

The scaling of goals via homeostasis: an evolutionary simulation

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Abstract

Traditional cognitive science focuses on the mental capacities of one individual: memory, perception, learning, anticipation, decision-making, goal-directed behaviour etc... All those mental capacities are taken to be the properties of a fixed, embodied agent. Scale-free cognition or the fact that all cognitive agents are made of cells which are themselves competent cognitive agents by actively proliferating and making decisions is usually relegated to developmental biologists. The assumption behind it corresponds to the mainstream genetic paradigm in which genomes code for specific bodyplans, which changes slowly at evolutionary timescales with a cognitive apparatus bound to an invariant brain and body structure. Regenerative biology and the creation of synthetic organisms *de novo* like xenobots reveal that this traditional paradigm on cognition is incomplete: the plasticity of dynamic morphogenesis of biological forms support diverse types of cognition. It is now essential to begin to investigate the plasticity of both, bodies and minds that highlight the scale-free cognition, or the fact that no agent is an indivisible mind. We propose to investigate this new paradigm of unconventional cognition by modelling scale-free cognition in morphogenetic systems. We developed an evolutionary simulation and showed that anatomical homeostasis is reached (resolution of the french-flag problem) and the tissue learns error-minimization, is robust to perturbation and uses stress as an instructive signal and has evolved allostatic capacities for long-term maintenance of the tissue.

1 Introduction

How cognition of one agent arises from the activity of smaller cognitive sub-agents ? How complex anatomical structure has been formed from unicellular organisms ? How can we control morphogenetic systems composed of several levels of organization from cell to tissue ? All these questions raise the problem of the relation of both body plasticity and mind. Traditional cognitive science focus on the cognitive capacities of one individual: memory, perception, learning, anticipation, decision-making, goal-directed behaviour and make two assumptions. Usually, the body is conceived as a fixed structure and it follows the mainstream paradigm that the genome codes for specific bodyplans. In second, the brain structure is stable and the individuality of the neuronal cells is gone for good. Our primary experience is that of a centralized, coherent Self. Even organisms that undergo metamorphosis, with drastic changes of body, brain, and behavior, like the butterfly are usually studied in their separate phases of life. The transitional states are usually not studied [40].

However, all intelligences are collective intelligences. We are all made of parts that are themselves biological agents. Regenerative biology and the development of new organisms like biobots [33] reveal that studies of cognition in intact bodies are just a narrow slice of a much bigger picture on the multi-scale interface between body and mind. Indeed, cognition is tightly linked to the physical structure of the body, at the anatomical level to the molecular and bioelectric levels that store its information [40]. Second, structure and function are both highly plastic. Cognition continues to function despite important changes to the body/brain and modification of its information at the cellular, molecular, or bioelectric level [41].

In the age of regenerative medicine, body and mind extension via brain-machine interfaces or neural implants, it seems essential to understand how mind and body co-evolve [87], to explore the origin of multicellularity and the scaling of basal cognition of individual cells into larger organisms. Understanding the

“software” or algorithms allowing cells to make goal-directed decision-making and the transition to larger selves is still poorly understood [40, 55]. In order to integrate and advance the fields of developmental/evolutionary biology, synthetic bioengineering, machine learning, and cognitive science, it is fundamental to begin to develop computational frameworks for asking how cognitive capacities arise in agents made of parts, and how these emergent subjects of cognition scale up during evolution.

In the scale-free cognition framework [40], we rely on ideas from control and information theory to identify multi-scale information processing principles. Earlier studies showed that individuals can be outperformed by collectives [96], and that their overall performance is dependent on several characteristics including their organisational or network structures [54], the information aggregation and communication system among their individuals, and the diversity between their members [32]. These studies mainly focused on human networks and the associated wisdom of the crowd. We focus here on morphogenetic systems.

In this article, we present an evolutionary multi-agent model of scale-free cognition. By testing specific hypotheses in this novel simulated virtual evolution system, we show that from the interaction of proto-cognitive agent and the ability to reduce stress emerges several high-level component of cognition like error-minimization to reach an anatomical goal, multi-scale homeostasis, robustness to perturbation and the evolved system uses stress as an instructive signal and that stress is needed for long-term survival.

2 Scale-free cognition

2.1 Basal cognition or neuroscience beyond neurons

All cognitive agents are made of parts and in the case of living beings, those parts are cells which are themselves competent cognitive agents [23]. Indeed, the emerging field of Basal (Minimal or Proto-Cognitive) Cognition provides many examples of cognition throughout a wide range of non-neural systems. This field tracks the history of learning and decision-making processes, starting from the problem-solving capacities of cellular and sub-cellular forms [49]. Indeed, even single cells that are themselves competent agents can exhibit adaptive behavior and can also be divided into even smaller sub-cellular agents because of their protein, gene-regulatory or cytoplasmic, or metabolic networks that can show similar computational properties than neural networks [3, 12, 19, 24, 75, 81, 84, 93]. All these features we can find in unicellulars was also presaging their multicellular behaviour, indeed, many features we can observe during anatomical control including plasticity [35], differentiation [82], regenerative repair [61], were already existing in ancient and unicellular life forms. Single cells can alter their motility, or metabolism in response to changing environmental conditions and they are very competent at keeping their homeostasis by managing their morphology, behavior, and physiology as needed for survival. Many organisms, including bacteria [50], biofilms [97], protozoa [21], plants [25] or somatic cells [66], have been shown to exhibit brain-like behaviour such as memory, decision-making, learning, anticipation or prediction. These aneural organisms have even been used as model organisms to understand neural function [36, 62, 78].

Body patterning and cognition also seems to share a common origin. Indeed, several tissues exhibit evidence of learning and basal cognition, including cardiac [98], bone [90] and pancreatic tissues [27]. These findings are very old [9, 45] and the molecular apparatus of higher cognition (ion channels, neurotransmitters and synaptic mechanisms) was already present in our unicellular ancestors [2]. Brains and neurons have been speed-optimized by evolution from other cell types [88]. Somatic cells did not lose their cognitive repertoire and computational capabilities during their transitions to multicellularity and in becoming part of metazoan swarms (bodies): they scaled them to pursue larger anatomical goals [68]. Development/regeneration and cognition are both very similar. This is not simply an emergent result of hardwired processes but a very plastic, context-dependent system that achieves invariant patterning outcomes under uncertainty. Several examples can highlight this behavior including regulative development (when an early embryo is cut in half, the end result is two normal bodies), the experimentally induced picasso head (in tadpoles when craniofacial organs are in abnormal positions, they still make largely normal frog faces) [72, 92]. Similarly in regeneration, limbs are remodeled from grafts of salamander tails on the flank [22].

