LinMap: Visualising Complexity Gradients in Evolutionary Landscapes

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Abstract

This paper describes an interactive visualisation tool, *LinMap*, for exploring the structure of complexity gradients in evolutionary landscapes. *LinMap* is a computationally efficient and intuitive tool for visualising and exploring multidimensional parameter spaces. An artificial cell lineage model is presented that allows complexity to be quantified according to several different developmental and phenotypic metrics. *LinMap* is applied to the evolutionary landscapes generated by this model to demonstrate that different definitions of complexity produce different gradients across the same landscape; that landscapes are characterised by a phase transition between proliferating and quiescent cell lineages where both complexity and diversity are maximised; and that landscapes defined by adaptive fitness and complexity can display different topographical features.

Keywords: complexity, development, evolution, visualisation, artificial cell lineages

1 Introduction

In order to understand the evolution of complexity, we need to understand how complexity is distributed across the multidimensional landscape in which evolution occurs. The evolution of a species is often viewed as a trajectory through a landscape of possibilities. Each point in such a landscape corresponds to a particular genotype (or phenotype), while the height of a point reflects the adaptive fitness of that genotype/phenotype [2]. Over time, natural selection will draw populations toward the fitness peaks. An alternative view of an evolutionary landscape is to associate height with complexity. The contours of the landscape will then represent complexity gradients; and questions about how complexity changes during evolution can be phrased in terms of the alignment between gradients of complexity and adaptive fitness.

There is a pervasive belief that complexity has increased over evolutionary time [6, 45, 34, 8, 50]. Major transitions in evolution have increased the dimensionality of evolutionary landscapes such that more complex organisms (with a greater number of parts, or types of parts) become possible [27]. In particular, the advent of multicellularity and development has resulted in an explosion of new vectors of possible variation [8]. Nonetheless, the view that complexity has increased during evolution is difficult to prove definitively: several instances have been found in which species appear to have evolved toward more simple configurations [34].

Existing studies of biological complexity provide a picture of how certain aspects of complexity (*e.g.*, number of cell types or body parts) can vary across localised regions of evolutionary landscapes [6, 45, 34]. These studies, by drawing on empirical data, focus on regions that have been realised throughout evolutionary history. However, the set of possible organisms is

likely to be far larger than the subset so far encountered by evolution, and it has been observed that regions of interest (*i.e.*, successful species) are distributed in a non-uniform fashion [15, 39]. Modelling is one approach to obtaining a broader understanding of the evolution of complexity.

Several models have been proposed for exploring evolutionary trends in complexity using stochastic processes [45, 33, 37]. These abstract models omit the genetic and developmental mechanisms that underly the production of complex organisms and focus on the macroscopic evolutionary mechanisms that may affect complexity. Such models have been used to distinguish between two possible evolutionary trends: 'passive', in which there is no bias toward increasing complexity, but probabilistic changes to complexity (possibly limited by a lower boundary) result in diffusion toward high complexity; and 'driven', in which changes to complexity are biased toward increases rather than decreases [34]. An alternative approach is to consider how microscopic evolutionary mechanisms affect complexity. Models simulating the evolution of cell lineages have demonstrated how the developmental processes of some organisms may have become simpler over time [3] and that there may be an overall trend toward average complexity [30].

At a lower level of description, a variety of models have been constructed to explore how genetic and developmental mechanisms interact to generate phenotypic complexity (see [42] for a comprehensive review). Some of these models enable complexity to be quantitatively measured (*e.g.*, [5, 29, 26]). Other models have bridged the divide between the genetic and developmental mechanisms that build complex forms and the evolutionary mechanisms with which they interact [23, 40]. In general, the complexity of these models limits their application to a relatively small number of possible parameter combinations and experimental replications, illustrating only limited regions

of evolutionary landscapes that they define.

There are several challenges for the design of a modelling framework that can achieve a synthesis between the short-term developmental processes that build complex phenotypes and the long-term evolutionary processes that result in observable trends. Most importantly, it is necessary to understand how genetics, development and environment interact to generate an evolutionary landscape. We currently lack a general framework that allows evolutionary landscapes to be simulated and visualised. General models of adaptive landscapes have previously been proposed (such as Kauffman's NK landscapes [24]); however, these omit a developmental component, which limits the ways in which complexity gradients can be defined.

The aim of this paper is to introduce a modelling framework and an associated visualisation tool that allows the complexity gradients in evolutionary landscapes to be explored in an intuitive fashion. The following section describes the network-lineage model that was used to define the evolutionary landscape. Several theoretical notions of complexity are then reviewed and interpreted such that they can be applied to the network-lineage model. The visualisation tool, *LinMap*, and its application to the network-lineage model are described. Finally, we discuss several insights into the distribution of complexity across evolutionary landscapes that were obtained using *LinMap*.

2 Artificial Ontogenies: A Cell Lineage Model of Development

During biological development, a single egg cell in a suitable environment transforms into a complex, multicellular organism [48]. While contextual

information from the environment plays a role in this process, the key locus of ontogenetic control is the information encoded in an organism's genome – specifically, in the network of regulatory interactions between its genes [9]. As development proceeds, these genes are expressed in a complex fashion, giving rise to temporal and spatial patterns of cell division and differentiation. Simultaneously, these cells interact, both physically and chemically, to produce complex morphological forms.

Many different approaches to modelling development have been proposed ([42] provides a comprehensive review). Many of these models focus on morphological aspects of development: the growth and organisation of clusters of cells into ordered forms (*e.g.*, [13, 11]). While such models have produced impressive and realistic looking behaviours, their viability as general research tools can be limited by two factors: First, the calculations required to simulate three-dimensional dynamics are computationally expensive, imposing restrictions on the size and number of simulations that can be performed. Second, it can be difficult to automate the measurement and assessment of morphologies. Studies employing complex morphological models have therefore been restricted to exploring only small regions of phenotypic space, and evaluating the phenotypic outcomes in a primarily qualitative fashion (*e.g.*, in terms of visual comparison with biological forms).

An alternative approach is to focus on organisational, rather than morphological, aspects of development. In several biological species (the nematode worm, *Caenorhabditis elegans* being possibly the most widely studied example), the patterns of division and differentiation that occur during development are highly stereotyped and have been precisely measured, providing detailed insight into the type of control decisions involved [44, 43]. The ontogeny of such organisms can be represented by cell lineage dia-

grams: binary trees in which the root node represents the initial egg cell, terminal nodes represent the cells that constitute the final phenotype, and non-terminal nodes represent intermediate developmental state. Cell lineages are useful representations of development because they describe not only the identities of the cells constituting the final phenotype, but also their individual ontogenetic histories. This record facilitates insight into both the nature of converging developmental pathways and the relationship between genotypic change and phenotypic variability [18, 30].

For this study, we used a developmental model in which a cell lineage was generated from the behaviour of a dynamic network. The network-lineage model consists of two components: a network component that generates the gene expression dynamics controlling development and a cell lineage component that defines how these dynamics are interpreted to define an ontogeny. The network component is based on a standard recurrent neural network architecture [12, 20]. Such networks have been widely used in previous models of developmental dynamics [38, 14, 26, 46, 47]. The cell lineage component interprets the dynamics of this network in terms of developmental control. The existence of a cell lineage is implicit in many previous models of development: the output of any simulation model incorporating division and differentiation could, in theory, be described by a cell lineage. While this approach is rarely taken ([49, 30] are notable exceptions), it has several advantages, particularly when measuring the complexity of a developmental process (as described in Section 3).

2.1 The network component

For our purposes, a genetic system is defined as a network of interacting nodes (Figure 1). The network is structured in three layers, consisting of

N_I input nodes, N_R regulatory nodes and N_O output nodes respectively. The input nodes are used to provide information to the network on its current regulatory context (see Section 2.2 below) and their activation is determined by extracellular events rather than the network dynamics. The regulatory nodes represent genes that play a regulatory role only. That is, they have no direct effect on functional behaviour, but mediate between the input nodes and output nodes. The output nodes represent a subset of genes that specifies the functional behaviour of the network (see Section 2.2 below). These nodes have no regulatory outputs; that is, their level of expression has no direct influence on the future dynamics of the network. The activation state of each node is a continuous variable in the range $[0, 1]$, where 0.0 represents a completely inactive gene and 1.0 a fully expressed gene.

Information flows through the network from the input nodes, through the regulatory nodes to the output nodes. The state of the output nodes at a given time is a product, not only of the network's current inputs, but also its dynamic history, as stored by the recurrent interactions between the regulatory nodes. The interactions between nodes in the three layers can be summarised as follows. All input nodes are connected to all regulatory nodes, all regulatory nodes are connected to all output nodes, and all regulatory nodes are optionally connected to all regulatory nodes (including self connections). The level of regulatory connectivity of the network (K) determines the number of inputs each regulatory node receives from other regulatory nodes.

