatively robust. Adequate choice of task and use of noise would probably transform this model into a minimal simulation model (Jakobi, 1997), and these have been shown to transfer adequately to real robots.

An initially surprising feature of adaptation as observed in the model has been the ‘hardening’ of plasticity shown in Fig. 5. Decreased plastic capacity corresponds rather well with basic knowledge about ‘sensitive’ periods of development. In the barn owl, for instance, adaptation to wearing distorting prisms occurs only if the individual is young enough, or if it has had experience wearing the prisms when young; it does not happen in adults (Knudsen, 1998). Unlike the decrease in magnitude of plasticity obtained by explicitly handcoding developmental phases with increasingly lower plasticity (Almásson and Sporns, 1998), in our case, hardening is intrinsic and due to the fact that the method is designed to favour long-term structural stability. This adds to the explanatory power of the hypothesis that links homeostasis and adaptation. However, the flip-side of this same phenomenon is that reverse adaptation after re-inversion of the visual field has not been observed in these robots, presumably because of their diminished capability for plastic change. Tests to verify whether this is the case still have to be performed.

The power behind Ashby’s idea of homeostatic adaptation is that *seemingly purposeful* changes in behaviour arise from the activity of homeostatic, but *be-*

haviourally purposeless, physiological mechanisms. Unlike the related concept of value-based adaptation, where value systems act by following pre-tuned or pre-adapted norms (which in most actual models are also handcoded) telling the robot what is a good or bad change at the behavioural/perceptual level, thus being *intrinsically purposeful* (Reeke Jr. et al., 1990, Sporns and Edelman, 1993), the plastic changes envisioned by Ashby need not ‘push’ the organism teleologically into any predetermined direction. They will keep on changing until stability is achieved. And internal stability, in Ashby’s view, by definition goes hand in hand with adaptation. (Although, as we said earlier, this may only be a sub-set of all possible instances of adaptation.) Obviously, this idea has to be tied up with evolutionary history, as it also has to be tied up with physiological details, if one wishes to address specific cases. But its strength resides in that it is able to account for adaptation in general terms without being buttressed by those details.

In our case, the use of an evolutionary search process begs the question of whether the triggering of local plasticity is eventually selected to act as a value system for the whole robot. If this were the case, often made claims about the pre-adaptation of value systems would be supported by this work. We are not totally convinced at this stage that this is indeed the case. An experimental approach to this question would probably involve hand-designing strictly homeostatic mechanisms, and then studying the behaviour of the whole robot.

Since his own Homeostat, built in the 1940s, Ashby’s idea has seen very few embodiments. We hope to have contributed to a reversion of this situation by combining homeostatic adaptation inspired by recent neurobiological findings with techniques derived from evolutionary robotics, and successfully replicating adaptation to visual inversion.

Acknowledgments

Thanks to Seth Bullock, Inman Harvey, Phil Husbands and Carol Shergold for valuable discussions that helped to shape some of the ideas presented here.

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