Several correspondences can also be done with cognitive science concepts between the control of morphogenetic systems and adaptive behaviour [67–69].

The case of memory is also particularly relevant. Memory spans different levels of organization and are not only located in neural networks. For example, moths can remember learned information when they were caterpillars [6]. Memory can also be transferred from a single egg cell to the resulting multi-cellular system after development [30]. Therefore, memories are maintained across different levels of organization and even after drastic brain remodelling [7]. Memories can also move across tissues. For example, planarian flatworms can be trained to learn different tasks. Memory can be remembered after regeneration of trained flatworms which heads have been amputated [5]. This suggests that memories can be stored outside the head and the nervous system. Memories can also be moved across individuals. Memory transplants have been accomplished with cells, RNA or protein extracts form one animal to another [4, 59, 91].

2.2 Developmental bioelectricity

The similarities between body patterning and goal-directed behaviour are not only functional but can also be found at the molecular level. Brains act in a 3D space and navigate this space, bioelectrical signaling in a collective of cells will control the navigation through anatomical morphospace during embryogenesis, repair, and regeneration [55, 57, 85]. Developmental bioelectricity can be defined as "the ubiquitous exchange of slowly changing ion-based voltage signals within and among cell" [40]. All cells are electrically active, and during evolution, neurons evolved from pre-neuronal cells that were already using the benefits of ionic signaling for computation. Indeed, electrical networks are extremely convenient for memory and computation, a fact that was exploited by our technology but discovered very early in evolution. Bioelectrical networks for control can be found in bacterial biofilms [46], multi-cellular regenerative systems [55, 67] and in synthetic excitable tissues [58].

Transcriptional cascades can be modified by developmental bioelectricity dynamics, it controls axial patterning, organ determination, size control, and the behavior of individual cells [39, 43, 86]. Drugs that modify cognitive capacities can also be important teratogens [31, 73], while anesthetics alters regeneration [11, 89]. The isomorphism between morphological and behavioral processes is a continuum, the ancestral basis for psychology could be found in membrane excitability [17].

In the overall, the cognitive lens on non-neural systems, and recent results on the molecular genomics of pathways involved in learning and memory in brains, are consistent with the fact that brains and aneural systems share not only molecular components but also computational functions, allowing adaptive behavior and broadening our understanding of the substrates of cognition [1, 49, 52, 83]. Thus, it is likely that there is a continuum between computational processes that are widely conserved at both the functional and mechanism (molecular) levels and higher cognitive capacities. From the capabilities of single cells arised the information processing and spatio-temporal integration needed to development of complex anatomies and regeneration, which evolution then scaled up complex nervous systems producing a large variety of behaviours that underlie familiar examples of Selves. These cognitive lens on developmental biology blurs the frontiers between mind and body.

2.3 Multi-scale competency and evolution

As we mentioned it, each intelligence is a collective intelligence. This also means that any intelligence integrates multi-scale homeostatic loops. Indeed, the subunits making up each level of organization are themselves homeostatic agents. This multi-scale homeostasis implies multi-scale error-correction loops.

Each cell wants to stay alive and as a collective, morphogenetic systems act to reach the target morphology, applying anatomical homeostasis. These are goal-directed (in the cybernetic sense) systems as they can reach a specific target morphology state despite perturbations or changes in local/starting conditions. Anatomical homeostasis can be found in regeneration and development. This multi-scale homeostasis/autonomy or multi-scale goal-directed behavior has several implications for evolution.

First, it will increase the smoothness of the fitness landscape. Goal-directed systems can handle errors or deviations from the steady-states, therefore mutations that would have been detrimental in a biological system that follow a hardwired (genetically-specified) set of steps are neutral because of the competency of the goal-directed morphogenetic subsystems. This multi-scale competency is another layer of defense against mutations negative effects.

Second, MCA reduces the fact that most mutations have multiple effects or pleiotropy [10]. Biological systems integrating MCA will have less negative effects of mutations while taking profits of positive effects of the same mutation via local adjustments to new changes that reduce the downside effects. This is particularly important, indeed evolution does not have to solve the very difficult search problem of how to improve feature X in an orthogonal manner that won't affect other features which are already efficient.

Third, MCA allows biological systems to also exploit opportunities. The MCA allows a biological Baldwin effect. Useful behaviors that have been learned can be hardwired by evolution. Or in other words, the compensatory nature of MCA with mutations gives time for new another mutations to take place that hardwire the compensatory changes that had to be applied before.

Fourth, the relationship between genotype and anatomical phenotype is more linear because of MCA [48,64], and in second it improves controllability [26,74]. By using a computational layer to encode target morphology, goal-directed systems does not need to solve the difficult and ill-posed inverse problem. Bioelectric pattern memories (such as the voltage distribution that tells wild-type planarian cells whether to build 1 head or 2) is such a kind of encoded goal in the top-down layer. It is easier for evolution to change the homeostatic setpoints in this layer, such as the electric face prepatter [92], than to make genetically hard-wired changes at the micro level. This allows to use the same molecular machinery to achieve very different (anatomical) goals. The MCA allows for an important complexity reduction of the navigation in physiological/anatomical space [42].

2.4 Limits of current models of the mind/body co-evolution

Understanding the “software” or algorithms allowing cells to make goal-directed decision-making and the transition to larger selves is still poorly understood [40,55]. Different works have been developed to investigate the multi-scale interface of body and mind, notably with “morphological computation” in Artificial Life and Soft Evolutionary Robotics [8,13,65,70,71]. These studies model and exploit the fact that brains, like other developing organs, are not hardwired but are able to ascertain the structure of the body and adjust their functional programs accordingly. Similarly to scale-free cognition, morphological computation is about connecting the body and cognition. However, even if they investigate mind and body co-evolution, they did not take into account basal cognition and that the constituting elements of the bodies are themselves cognitive agents. It is now essential to fill this critical knowledge gap.