The interactions between two network layers can be represented by a weight matrix, in which the entry at row i, column j specifies the influence that gene j has on gene i. These entries may be positive or negative, depend-

ing on whether the product produced by gene j is an activator or a repressor in the regulatory context of gene i. A zero entry indicates that there is no interaction between the two genes. The inclusion of self-connections (i.e. from node i to node i) allows for the possibility of genes influencing their own regulation. When a random network is created, each of the non-zero entries in its weight matrix are typically initialised to a value drawn from the Gaussian distribution $G(0, W)$, where W defines the interaction strength (or weight scale) of the network. Collectively, the three parameters N, K and W are referred to as the genotypic parameters, as they define a class of network genotypes.

The state of the network was updated synchronously in discrete time steps, with the activation of node i at time $t+1$, $a_i(t+1)$, given by

$$a_i(t+1) = \sigma\Big(\sum_{j=1}^{N_I} w_{ij} a_j(t) + \sum_{j=1}^{N_R} w_{ij} a_j(t) - \theta_i\Big) \tag{1}$$

where N_I and N_R are the number of nodes in the input and regulatory layers respectively, w_{ij} is the level of the interaction from node j to node i, θ_i is the activation threshold of node i, and $\sigma(x)$ is the sigmoid function, given by

$$\sigma(x) = \frac{1}{1 + e^{-x}} \tag{2}$$

Note that, for the N_O output nodes, all of the w_{ij} terms from the input layer will be equal to zero, as there is no direct connection from the input to output layers.

2.2 The cell lineage component

A cell lineage is a record of a developmental trajectory [43]. Considering a cell lineage as a binary tree: the root node represents the fertilised egg cell;

the non-terminal nodes represent the transient states that cells pass through whilst differentiating; and the terminal nodes represent the final differentiated cells that exist at the end of the developmental process. Therefore, it is the terminal nodes of the cell lineage that represent an organism's phenotype, and the topology of the tree describes the relationship between all of the cells that existed at some point during development.

The network model described above is a general purpose computing device. In a developmental system, the computation performed is the transformation of a temporal sequence of contextual inputs into an ordered pattern of cell division and differentiation events. Two input nodes were used to specify the relative position of a cell with respect to its sibling. After division, the activation of these nodes was set to $\{0, 1\}$ in the left daughter and $\{1, 0\}$ in the right daughter. This minimal external input reflects the combined effects of the different contextual signals received by the two cells resulting from their respective positions in the embryo.

The nodes in the output layer of the network were used to determine cell division and differentiation. If the activation of the first output node was above a certain division threshold θ_d, that cell would divide, otherwise it would differentiate. In development, the likelihood of a cell continuing to divide decreases over time. To simulate this, the division threshold was scaled dynamically, according to

$$\theta_d = 1 - 0.01e^{\lambda d} \tag{3}$$

where d was the depth of the current cell and λ was a scaling parameter. Once a cell had stopped dividing, the remaining $N_O - 1$ output nodes were used to determine its differentiation type. A simple 'one-hot', or exclusive,

encoding scheme was used, in which each output node corresponded to a single cell type. A cell was assigned the type corresponding to the output node with the highest activation values. When λ was low, it was possible for a network to continue dividing indefinitely. To ensure that simulations completed in a reasonable time, an upper limit was imposed on the number of levels of division that could occur (*i.e.*, the maximum depth of the cell lineage tree). Any cells that had not differentiated by this time were labelled as undifferentiated.

In summary, a network was used to generate a cell lineage as follows:

1. A single instance of the network, representing a fertilised egg cell, was initialised by setting the activation of all of its nodes to 0.0 (Figure 2, Step 1).

2. The activation of each of the nodes in the regulatory and output layers was updated.

3. If the activation of the division output node was less than θ_d, division occurred (Figure 2, Step 2):

   (a) Two copies of the network were created with identical weights and node activations.

   (b) The activation of the two input nodes was set to $(0, 1)$ in the left daughter and $(1, 0)$ in the right daughter.

4. Otherwise, if the activation of the division output node was greater than θ_d, differentiation occurred and the current cell was assigned the type corresponding to its most active differentiation node (Figure 2, Step 3).

5. Cells that had differentiated underwent no further change. Steps 2 to 4 were repeated for each of the remaining cells.

6. Development ceased when all cells had been differentiated, or some predefined limit on division depth had been reached. Any remaining undifferentiated cells at this stage were labelled as such.

3 Metrics for Measuring Complexity

Complexity is an amorphous subject: while easy to recognise in an intuitive fashion, crystallising these intuitions into a formal definition has proven more difficult [34, 21, 1]. An early insight from complex systems theory was that dynamic processes can be located on a continuous spectrum according to their level of order/disorder: At one end of the spectrum lie stable or periodic processes and at the other end lie random processes. In this view, both stable and random processes are considered to be 'simple' because they can be described simply, by deterministic methods in the first case and statistical methods in the second. Complex processes, in contrast, fall somewhere between: They are neither totally ordered nor totally random [28]. Capturing the aspects of diversity and regularity that combine to produce complexity in a quantitative fashion is therefore more challenging, and a variety of solutions have been proposed ([10] alone reviews several dozen different approaches to formulating complexity definitions).

Within the domain of biological complexity, three classes of complexity definition may be identified, applying to genotypic, phenotypic and behavioural levels of description. Genotypic complexity definitions typically focus on the nucleotide sequences that constitute an organism's genome. Because sequences are amenable to formal characterisation, concepts from

mathematics and physics are readily applicable (see, *e.g.*, [4]). While convenient (given the increasing availability of genome sequence data) measures of sequence complexity suffer from two related problems: they tend to focus on characterising the difficulty of predicting the next symbol in a sequence, rather than the meaning of the sequence. Furthermore, the mapping from nucleotide sequences to protein structure and organismic function is highly nonlinear, and it is not clear that sequence complexity necessarily equates to ideas about complexity at a functional level [1]. Phenotypic complexity mechanisms focus on the structural form of an organism. These measures are defined in terms of, for example, the number of different types of parts or interactions between parts in an organism [34]. Definitions of structural complexity may be further divided into those that measure the complexity of an object (morphological complexity) and those that measure the complexity of a process (developmental complexity). The final class of measures considers the functional complexity of an organism—the variety of possible behaviours it is capable of exhibiting [35]. Functional complexity is particularly intriguing because it assesses an organism not in isolation, but in the context of the environment in which it has involved [31].

For this study, we restricted our investigation to measures of structural complexity, focusing on the complexity both of a given form, and of the process required to generate that form. Even within this class, there are multiple possible metrics that may be defined, building upon the intuition that more complex organisms are likely to consist of (a) a greater number of parts; (b) a greater variety of different types of parts; and (c) a greater variety of different configurations or arrangements of those parts [22, 32]. In the following section several notions of structural complexity are reviewed and formalised in such a way that they can be applied to the cell lineages

generated by the model described in Section 2.

3.1 Morphological metrics: complexity of form

The metrics discussed below focus broadly on morphological aspects of a cell lineage; that is, the fate distribution of the terminal cells (Figure 3).

Number of cells: One of the simplest proposed indicators of the complexity of an organism is its size. Bonner [6] argues that as organisms grow larger they must by necessity become more complex; as the internal requirements for supporting their larger size will become more specialised. The primary advantage of this metric is its simplicity. However, interpreting this metric requires caution: applied strictly it would imply that larger organisms are always more complex than smaller organisms, which is clearly not always the case (despite being larger, a blue whale is not necessarily more complex than a dolphin). The *number of cells* of a cell lineage was defined as its number of terminal nodes.

Number of cell types: Bonner [6] also proposed that an increase in the complexity of an organism would be reflected by an increase in the number of specialised cell types it contained. As with the number of cells, this metric has considerable intuitive appeal. A potential problem with applying this metric to real organisms is the difficulty in classifying different types of cells and the potential for bias in favour of more well-studied organisms [34]. The *number of cell types* of a cell lineage was defined as the number of different types of terminal nodes. Given the definition of the network-lineage model, an upper limit was imposed on this metric by the structure of the underlying control network. Therefore, while there was some scope for variation due to a network not employing all possible cell types during development, its

range was limited by the number of output nodes in the network.

Number of hierarchical levels: Between two organisms containing an equal number and type of components, there may be a difference in how those components are arranged. Hierarchical, in this context, refers to the number of levels of nestedness of a morphology [36]. For example, in the sequence {organelle, cell, organ, organism}, each component contains and partially constrains the behaviour of the earlier components. Given that a cell lineage captures an ontogenetic, rather than a physical, relationship between cells, it is not possible to define a formal measure of hierarchy in this context. However, if we accept a relationship between the ontogeny of a cell and its morphological context, the algorithmic measures of complexity described below may be taken as a proxy for levels of hierarchical organisation.

3.2 Developmental metrics: complexity of process

An alternative approach to defining complexity metrics is in terms of the process that constructed that object. The following metrics consider the development of a cell lineage: how a cell fate distribution is generated.