3 The evolutionary system: from single-cell homeostasis to anatomical homeostasis

3.1 The general scheme of the evolutionary simulation

General system. We developed an evolutionary simulation system composed of cells on a 2D grid that forms a tissue. The system is an evolutionary, agent-based, spatialized model and have two main time-scale loops. The outside loop is an evolutionary (phylogenetic) long time-scale where genomes are mutated and agents are selected. The inner loop is a short time-scale ontogenetic loop which constructs each agent from its genome, simulates the development, and then tests the phenotype for fitness. Each agent is one cell, which has a genome that encodes some very simple metabolic processes (interacts with the other cells).

We integrated a homeostatic loop that enables it to optimize for the levels of some particular chemical (we are not trying to model the origin of life itself – we start with cells that can keep themselves alive via basic homeostasis, e.g. need to keep a level of energy superior to 0). We allow cells to attach to each other using gap junctions, to send intracellular signals and spread metabolites (these signals, and their properties – like speed of spread, ability to propagate gap junctions, gating properties of gap junctions based on signal and cell state, etc., are all coded by genomes through evolution). Cells don't know from which neighbours they receive molecules, in other words, we encoded the informational owner wiping property.

The general approach is related to cellular neural networks [16] and more particularly to the growing neural cellular automata [60] where each cell have access to the states of their neighbours and contains a neural network to drive actions. In our case, the number of cells is fixed and does not grow with time. The

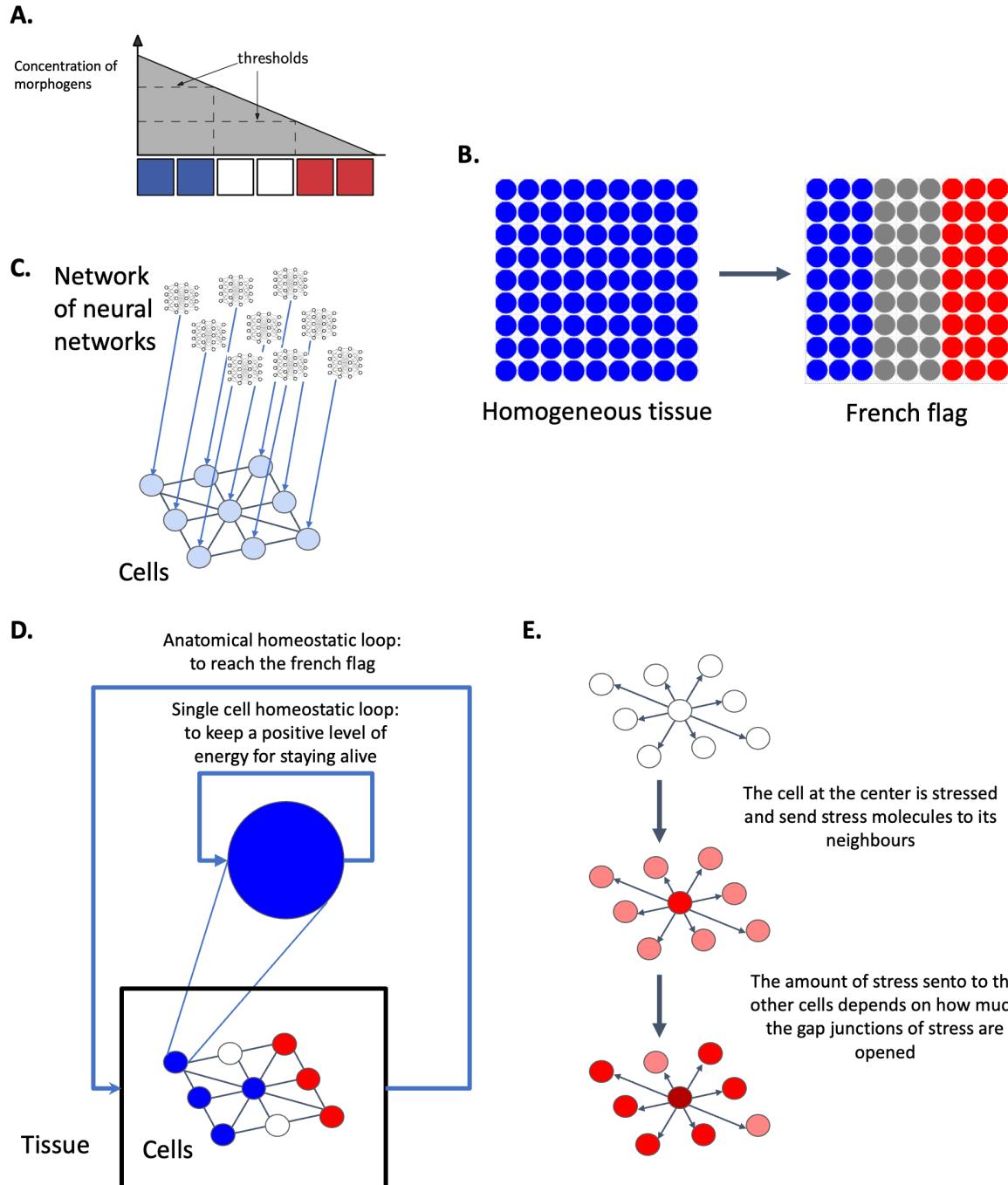


Figure 1: **A.** Description of the French flag problem. The concentration of morphogen triggers a particular gene expression leading to 3 different states: blue, white or red. **B.** Description of the virtual environment. At left, the tissue is randomly initialized. At right, the tissue reached the French flag. **C.** Each cell is connected to its neighbours and can exchange molecules by gap junctions. The behavior of each cell is controlled by its neural network. **D.** Two homeostatic loops have been implemented: one homeostatic single-cell loop, each cell has to maintain a positive level of energy for staying alive, and an anatomical homeostatic loop, all cells receive energy depending on how much the collective (or the tissue) reach the anatomical goal.

Algorithm 1 Pseudo-code of agent.step()

```
1: procedure AGENT.STEP((self, Input)                                ▷ Memory at t-1
2:   self.energy(t-1) = self.energy
3:   self.stress(t-1) = self.stress
4:   output = self.communication()
5:   self.reward = Reward(self.position)
6:   self.energy += self.reward - 0.8
7:   UpdateStress()
8:   if self.energy  $\leq 0$  then
9:     self.death()
```

Algorithm 2 Pseudo-code of agent.communication()

```
1: procedure AGENT.COMMUNICATION((self, OutputANNMol, OutputANNStress)
2:   for i in directions do:
3:     for neighbour in neighbours do
4:       if self.GJ[directions[i]]*neighbour.GJ[i] > 0 then:      ▷ If the corresponding GJs are opened
5:         SendMoleculeNeighbour(i, OutputANNMol)
6:         neighbour.molecules += OutputANNMol*self.GJ[i]*neighbour.GJ[i]
7:         self.molecules -= OutputANNMol*self.GJ[i]*neighbour.GJ[i]
8:         neighbour.UpdateState()
9:     for neighbour in neighbours do
10:    if self.GJStress > 0 then
11:      neighbour.stress+=OutputANNStress*((self.GJStress * neighbour.GJStress))
12:      neighbour.stress-=OutputANNStress*((self.GJStress * neighbour.GJStress))
13:    if self.GJStress > 0 then
14:      self.stress+=OutputANNStress
15:      self.stress-=OutputANNAnxiolyticss
16:    self.UpdateState()                                         ▷ The level of molecules determine the state
```

tissue has borders, meaning that this is not a torus.

Cell description. Each cell integrates an artificial neural network that controls the opening and closure of gap junctions in 4 directions: up, down, left, right. Each cell has one type of molecules that can go through the gap junctions depending on their openings and that will trigger different kind of genomic expression leading to three different cell states: blue, white, or red (see Figure 11). These states depend on the level of molecules inside the cells and we imposed an energy cost on state change. They also have a stress molecule and its counterpart that can be send to their neighbours.

The stress system is completely evolved as the amount of molecules to be transferred is chosen by the embedded neural network. The neural network, which is identical for each cell, has 6 inputs: the internal levels of molecules, energy, stress and the internal state, the perception of the collective (how big is the collective in the tissue, the collective being cells of the same state connected by opened gap junctions) and the perception of the cell neighborhood (geometrical frustration or how much the cell is similar to the neighbors). The 4 outputs of the ANN are: the amounts of molecules to send to neighbours, the amount of stress molecules to send to neighbours and to be applied to the cell itself, the amount of anxiolytics to send to neighbours and to be applied to the cell itself, the opening of gap junctions in the 4 directions.

Learning task. The learning task is the French flag problem [94, 95] (see Figure 1B). Originally developed by Wolpert, it is his first comprehensive discussion about how spatial gradients might specify patterns of cell fates in a tissue. In this simulation, the spatial gradient that determine the cell state is the amount of molecules inside the cells. The two thresholds are 5 and 10.

From local to anatomical homeostasis. There is a scaling of goals when we tie the single-cell and the anatomical homeostatic loops. Each cell has one goal, to survive and it corresponds to be in the appropriate state in order to receive energy (with the other members of the collective), e.g navigating in a 1-dimensional metabolic continuous space but by discretizing it, the problem space solutions has three instances (blue, red, white). In the other hand, the collective/tissue has a morphogenetic goal, the French flag and it is navigating a problem space of 3^n instances (n: number of cells).

Each cell receives an amount of energy at each step which is proportional to how much its corresponding sub-collective corresponding to one stripe is reaching appropriately the anatomical goal. Each cell has therefore a reward uncertainty as it is not depending on its own behavior but on the (sub)collective behavior, or in other words, the reward also depends on the decision of distant cells. Therefore, the environment of the cells is of high uncertainty. In a sense, this scheme can be understood as a problem of multi-agent reinforcement learning under reward uncertainty.

Goal-directed systems have the ability to focus on relevant information and ignore distractors. To do so, they rely on selective attention and/or interference suppression. Selective attention would rely on top-down biasing mechanisms as proposed by Desimone and Duncan [20]. In our case, the top-down biasing mechanism is represented by the reward in energy that ties the two homeostatic levels.

Stress system. Stress can be defined as three related concepts — the external and internal stimuli that cause stress, the emergency physiological and behavioral responses activated in response to those stimuli, and the pathological consequences of over-stimulation of the emergency responses [38, 77]. In our case, what we named the stress system is another communication system that can diffuse a stress molecule through the tissue. This is related to the physiological response to one stimulus. When the ANN makes the decision to send stress, each cell will diffuse a molecule to its neighbours that will increase equally the stress level of the stressed cell itself and its neighbours. The ANN can also send an anxiolytics molecule that will be sent in the same manner to the neighbours that will decrease the cell stress levels (see Figure 1E). Evolution can choose or not to use this communication system.

Evolutionary algorithm. We use ES-Hyperneat [76] as the evolutionary learning algorithm. The fitness function is the percentage of good states the tissue has:

$$\text{fitness} = (\text{number of good states}/\text{number of cells}) * 100 \quad (1)$$

The whole system has been coded in Python programming language, using the agent-based modeling framework Mesa [53] and is freely available on <https://github.com/LPiOL/scalefreecognition>.

3.2 Information-theoretic analysis

Information theory [51] is a very useful tool to understand the information dynamics in complex systems. We used two information-theoretic measures to analyze the information dynamics of the results: active information storage [47] and transfer entropy [79].

Active information storage. The amount of information in the past of one agent that is relevant to predict its future state is the information storage. In this article, we will focus on the active information storage (AIS) component, which is the stored information that is currently in use in computing the next state of the agent [47]. Formally, the AIS of an agent Q is defined as the local (or unaveraged) mutual information between its semi-infinite past $q_n^{(k)}$ as $k \rightarrow \infty$ and its next state q_{n+1} at time step $n + 1$:

$$a_Q(n+1) = \lim_{k \rightarrow \infty} \log_2 \frac{p(q_n^{(k)}, q_{n+1})}{p(q_n^{(k)})p(q_{n+1})} \quad (2)$$

$a_Q(n, k)$ represents an approximation of history length k . The average over time (or equivalently weighted by the distribution of $(q_n^{(k)}, q_{n+1})$): $A_Q(k) = \langle a_Q(n, k) \rangle$. With AIS, an agent can store information regardless of whether it is causally connected with itself [47].