Algorithmic complexity (deterministic): One approach to measuring the complexity of a system is by considering how much information is required to describe it. It is here that cell lineages are a particularly useful representation of an organism, as they maintain a record of how that system was constructed [7, 3]. First, a cell lineage is converted into a series of unique production rules of the form $X \rightarrow \{Y, Z\}$, indicating that a cell of type X divides to form cells of type Y and Z. X is necessarily an undifferentiated (non-terminal) cell, while Y and Z may be differentiated (terminal) or undif-

ferentiated. The plane of cell divisions is lost ($X \rightarrow \{Y, Z\}$ is equivalent to $X \rightarrow \{Z, Y\}$), but otherwise these rules provide a complete description of a lineage. This initial set of rules is then reduced by removing equivalent rules until a minimal set is arrived at. The *deterministic algorithmic complexity* of a lineage was defined as the size of this minimal set as a proportion of the total number of divisions (Figure 3).

Algorithmic complexity (non-deterministic): One of the implications of the algorithmic complexity measure used by Azevedo *et al.* [3] is that each of the rules corresponds to an intermediate cell state that will always produce an identical sublineage in a deterministic fashion. An alternative view is that an intermediate cell state could define the subset of terminal cell fates possible in that sublineage, but not necessarily the exact structure of that sublineage. To investigate the effect of this definition, we defined a second algorithmic complexity metric in which the production rules were non-deterministic. Rules now took the form of $\{A, B, C\} \rightarrow \{\{A, B\}, C\}$, where $\{A, B, C\}$ and $\{A, B\}$ are undifferentiated cells that will eventually give rise to differentiated cells of types A, B and C, or A and B, respectively, and C is either a differentiated cell that may or may not continue to divide in a proliferative fashion (*i.e.*, to give rise to more cells of type C). As with deterministic algorithmic complexity, redundant rules are removed from this set, and *non-deterministic algorithmic complexity* was defined in terms of the size of this minimal set as a proportion of the total number of divisions.

Weighted complexity: Comparative studies indicated that each complexity metric displayed certain limitations when applied to cell lineages [16]. A final complexity metric, designed to address these limitations, combined both morphological and developmental aspects of complexity. The *weighted*

complexity of a lineage was defined as the product of its number of terminal cells and its non-deterministic algorithmic complexity.

4 Visualising and Exploring Evolutionary Space

The complexity metrics described above provide a means of quantifying lineages. One way that these metrics can be used to characterise an evolutionary landscape is through the use of statistical ensembles, in which random samples of lineages are generated using particular parameter combinations. In this way, a relationship can be derived between these parameters and the complexity distributions of the corresponding lineages. Results obtained using ensembles of artificial cell lineages have been reported in [19, 16, 30]. However, a statistical approach provides only a summary view of complexity, with limited information about the micro-level structure of the landscape. But it is at the micro-level that much evolutionary change is likely to occur (*e.g.*, as a result of mutation events). Questions that cannot be answered using a statistical approach include: Where in a parameter space are complex behaviours likely to be located? How uniform is the distribution of complexity across a parameter space? What shape do complexity thresholds take? What types of phenotypes are located around a particular point in an evolutionary landscape? How are the complexity gradients in a landscape correlated with adaptive gradients? And finally, what do the members of these statistical ensembles actually look like?

To address these questions, we designed a visualisation tool, *LinMap*, that enabled the micro-scale structure of an evolutionary landscape to be explored interactively. This section describes the design and implementation of *LinMap* and its application to the evolutionary landscapes generated by the network-lineage model described in Section 2.

Several factors influenced the design of *LinMap*. One was the dimensionality of the parameter space: Each gene interaction in the network-lineage model constitutes a dimension along which evolutionary variation may occur; therefore the evolutionary landscape, even of very small systems, is highly multidimensional. Another factor was the importance of conveying the structure of this space, such that neighbourhoods and gradients in an evolutionary landscape could be clearly visualised. Finally, the computational efficiency of the visualisation was important: we did not want data generation to be a bottleneck that restricted the scope of exploration. As noted earlier, many models of development are computationally intensive. Visualising the behaviour of such models typically involves running batch jobs offline to generate data and then rendering them in some graphical form 'after the event'. If the results of this visualisation suggest further explorations, another iteration of the generate/visualise cycle must be performed. The resulting delay between the analysis of current results and the generation of further results discourages lengthy or in depth exploration of a model system. In contrast, by reducing the length of this cycle (to a matter of seconds) it becomes possible to view a much larger range of lineages and landscapes, and to explore these landscapes in greater depth.

Dimensionality: The high dimensionality of the parameter space was addressed by choosing two parameters to vary such that a comprehensible two dimensional 'slice' could be taken through space in a sensible way. Both the network and cell lineage components of the model are amenable to parameterisation. As described above, the network component of the developmental model can be parameterised by size (N), connectivity (K) and weight scale (W). When considering the individual behaviour of a single network (rather

than the average behaviour of an ensemble of networks), varying either N or K causes a discontinuous transformation. That is, changes to the pattern of interaction in a single network produces new behaviour that is effectively uncorrelated with its prior behaviour. In contrast, W may be varied in an continuous fashion, with smooth transitions in dynamic behaviours, and it was used to define the first axis.

The second axis was defined by the division threshold scaling parameter λ. As described above, a cell will divide if the activation of its division node is above a certain value, θ_d. This value increases over developmental time as described by Equation 3. One useful property of λ is that the sequence of lineages obtained as it is varied is monotonic; that is, once a particular transition from lineage l_a to l_b has been observed upon increasing from λ_a to λ_b, no further increases to λ_b will result in l_a being observed again. Therefore if two lineages l_a and l_b are equivalent, all values of λ between λ_a and λ_b will also produce equivalent lineages. As a result of this property, it is possible to efficiently explore the space of possible lineages in the direction of the λ axis in a recursive fashion as follows:

RecursiveExplore(λ_a, λ_b, *depth*):

    generate lineages l_a and l_b

    set $\lambda_c = \frac{\lambda_a + \lambda_b}{2}$

    generate lineage l_c

    if $(l_a \neq l_c)$ **and** $(depth > 0)$:

        RecursiveExplore(λ_a, λ_c, $depth - 1$)

    if $(l_c \neq l_b)$ **and** $(depth > 0)$:

        RecursiveExplore(λ_c, λ_b, $depth - 1$)

    else return

Initially, the RecursiveExplore procedure is called with λ_a and λ_b equal to the minimum and maximum bounds of the range being explored (typically $[0.0, 1.0]$). As the procedure is called recursively, this range is continually subdivided, with regions of equivalent lineages being ignored and regions of varying lineages being explored in greater detail. The *depth* parameter imposes a limit on the level of recursion. Increasing *depth* results in a map with greater resolution along the λ axis, at the expense of increased processing time.

Switching from a regular to a recursive mode of parameter exploration has two advantages. The first is increased computational efficiency: more processing time is spent evaluating unique individuals while less time is wasted on repeatedly evolving the same individual. The second advantage is that the resolution of the map is automatically increased around regions of interest, without the need for user intervention. Figure 5 demonstrates the benefits of recursive versus regular parameter exploration using a simple sigmoidal function. Figure 6 shows a sample heat map ($N = 8, K = 8, \lambda = [0.0, 1.0], W = [0.01, 2.0]$). Each vertical slice on the map describes the lineages produced when the weight scale (W) is held fixed and the division threshold scale (λ) is varied. Comparing the lineages A, B and C in Figure 6 demonstrates that, for relatively low values of W, increasing λ decreases the depth at which cells switch from division to differentiation. Horizontal slices describe how the lineages produced vary as W is varied while λ is held fixed: the probability of a lineage containing undifferentiated cells decreases, as does the size of the lineage.

Interactivity: A second feature of *LinMap* that addressed the requirements defined above was the use of an interactive heat map as both a repre-

sentation of the parameter space and an interface for exploring the lineages within that space. The heat map responded to several types of user input: Clicking any location on the heat map region of the *LinMap* interface displays the lineage corresponding to the parameter combination at that location. Selecting a rectangular region of the map allowed that region to be enlarged. In addition, a control panel allowed other parameters, such as the bounds of the map, network size and connectivity and random seed, to be adjusted.

A key requirement for achieving interactivity was ensuring that the response to user action was as rapid as possible. Several steps were taken to increase the computational efficiency of *LinMap* so that users could interact with the model in real time. When an evolutionary landscape is first generated, a relatively coarse-grained resolution is used. Numerous maps could therefore be generated very rapidly, enabling a broad overview. If more detail was required for a particular map, the resolution can be increased (Figure 7), and regions of interest can be selectively enlarged (Figure 8). Further options provided included the ability to view multiple maps simultaneously (Figures 4 and 9), and the definition and generation of contours showing a cross-section of the landscape (Figure 10).

Availability: *LinMap* should run on any platform with a suitably configured Java^{TM}5.0 environment. Full source and binaries for the most recent version are freely available from http://www.itee.uq.edu.au/∼nic/_linmap/.