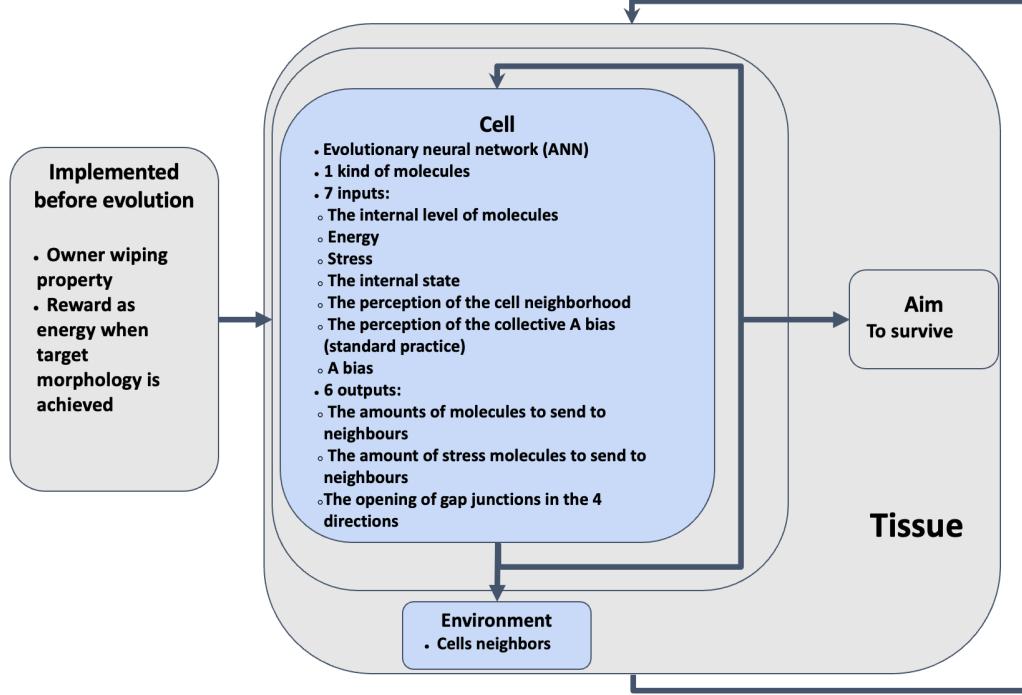


Figure 2: General scheme. The system is an evolutionary, agent-based, spatialized model and have two main time-scale homeostatic loops. The outside loop is an evolutionary (phylogenetic) long time-scale where genomes are mutated and agents are selected. The inner loop is a short time-scale ontogenetic loop which constructs each agent from its genome, simulates the development, and then tests the phenotype for fitness. Each agent is one cell and embed an artificial neural network that controls the cell behavior and signalling with the cell neighbours.

In this article, we compute the local active information storage over the states of the cells.

Transfer entropy. Transfer entropy is the information provided by the agent source about the destination's next state that was not contained in the past of the destination agent. In this article, we use the local transfer entropy introduced by Lizier. The local transfer entropy from a source agent Z to a destination agent Q is the local mutual information between the previous state of the source z_n and the next state of the destination agent q_{n+1} , conditioned on the semi-infinite past of the destination $q_n^{(k)}$ (as $k \rightarrow \infty$):

$$t_{Z \rightarrow Q}(n+1) = \lim_{k \rightarrow \infty} \log_2 \frac{p(q_{n+1}|q_n^{(k)}, z_n)}{p(q_{n+1}|q_n^{(k)})} \quad (3)$$

Transfer entropy $T_Q(n, k)$ is the (time or distribution) average: $T_Q(k) = \langle t_Q(n, k) \rangle$ and $t_Q(n, k)$ represents an approximation of history length k . When mutual information measures correlation only, the transfer entropy measures a directed and dynamic flow of information in the network of agents.

We computed for the analysis of our information dynamics different kind of transfer entropy: the transfer entropy from stress to the states of the neighbours (blue, white, red). We compute the transfer entropy pairwise with all the neighbours of one cell and average it. We also computed the transfer entropy from the stress to the internal state of the cell. Finally, we defined the transfer entropy from one stripe to another as the sum of all transfer entropy of one cell of one stripe to all cells of the other stripes computed pairwised on the time series of the internal states. Formally, we define the average transfer entropy over a range of source-destination pairs in the spatial locations of different stripes S_1 and S_2 :

$$t_{S_1 \rightarrow S_2} = \sum_{Z_i \in S_1} \sum_{Q_j \in S_2} t_{Z_i \rightarrow Q_j} \quad (4)$$

The transfer entropy defined for specific subsets of tissue processes is useful in considering distributed communications across agents with specific roles.

4 Results

In the next subsections, we present different results we obtained with the scheme we defined above. The results are part of different simulations and we precise when we applied different experiments on the same simulation.

4.1 Error minimization for reaching the target morphology

In this simulation, the tissue has to resolve the French flag problem. Each cell receives energy according to its location on the tissue and its reward is proportional to how much the other cells of one stripe of the French flag are resolving the French flag problem. All cells start in the blue states and have to cooperate to be in the appropriate state and send the appropriate signals to its neighbours. The fitness function is computed at 90 steps. We can observe that the ANN inside each cell evolved by using the stress system as another communication channel (see Figure 3). First, the locations of the grey and red stripes are starting to be stressed and then it is mainly the red stripe. All gap junctions are opened, the upper and right gap junctions are fully opened and the low and left gap junctions are half opened. It means on our scheme as the gap junctions are like weighting the flow of molecules that the stream from right to left and the opposite are equal as for the streams in the up-down plane. The diffusion of molecules from left to right happens as the cells of the blue stripes act as reservoir of molecules and the cells decide during the dynamics to send more molecules or less to their neighbours alternatively.

The stress is increasing at 55 steps and it is decreasing while the French flag problem is resolved. At 90 steps, the tissue reaches 95,1% of the French flag target morphology. There are 4 blue cells in the red stripe. The cells learned to minimize error during their lifetime in order to stay alive and by doing so the tissue reaches the target morphology and resolve the French flag problem.

4.2 Robustness to perturbation

The cells seemed to have learned error-minimization in order to reach the anatomical goal, here the French flag in 90 steps. But do they follow a hardwired plan or do we have a multi-scale homeostasis that allow the tissue to reach the target morphology in case of perturbations ?

In this simulation, we artificially perturbed the tissue by changing at 110 steps the states of the last two columns of the red stripe to grey cells. The cells have never been evolved on more than 90 steps. However, after the perturbation at 110 steps, the tissue corrects the red stripe in 10 steps and a few blue cells appear in the grey stripe (see Figure 4). The stress increases and decreases in parallel to the perturbation and its resolution similarly to the resolution of the French flag problem. It is similar to what we can observe in biological systems. The system tries to get rid of the blue cells left in the grey stripe. At 200 steps, the tissue reached 95,1% of the French flag. The dynamics of the gap junctions stay the same as during the French flag resolution as seen in Figure 3 without perturbation. It seems that the cells do not follow a specific hard-wired plan to reach the anatomical goal and that multi-scale homeostasis is applied. Without it, it would not be possible to recover from the perturbation.

4.3 Stress: different use-cases

4.3.1 Stress is instructive

In the last simulations and experiments, we observed that the cells used the stress system after evolution. Is this stress really instructive ? To answer this question, we artificially added anxiolytics to the tissue that

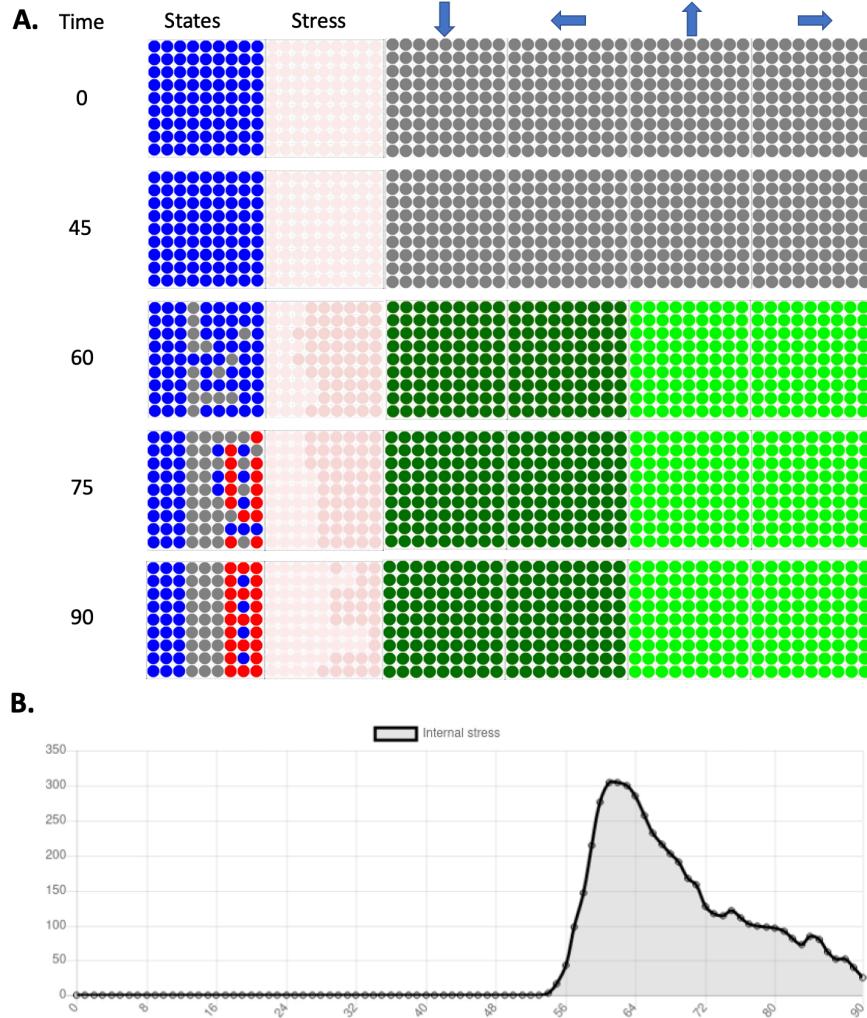


Figure 3: **A.** Time-lapses of the dynamics of resolution of the French flag problem by the tissue. The dynamics of the tissue is represented by 5 timelapses taken at 0, 30, 60, 90 and 120 steps of the dynamics of the tissue. The first column represent these steps, the second column the states of the cells in the tissue (blue, grey, red). The third column represent the use of the stress system on a red scale, the darker the red, more the system is stressed. The last four columns represent the opening in the gap junctions in 4 directions: low, left, up and right. The cells resolve the French flag almost entirely at the 90 steps. The stress system is used all along the resolution of the French flag problem. **B.** Amount of stress inside the tissue during the resolution of the French flag.