5 Insights into Complexity and Evolution

5.1 Typical cell lineage characteristics

One striking insight provided by *LinMap* was that even very small networks (two–eight regulatory nodes) are capable of generating a broad range of complex developmental patterns (several examples are shown in Figure 6). The cell lineages generated from network dynamics are characterised by a quasi-regular structure. Both the topology of lineages (generated by the pattern of cell division) and the distribution of cell fates over the terminal nodes of the lineage (generated by the pattern of differentiation) display a degree of systematicity (Figure 9). Specific classes of regularity that were observed included:

Translational symmetry: a cell divided to produce two daughter cells with identical potentials, that is, two cells giving rise to identical sublineages. When all cells divided to produce two daughter cells with the same potential as the parent cell, this led to proliferation. However, it was also possible that the first cell division (for example) would produce two daughter cells, with the same potential as each other, that would go on to produce non-homogeneous sublineages, in which case the entire cell lineage would display translational symmetry without being homogeneous.

Recursive production: a cell divided to produce one daughter cell with the same potential as its parent and one with different potential. It was commonly observed that either the left or right cell in a lineage would continually divide, while the other differentiated, producing a pattern analogous to the stem-cell mode of cell division.

Modularity: Identical sublineages could appear at multiple locations in a cell lineage, suggesting that the cells producing these sublineages share a common potential. The appearance of modularity also suggests that a particular cell fate potential can be achieved via multiple developmental trajectories, since each cell in a lineage has received a unique sequence of inputs.

Such regularities have also been recognised in biological lineages such as that of the nematode *Caenorhabditis elegans* [44, 25, 3]. The dynamic behaviours in biological development is likely to be more sophisticated than those generated by our stylized model. However, the ease with which such realistic lineages could be obtained suggests that features such as symmetry and modularity may not be difficult control tasks, but may be generic phenomena of particular types of dynamic systems (as previously observed in [23]).

5.2 Complexity gradients

Depending on the precise pattern of gene interactions, the network-lineage model was capable of generating a wide range of different complexity maps, with considerable variation in the distributions of complexity and lineage diversity. Whereas some networks mapped to regions of parameter space filled with a diverse range of lineages (such as that shown in Figure 9), others mapped to much more homogeneous regions containing only a limited number of different lineages.

Some features of the complexity maps *did* recur over multiple random network initialisations. For example, if W was low, high values of λ were required for any differentiation to occur. As W increased, the probability of an initial cell never dividing increased, particularly when λ was high.

Otherwise, the shape of the transitions from low to high complexity was strongly dependent on the properties of the individual network.

Within a single complexity map generated by a parameterised network, transitions between complexity values tended to be relatively smooth, with occasional large jumps. That is, as W and λ were varied, neighbouring lineages tended to share similar levels of complexity. Occasionally, larger jumps in complexity were observed (*e.g.*, the dominant transition running diagonally from top-left to bottom-right in the maps shown in Figure 9). In these cases, increasing the resolution of the map (*i.e.*, decreasing the size of increments for W and λ) frequently resulted in the appearance of intermediate lineages.

5.3 Comparing complexity metrics

Comparing the complexity maps produced by different metrics on the same set of lineages revealed some topographical inconsistencies between the complexity gradients they defined. While the location of the major complexity transitions were consistent across metrics, the size and orientation of these transitions varied considerably. The top left map in Figure 9 indicates that the total number of terminal cells in a lineage decreases as W and λ increase. In contrast, the top right map indicates that many of the lineages with a large number of terminal cells never cease dividing and hence contain no differentiated cells. The bottom left map highlights a shortcoming of the unweighted algorithmic complexity measures: they assign disproportionately high complexity values to very small lineages, as indicated by the bright patch that occurs for high values of both W and λ. The weighted complexity metric (bottom right) rectified this anomaly and suggested a link between high complexity and lineage diversity: the most dense concen-

trations of different lineages and the most complex lineages both co-occur in a region of parameter space between uncontrolled cell proliferation and absolute cell quiescence.

5.4 Complexity as a boundary phenomenon

High complexity lineages tend to be clustered into particular regions of parameter space. It has previously been noted that complex or interesting behaviours tend to be boundary phenomena, occurring in the transition from one type of simple behaviour to another [28]. The two types of simple behaviour that bracket interesting behaviours in this situation are unchecked proliferation—a cell lineage that continually divides without ever differentiating—and quiescence—a cell lineage in which the initial cell differentiates immediately without ever dividing. In between these two extremes lie a wide variety of more complex structures.

Complexity, regardless of which metric is used, is distributed in a non-uniform manner throughout ontogenetic space. While intermediate values of both W and λ consistently produced the greatest density of high-complexity lineages, the exact locations of these regions varied among networks. One consistent feature was a phase transition between trivial lineages (zero or very low complexity, occurring when both W and λ are low) and nontrivial lineages (*i.e.*, weighted complexity $>\sim 4.0$). Figure 10 illustrates how complexity tends to vary across this transition. In general, the most complex lineages are located in a boundary region whose location is defined by both W and λ. The lineages to the left of this region fail to differentiate, while the lineages to the right of this region fail to divide. The size of this boundary region was observed to vary substantially between different network seeds. In some cases, the region was virtually non-existent, and small increases

in W and/or λ transformed a proliferating lineage into a quiescent lineage without an intervening complex regime.

5.5 Complexity and evolution

LinMap also enables direct comparisons between complexity gradients and adaptive gradients in a single landscape. Figure 11 contrasts an adaptive gradient and a complexity gradient. The adaptive gradient was obtained by selecting a particular cell lineage to represent the optimal phenotype in a given context. The fitness of all other cell lineages was then defined by their distance from this optimum. For organisms with invariant patterns of development, the physical location of a cell is often closely tied to its position in the lineage [44]. We therefore defined the distance between cell lineages in terms of similarity between the order and composition of their terminal cell sequences. The fitness of a particular sequence defined as the Levenshtein distance [41] between its terminal cell fate sequence that of the optimal cell lineage. Levenshtein distance is defined in terms of the minimum number of transformations required to change one sequence into another, where possible transformations were the insertion, deletion and substitution of cell fates. An advantage of this distance metric was that, not only could sequences of dissimilar lengths be compared, but common sub-sequences could be recognised despite shifts in their location.

We used a simple hillclimbing to explore the dynamics of an adaptive process on the evolutionary landscape defined by such a fitness function. The target fate distribution was the *C. elegans* male.V6Lpap lineage (see [7]), which consists of twelve terminal cells with fate distribution: ABCBDAABCBDA. The hillclimbing algorithm was initialised with a random network ($N = 8, K = 8, W = 1.0, \lambda = 0.85$); at each time step, a new

network was generated from the current network by choosing a weight at random and replacing it with a value drawn from the distribution $G(0, W)$. If the fitness of the new network was equal to or greater than that of the current network (*i.e.*, it's fate distribution was at least as close, using the distance metric described above, to the target fate distribution), it replaced the current network.

While fitness increases monotonically over the course of an adaptive walk, deterministic algorithmic complexity undergoes periods of both increase and decrease (Figure 12). The high rate of change of complexity indicates the presence of neutrality in the landscape: Multiple genotypes, each generating lineages of differing complexity, nonetheless map to equally fit fate distributions. While there is a general upward trend to complexity, the final (perfectly fit) lineage is in fact simpler than many of it's predecessors in the adaptive walk. A possible explanation for this is the translational symmetry of the target lineage: After the first (symmetric) division, both daughter cells produce sublineages that are identical to one another. Such a lineage may be described, and controlled, in a simpler fashion than one that is less modular.

5.6 Beyond cell lineages

This paper has described a visualisation tool applied to a specific developmental model, and motivated by a particular set of questions. However, we believe that the design principled embodied in *LinMap* are more generally applicable to a wide range of models and research agendas. Within the domain of development and evolution, one possibility would be to extend the scope of *LinMap* to encompass more detailed developmental models. While the cell lineage representation was particularly useful for the purposes of

our study, two or three dimensional models incorporating spatial information, signalling and morphogenesis could be substituted ((*e.g.*, [26, 23]). One challenge associated with extending *LinMap* in this direction would be maintaining a balance between the computational expense of evaluating the developmental process and the responsiveness of the visualisation.

Beyond the domain of development, we have also considered the application of a *LinMap*-like tool to the evaluation of ecosystem models, to determine the properties of phase transitions arising from interacting populations. The practical steps required to generalise *LinMap* are relatively simple: The software has been designed such that the visualisation framework can be decoupled from the underlying model in a straightforward fashion. The domain-specific components that must be specified are the system parameters that define the heat map axes, and the evaluation metric that determines the height at each point. A high level description of the design patterns used in *LinMap* is provided in [17].

6 Conclusions

This paper has presented an interactive visualisation tool, *LinMap*, for exploring the complexity gradients in evolutionary landscapes. This tool has been applied to exploring the landscapes generated by a stylised model of biological development. The network-lineage model, to which *LinMap* was applied, is a useful framework for modelling the interaction of evolutionary and developmental dynamics. It combines a rich variety of dynamic behaviours with a high level of computational efficiency, enabling large and detailed evolutionary landscapes to be explored rapidly and efficiently. Several uses of *LinMap* have been demonstrated: the rapid exploration of a multidimensional parameter space; the comparison of different complexity

measures; the identification of a complexity phase transition in evolutionary space; and the comparison between complexity gradients and adaptive gradients.

Complexity and adaptive fitness constitute parallel views of an evolutionary landscape. While a complexity gradient may be constant, adaptive gradients will change depending on the adaptive context of a population: Visualisation allows us to assess the degree of correlation between these gradients in different adaptive contexts, and to begin to understand the conditions governing increases and decreases in complexity during evolution.

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References

[1] Adami, C. (2002) What is complexity? *BioEssays*, **24**, 1085–1094.

[2] Arnold, S. J., Pfrender, M. E., and Jones, A. G. (2001) The adaptive landscape as a conceptual bridge between micro- and macroevolution. *Genetica*, **112–113**, 9–32.

[3] Azevedo, R. B. R., et al. (2005) The simplicity of metazoan cell lineages. *Nature*, **433**, 152–156.

[4] Badii, R. and Politi, A. (1997) *Complexity – Hierarchical Structures and Scaling in Physics*. Cambridge: Cambridge University Press.

[5] Bongard, J. and Pfeifer, R. (2001) Repeated structure and dissociation of genotypic and phenotypic complexity in artificial ontogeny. In L. Spector *et al.* (Eds.), *GECCO 2005: Proceedings of the 2001 Conference on Genetic and Evolutionary Computation*, (pp. 829–836), San Francisco, CA: Morgan Kauffman.

[6] Bonner, J. T. (1988) *The Evolution of Complexity*. Princeton, NJ: Princeton University Press.

[7] Braun, V., Azevedo, R. B. R., Gumbel, M., Agapow, P. M., Leroi, A. M., and Meinzer, H. P. (2003) ALES: cell lineage analysis and mapping of developmental events. *Bioinformatics*, **19**, 851–858.

[8] Carroll, S. B. (2001) Chance and necessity: the evolution of morphological complexity and diversity. *Nature*, **409**, 1102–1109.

[9] Davidson, E. H., McClay, D. R., and Hood, L. (2003) Regulatory gene networks and the properties of the developmental process. *Proceedings of the National Academy of Science, USA*, **100**, 1475–1480.

[10] Edmonds, B. (1999) *Syntactic Measures of Complexity*. Unpublished Ph.D. thesis, University of Manchester.

[11] Eggenberger, P. (1997) Evolving morpohologies of simulated 3D organisms based on diffferential gene expression. In P. Husbands and I. Harvey (Eds.), *Fourth European Conference on Artificial Life*, (pp. 205–213), Cambridge, MA: The MIT Press/Bradford Books.

[12] Elman, J. L. (1990) Finding structure in time. *Cognitive Science*, **14**, 179–211.

[13] Fleischer, K. and Barr, A. H. (1994) A simulation testbed for the study of multicellular development: The multiple mechanisms of morphogenesis. In C. Langton (Ed.), *Artificial Life III*, (pp. 389–416), Redwood City, CA: Addison-Wesley.

[14] Furusawa, C. and Kaneko, K. (1998) Emergence of rules in cell society: differentiation, hierarchy and stability. *Bulletin of Mathematical Biology*, **60**, 659–687.

[15] Gavrilets, S. (1997) Evolution and speciation on holey adaptive landscapes. *Trends in Ecology and Evolution*, **12**, 307–312.

[16] Geard, N. (2006) *Artificial Ontogenies: A Computational Model of the Control and Evolution of Development*. Unpublished Ph.D. thesis, The University of Queensland.

[17] Geard, N. (2006) *Two design patterns for visualising the parameter space of complex systems* (Technical report), ARC Centre for Complex Systems, The University of Queensland.

[18] Geard, N. and Wiles, J. (2005) A gene network model for developing cell lineages. *Artificial Life*, **11**, 249–268.

[19] Geard, N. and Wiles, J. (2006) Investigating ontogenetic space with developmental cell lineages. In L. M. Rocha *et al.* (Eds.), *Artificial Life X: Proceedings of the Tenth International Confereonce on the Synthesis and Simulation of Living Systems*, (pp. 56–62), Cambridge, MA: The MIT Press/Bradford Books.

[20] Hertz, J., Krogh, A., and Palmer, R. G. (1991) *Introduction to the Theory of Neural Computation*. Redwood City, CA: Addison-Wesley.

[21] Heylighen, F. (1999) The growth of structural and functional complexity during evolution. In F. Heylighen, J. Bollen, and A. Riegler, (Eds.), *The Evolution of Complexity*, (pp. 17–44), Dordrecht: Kluwer Academic.

[22] Hinegardner, R. and Engleberg, J. (2003) Biological complexity. *Journal of Theoretical Biology*, **104**, 7–20.

[23] Hogeweg, P. (2000) Shapes in the shadow: Evolutionary dynamics of morphogenesis. *Artificial Life*, **6**, 85–101.

[24] Kauffman, S. A. (1993) *The Origins of Order: Self-Organization and Selection in Evolution*. Oxford: Oxford University Press.

[25] Kenyon, C. (1985) Cell lineage and the control of *Caenorhabditis elegans* development. *Philosophical Transactions of the Royal Society B: Biological Sciences*, **312**, 21–38.

[26] Keränen, S. V. E. (2004) Simulation study on effects of signaling network structure on the developmental increase in complexity. *Journal of Theoretical Biology*, **231**, 3–21.

[27] Kirschner, M. W. and Gerhart, J. C. (2005) *The Plausibility of Life : Resolving Darwin's Dilemma*. New Haven, CT: Yale University Press.

[28] Langton, C. (1990) Computation at the edge of chaos: Phase transitions and emergent computation. *Physica D*, **42**, 12–37.

[29] Lehre, P. K. and Haddow, P. C. (2007) Phenotypic complexity and local variations in neutral degree. *BioSystems*, **87**, 233–42.

[30] Lohaus, R., Geard, N., Wiles, J., and Azevedo, R. B. R. (in press) A generative bias towards average complexity in artificial cell lineages. *Proceedings of the Royal Society B: Biological Sciences*.

[31] Maron, M. and Fernando, C. T. (2006) Food webs and the evolution of organism complexity. In *Artificial Life X: Workshop Proceedings*.

[32] McShea, D. W. (1991) Complexity and evolution: what everybody knows. *Biological Philosophy*, **6**, 303–324.

[33] McShea, D. W. (1994) Mechanisms of large-scale trends. *Evolution*, **48**, 1747–1763.

[34] McShea, D. W. (1996) Metazoan complexity and evolution: is there a trend? *Evolution*, **50**, 477–492.

[35] McShea, D. W. (2000) Functional complexity in organisms: parts as proxies. *Biology and Philosophy*, **15**, 641–668.

[36] McShea, D. W. (2001) The minor transitions in hierarchical evolution and the question of a directional bias. *Journal of Evolutionary Biology*, **14**, 502–518.

[37] McShea, D. W. (2005) The evolution of complexity without natural selection, a possible large-scale trend of the fourth kind. *Paleobiology*, **31**, 146–156.

[38] Mjolsness, E., Sharp, D. H., and Reinitz, J. (1991) A connectionist model of development. *Journal of Theoretical Biology*, **152**, 429–453.

[39] Raup, D. and Gould, S. (1974) Stochastic simulation and evolution of morphology: towards a nomothetic paleontology. *Systematic Zoology*, **23**, 305–322.

[40] Salazar-Ciudad, I. and Jernvall, J. (2004) How different types of pattern formation mechanisms affect the evolution of form and development. *Evolution & Development*, **6**, 6–16.

[41] Sankoff, D. and Kruskal, J. (eds.) (1983) *Time warps, string edits and macromolecules*. Redwood City, CA: Addison-Wesley.

[42] Stanley, K. O. and Miikkulainen, R. (2003) A taxonomy for artificial embryogeny. *Artificial Life*, **9**, 93–130.

[43] Stent, G. (1998) Developmental cell lineage. *International Journal of Developmental Biology*, **42**, 237–241.

[44] Sulston, J. E., Schierenberg, E., White, J. G., and Thompson, J. N. (1983) The embryonic cell lineage of the nematode *Caenorhabditis elegans*. *Developmental Biology*, **100**, 64–119.

[45] Valentine, J. W., Collins, A. G., and Meyer, C. P. (1994) Morphological complexity increase in metazoans. *Paleobiology*, **20**, 131–142.

[46] Vohradský, J. (2001) Neural model of the genetic network. *The Journal of Biological Chemistry*, **276**, 36168–36173.

[47] von Dassow, G., Meir, E., Munro, E. M., and Odell, G. M. (2000) The segment polarity network is a robust developmental module. *Nature*, **406**, 188–192.

[48] Wolpert, L. (1998) *The Principles of Development*. Oxford: Oxford University Press.

[49] Yoshida, H., Furusawa, C., and Kaneko, K. (2005) Selection of initial conditions for recursive production of multicellular organisms. *Journal of Theoretical Biology*, **233**, 501–514.

[50] Zuckerkandl, E. (2001) Intrinsically driven changes in gene interaction complexity. I. Growth of regulatory complexes and increase in number of genes. *Journal of Molecular Evolution*, **53**, 539–554.