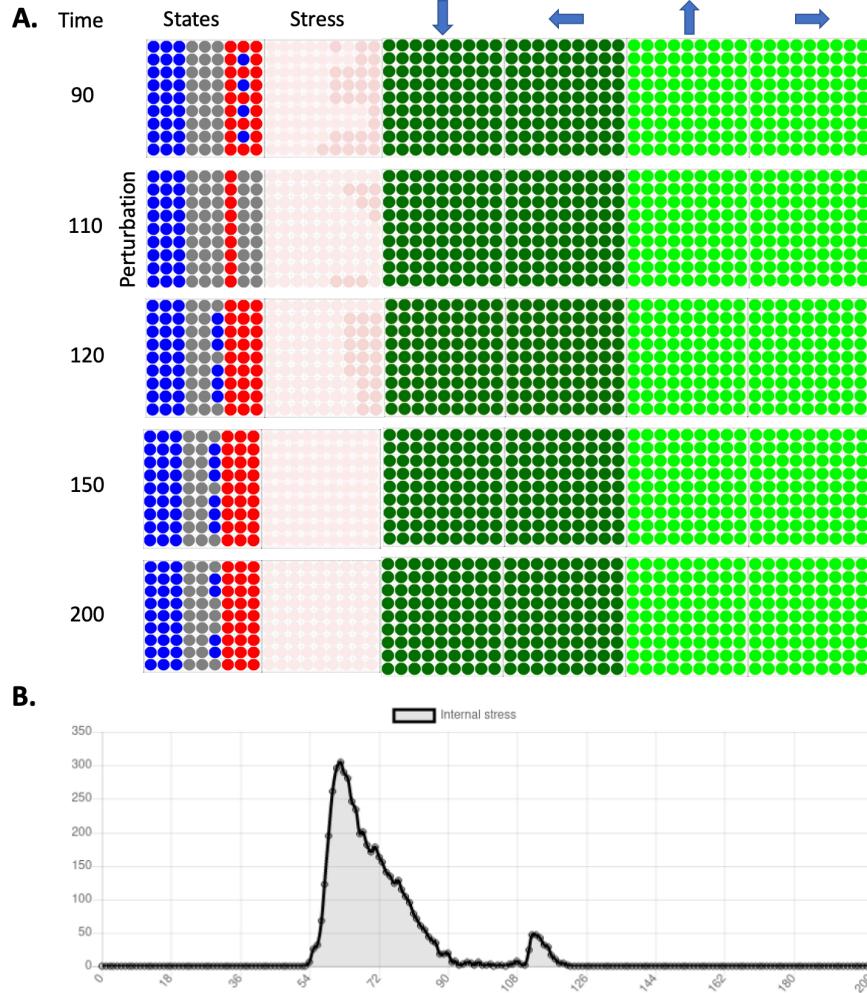


Figure 4: **A.** Time-lapses of the dynamics of resolution of the French flag problem by the tissue. The dynamics of the tissue is represented by 5 timelapses taken at 0, 30, 60, 90 and 120 steps of the dynamics of the tissue. The first column represent these steps, the second column the states of the cells in the tissue (blue, grey, red). The third column represent the use of the stress system on a red scale, the darker the red, more the system is stressed. The last four columns represent the opening in the gap junctions in 4 directions: low, left, up and right. The cells resolve the French flag almost entirely at the 200 steps after the artificial perturbation of the tissue at 110 steps. The stress system is used all along the resolution of the French flag problem and it increases and decreases with the resolution of the perturbation. **B.** Amount of stress inside the tissue during the resolution of the French flag.

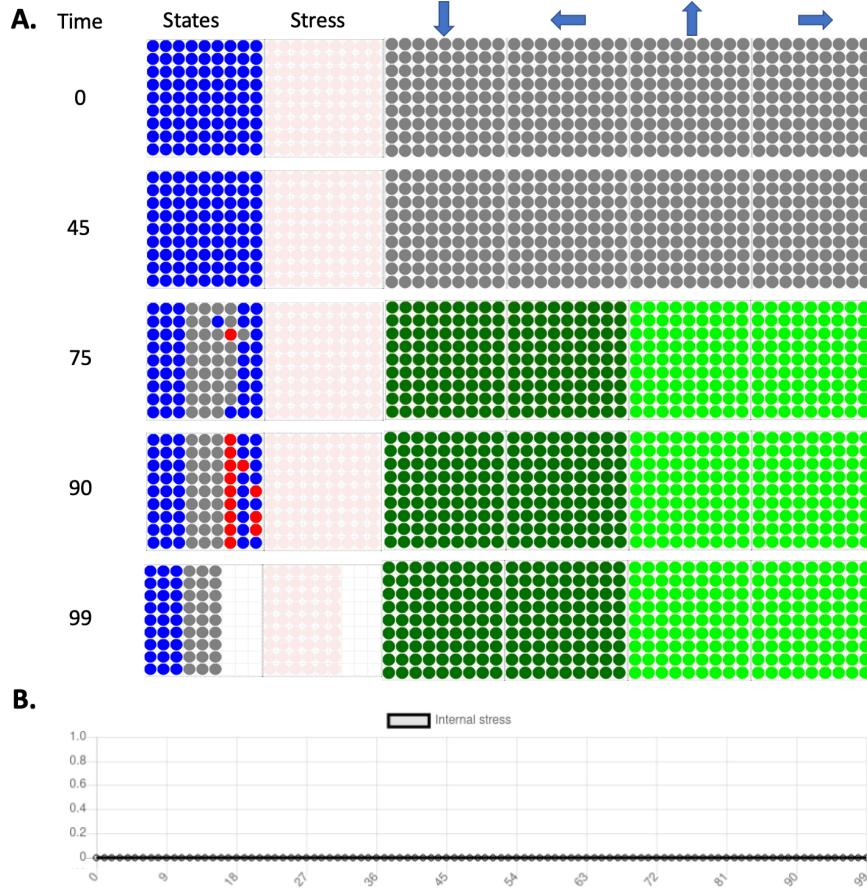


Figure 5: **A.** Time-lapses of the dynamics of resolution of the French flag problem by the tissue. The dynamics of the tissue is represented by 5 timelapses taken at 0, 30, 60, 90 and 120 steps of the dynamics of the tissue. The first column represent these steps, the second column the states of the cells in the tissue (blue, grey, red). The third column represent the use of the stress system on a red scale, the darker the red, more the system is stressed. The last four columns represent the opening in the gap junctions in 4 directions: low, left, up and right. The cells resolve the French flag almost entirely at the 90 steps. The stress system is used all along the resolution of the French flag problem. **B.** Amount of stress inside the tissue during the resolution of the French flag.

reduce the level of stress to 0 during the whole lifetime of the cells (see Figure 5).

The dynamics of the gap junctions is similar to the dynamics when stress is used (see Figure 3). However, we observe that at 90 steps, the tissue reached only 82,8% of the target morphology. And at 99 steps, the collective of cells corresponding to the location of the red stripe of the French flag dies. Anatomical homeostasis is not reached nor maintained during the lifetime of the cells without stress. While when the stress is used by the cells, at 90 steps, the tissue reach 95,1% of the French flag target morphology and it maintains the morphology after this step.

In this case study, stress seems instructive and allow the tissue to reach the target morphology with a high percentage and to maintain this morphology after the 90 steps on which the ANN of the cells have been evolved.