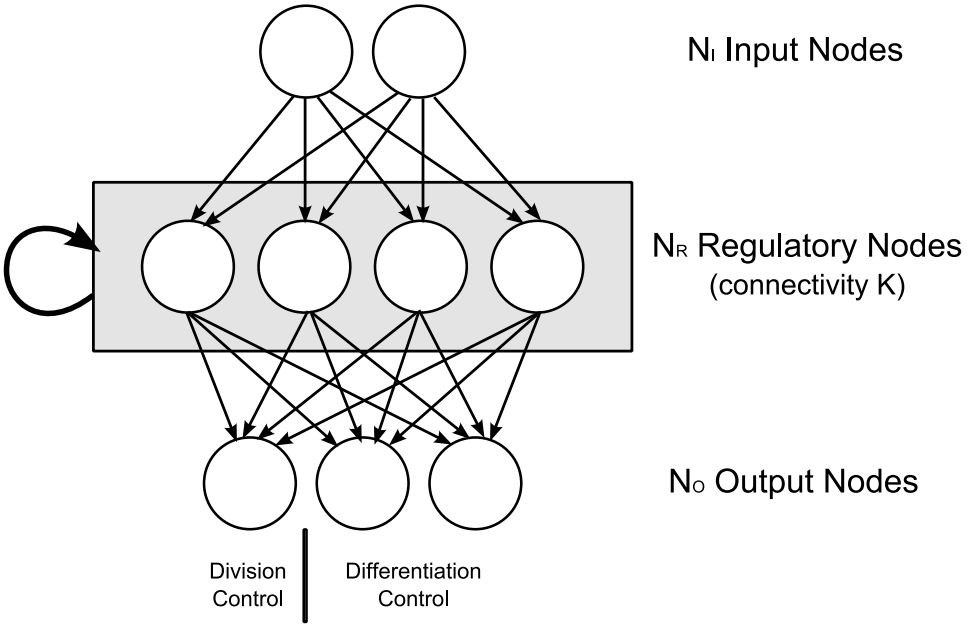

Figure 1: The structure of the network component, showing input, regulatory and output nodes. In this model, the input layer and the regulatory layer are fully connected, the regulatory layer is randomly connected such that each node has inputs from K regulatory nodes (including possible self connections), and the regulatory and output layers are fully connected. The first output node controls the division of a cell; all other output nodes represent possible cell fates. Note that individual recurrent interactions between regulatory nodes (within the gray box) are omitted for clarity.

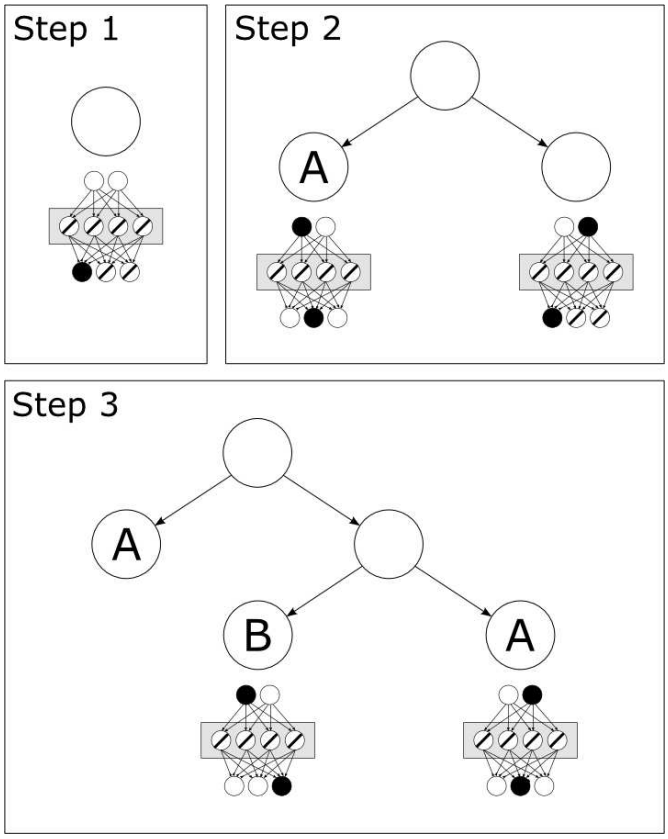

Figure 2: Generation of a cell lineage from network dynamics. Although node activations are actually continuous, they are represented here as binary switches for simplicity—relevant nodes are coloured black (on) or white (off). **Step 1:** The developing system is initialised with a single cell and both input nodes are switched off. The network is updated once. The *Division Control* output node (left) switches on indicating that cell division occurs. **Step 2:** The initial cell has divided and the regulatory network has been copied into each of the two daughter cells. The left input node has been switched on in the left daughter and the right input node has been switched on in the right daughter. The network is updated a second time. The *Division Control* output indicates that the right daughter will divide, but that the left daughter will not. The activation of the first *Differentiation Control* output node (centre) is greater than that of the second (right), therefore the left daughter cell adopts fate A. **Step 3:** The undifferentiated cell divides again. Both daughters differentiate and adopt fates B and A respectively. The 'phenotype' associated with this lineage consists of two cells of type A and one cell of type B.

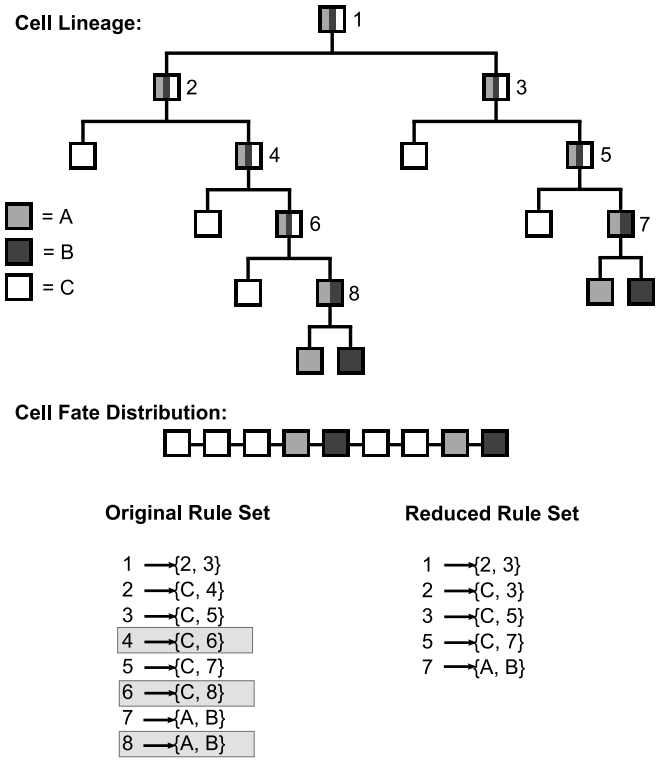

Figure 3: An example application of the deterministic algorithmic complexity metric. First, a cell lineage is transformed into a set of production rules by creating a rule for each division. Redundant rules (highlighted) from this set are then removed. Deterministic algorithmic complexity is measured as the proportion of unique cell divisions (Alg. Cx. $= 5 \div 8 = 0.625$). The non-deterministic algorithmic complexity of this lineage is slightly lower (0.5), because rules 2 and 3, despite producing different sublineage topologies, both map to the same rule: $\{A, B, C\} \rightarrow \{C, \{A, B\}\}$. The size, number of cell types and weighted algorithmic complexity for this lineage are 9, 3 and 4.5 respectively.

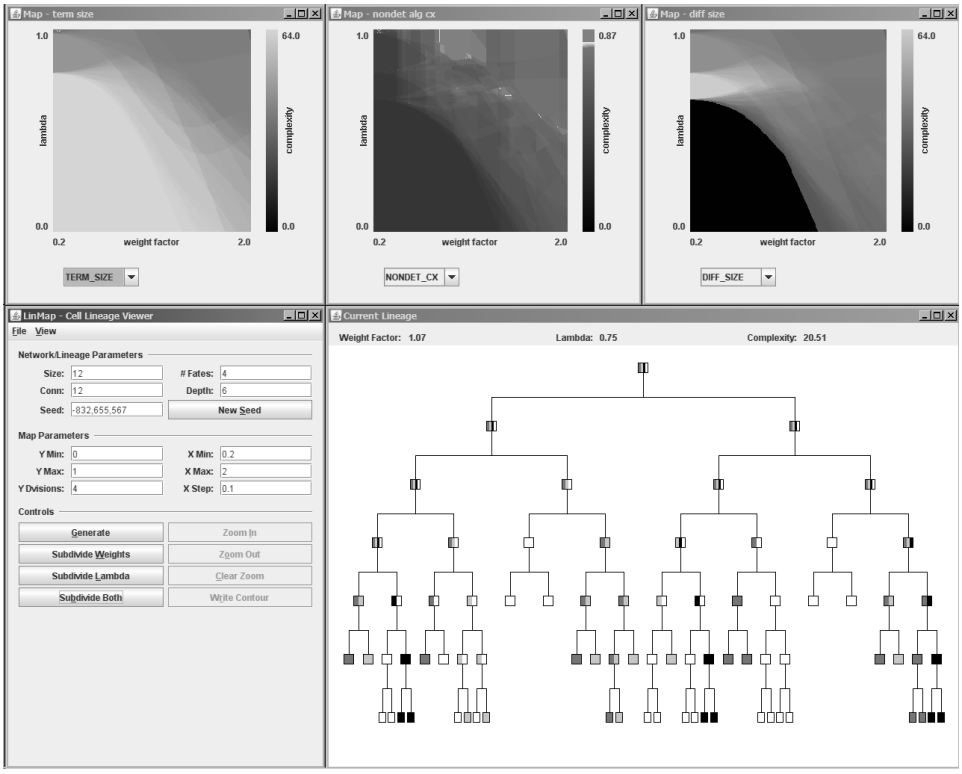

Figure 4: An example screenshot from *LinMap*. The three heat map panels along the top indicate different complexity gradients in the same evolutionary landscape. The cell lineage panel illustrates the currently selected developmental system. Selecting a location on the heat map causes the corresponding cell lineage to be displayed. The control panel in the lower left allows various genotypic and developmental parameters to be adjusted, control of sampling resolution and enlargement, and the saving and loading of generated landscapes.

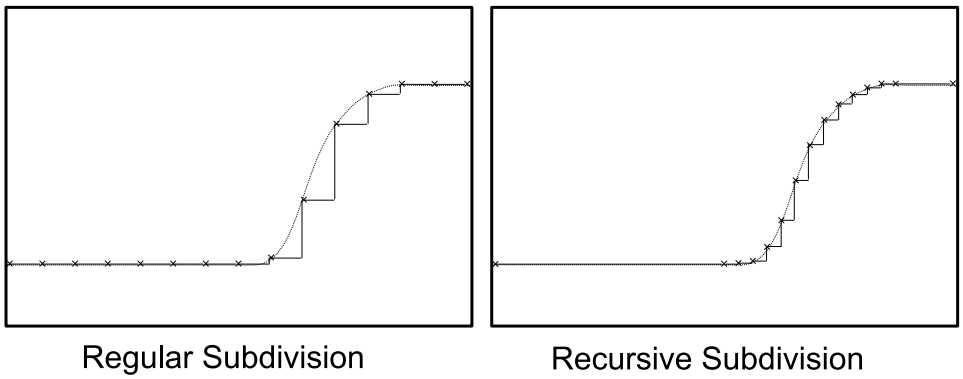

Figure 5: Reconstructed functions generated by regular and recursive subdivision and sampling. In both cases, 15 sample system behaviours have been generated. Recursive subdivision automatically produces increased sampling resolution in the heterogeneous transitional region and a reduced level of sampling in the homogeneous regions.

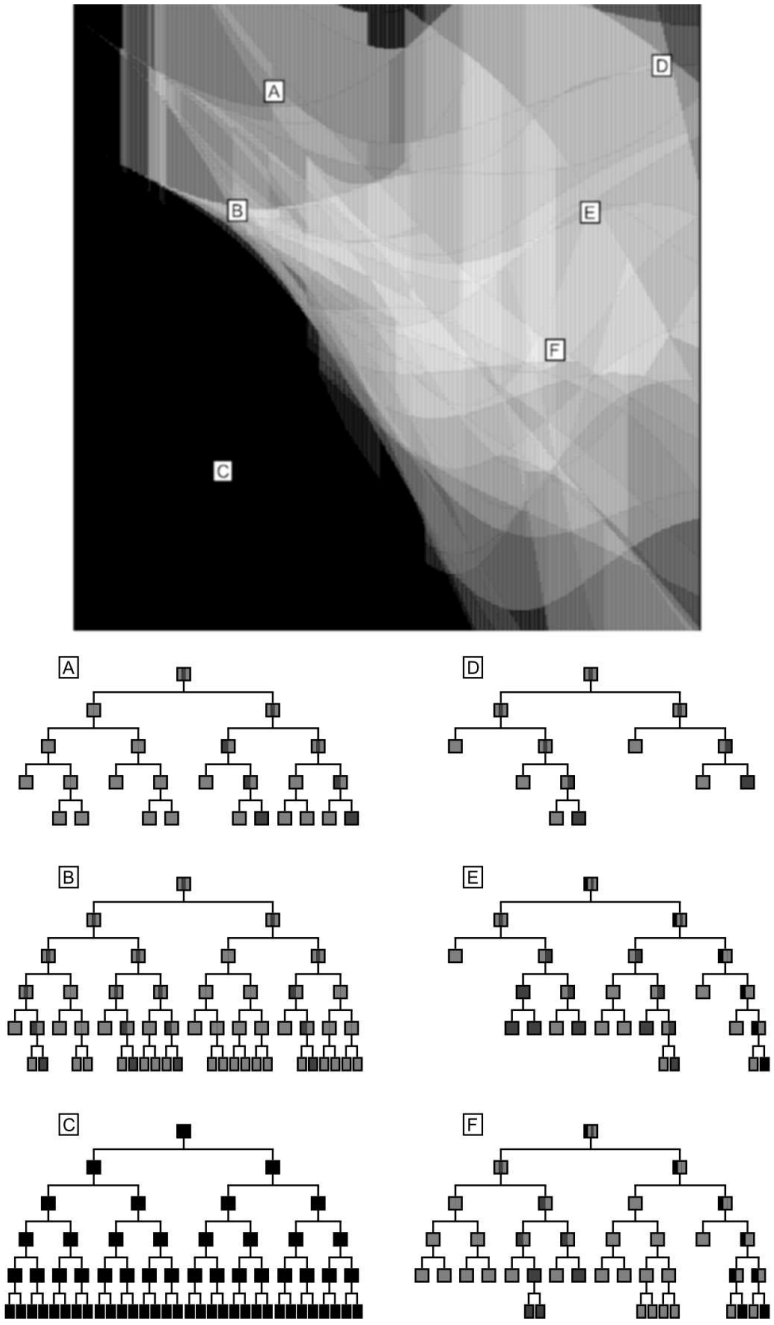

Figure 6: Six cell lineages from different regions of parameter space ($N = 8, K = 8, \lambda = [0, 1.0], W = [0.01, 2.0]$). The heat map is coloured according to weighted complexity. Further details of the lineages are provided in Table 1.

Table 1: Details of cell lineages shown in Figure 6

Lineage	Weight Scale (W)	Division Scale (λ)	Weighted Complexity
A	1.0	0.92	8.67
B	0.5	0.85	11.42
C	0.4	0.40	0.0
D	3.25	0.97	4.67
E	2.9	0.69	12.0
F	2.3	0.55	15.11

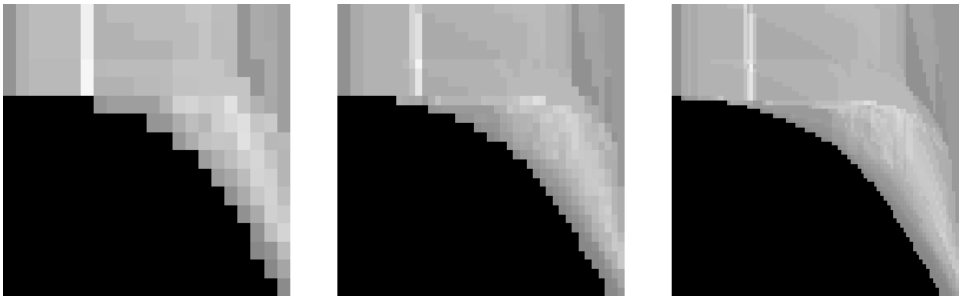

Figure 7: Three instances of the same heat map at increasing levels of resolution ($N = 12, K = 12, \lambda = [0, 1.0], W = [0.1, 2.0]$). The initial heat map (left) provides a rapid means of assessing whether this particular region of parameter space is interesting and worth exploring further. Resolution may then be interactively increased (centre and right) by interpolating between existing parameter values.

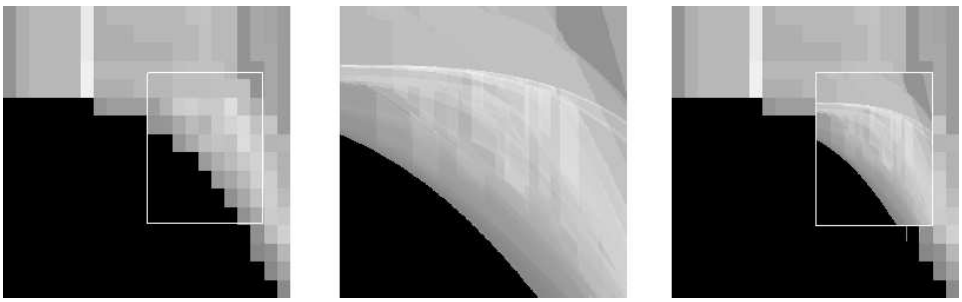

Figure 8: An example of increasing the resolution in a chosen region ($N = 12, K = 12, \lambda = [0, 1.0], W = [0.1, 2.0]$). First, a region of interest is identified and selected (left). The chosen region can then be enlarged and sampled at a higher resolution (centre). It is also possible for regions of more than one resolution to be displayed in a single map: Upon zooming back out to the original map, the enlarged region is highlighted and displayed at the increased resolution (right).

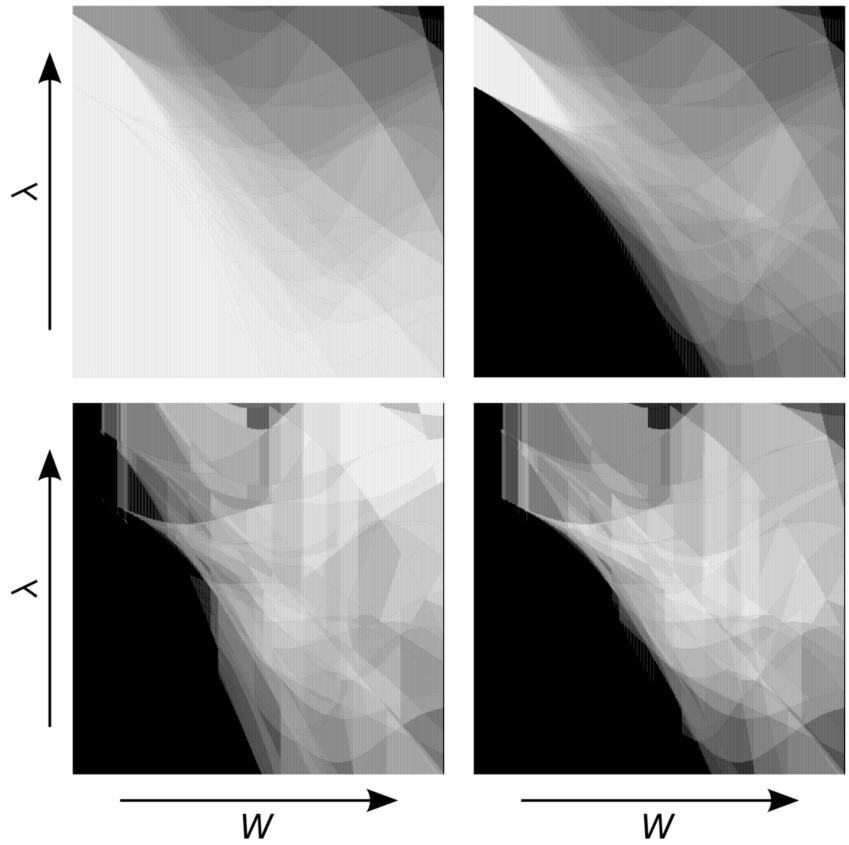

Figure 9: Four Complexity heat maps for the slice of parameter space ($N = 8, K = 8, \lambda = [0, 1.0], W = [0.01, 2.0]$). The four complexity metrics are: number of terminal cells (top left), number of differentiated cells (top right), non-deterministic complexity (bottom left) and weighted complexity (bottom right).

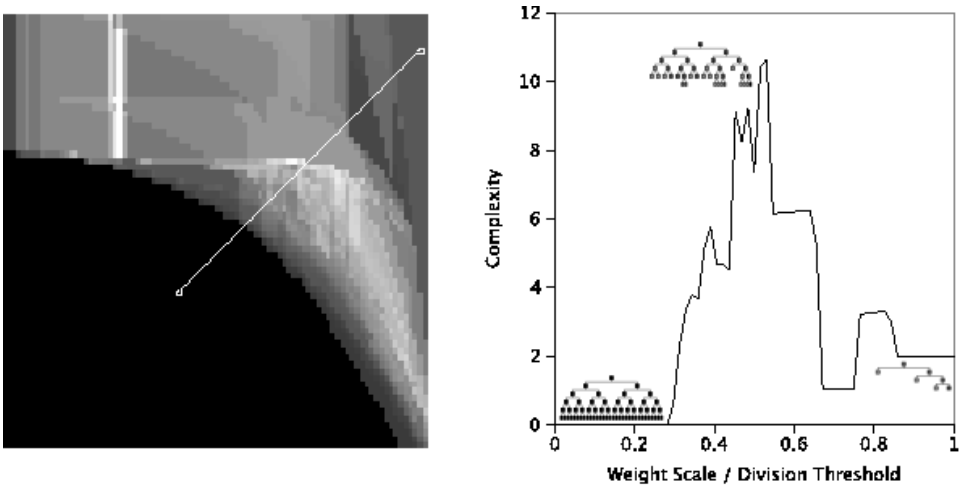

Figure 10: The complexity contour generated by taking a cross-section through space ($N = 12, K = 12, \lambda = [0, 1.0], W = [0.1, 2.0]$). At low values of W and λ, cells proliferate indefinitely and never differentiate. At high values of W and λ, cells divide at most once or twice, if at all. The most complex lineages are found between these two extremes, although the exact location varies depending on the structure of the network.

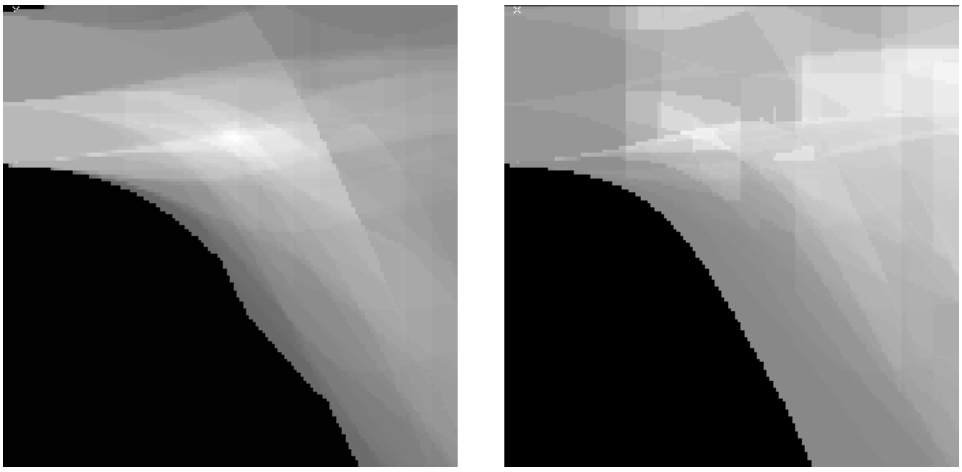

Figure 11: A comparison between the adaptive gradient in an evolutionary landscape (left) and a complexity gradient in the same landscape (right, the metric shown is weighted algorithmic complexity) ($N = 12, K = 12, \lambda = [0, 1.0], W = [0.1, 2.0]$). While the adaptive perspective shows a single peak near the centre of the landscape, the complexity perspective reveals a second plateau of high complexity in the upper right.

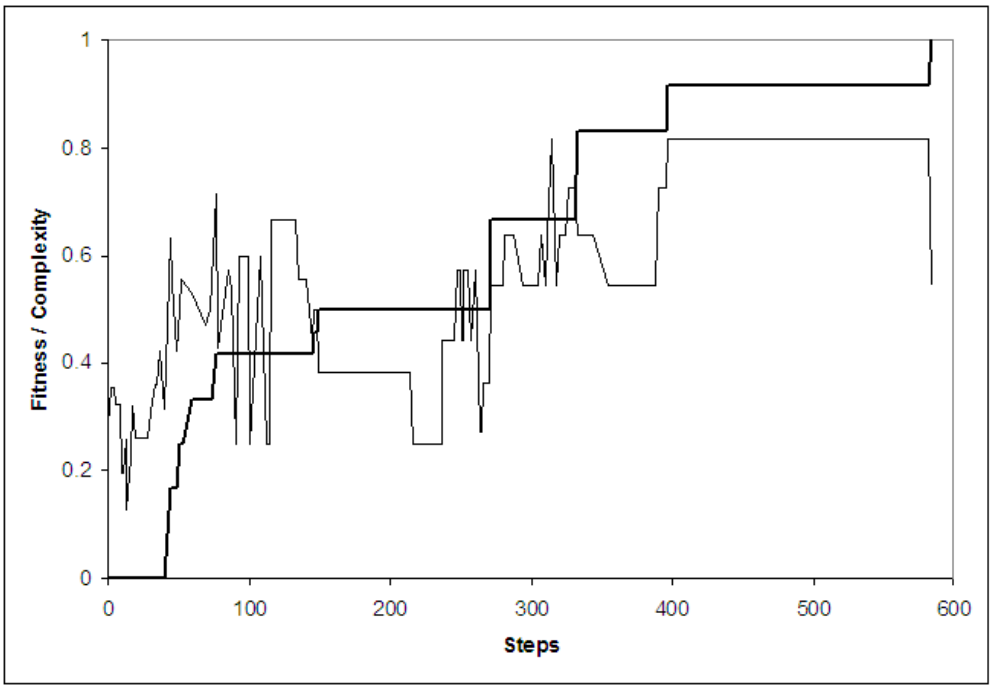

Figure 12: A sample adaptive walk using the network-lineage model. The solid line shows the increase in fitness over time. The dashed line shows the change in deterministic algorithmic complexity.