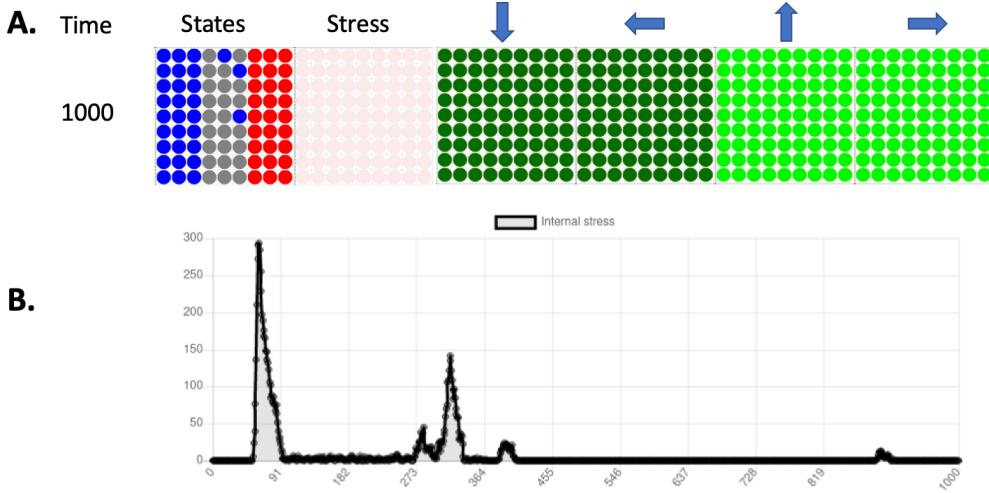


Figure 6: Long-term survival of the tissue and stress. The simulation has been running during 1000 steps. **A.** State of the tissue at 1000 steps. The first column represent these steps, the second column the states of the cells in the tissue (blue, grey, red). The third column represent the use of the stress system on a red scale, the darker the red, more the system is stressed. The last four columns represent the opening in the gap junctions in 4 directions: low, left, up and right. **B.** Amount of stress inside the tissue during its lifetime.

4.3.2 Stress is needed for long-term survival

We discovered that without stress, the tissue could not maintain its morphology after 99 steps. But is stress needed for long-term survival ? In this simulation, we still used the same evolved cells and ran the simulation until 1000 steps. We observe that the tissue maintains its morphology during the whole lifetime. In addition, at 1000 steps, the morphology is even better, the tissue reached 96,3% of the French flag target morphology (see Figure 6 A.). The tissue is stressed several times during its lifetime, first as we already described it before 90 steps in order to reach enough of the target morphology to stay alive and 4 times more at: 285, 322, 395, and 896 steps. At these steps, the intrinsic dynamics of the tissue creates deviations from the target morphology that will increase the level of stress and the tissue correct the new anatomical trajectory in order to reach homeostasis and stress decreases. Once it reached a morphology compatible with life, stress is reduced to 0.

In this scheme and the evolution of these cells, stress is needed for long-term survival. Interestingly, the cells have never been evolved on a period of time superior 90 steps. However, when they use stress, they are able to maintain the tissue more than 10 times more. It seems that they also learned allostasis. We will keep the definition of McEwen and Wingfield [56]: allostasis is the process of maintaining stability (homeostasis) through change in both environmental stimuli and physiological mechanisms. In this simulation, the tissue changes regularly as it tries to get rid of the blue cells and sometimes it will become distant enough to the French flag target morphology to activate homeostatic mechanisms to go back to normal and allow long-term maintenance and survival of the tissue.

4.3.3 Artificially-induced stress

4.4 Information-theoretic analysis of the dynamics of the anatomical homeostasis

In this section, we applied information-theoretic measures to analyze the dynamics of the tissue during anatomical homeostasis. We computed the local active information and local transfer entropy for the original simulation without any perturbation (see Figure 3). We also computed the transfer entropy from one stripe to another to understand if one sub-collective can drive the information dynamics and ultimately the anatomical homeostasis (see Figure 7).

The transfer entropy from stress of one agent to its neighbour starts to increase the tissue at timestep 55. Then we wave an important increase at timestep 59 in the locations of the grey and red stripes. This correlates with the increase of stress in the same spatial locations. The overall transfer entropy is almost 300 bits at this timestep (see Figure 7C). Then, the transfer entropy decrease and reaches a steady-state around 0. The black squares represent negative local transfer entropy, meaning that the cells receive information from the stress that reduces the prediction of their next state. We also compared the transfer entropy from stress of one cell to its own state and it is much less important than the transfer entropy from the stress on one cell to the states of their neighbours (see Figure 7C).

The dynamics of the local active information storage on the state states of the cells follow a different dynamics. When transfer entropy is increasing, we can observe several negative local active information storage among the spatial locations of the grey and red stripes. At 55, 59 and 60 timesteps, the AIS is mainly negative and then it will increase gradually for the grey until it reaches the steady-state (see Figure 7A and 7B). For the red stripe, AIS is also globally decreasing and then we observe an important increase around 70 steps, the cells can predict better their future state from the past memory. Concurrently, the stress is decreasing. The local AIS of the blue stripe is 0 all along the dynamics as the cell never change their states.

We also computed the average local transfer entropy from one stripe to another, from the cells corresponding to the spatial locations of the red stripe to the grey and conversely (see Figure 7D). This averaged transfer entropy is 0 at each time step for the blue to red and grey stripes and oppositely. With a time window of $k = 4$, before 60 steps, the tissue change seems driven by the red stripe as we have a peak of 250 bits of transfer entropy from the red stripe to the grey. Just after 60 steps, it is the opposite, transfer entropy is much higher from the grey stripe to the red with a peak around 450 bits of information suggesting that it is now the grey stripe that is driven the change in the states. The ‘organizer’ region seems to change over time.

5 Discussion

In this article, we presented an evolutionary simulation of the scaling of goals based of scale-free cognition and on multi-scale homeostasis. We showed that our simulation used the stress system and evolved it to become instructive, and that our scheme developed error-minimization and homeostatic capacities (robustness to perturbation). The tissue is robust to external perturbation and stress is both instructive and necessary for long-term survival.

Network structure is key to collective intelligence. It has been shown that flat, fully-connected, network structures are the most efficient for collective to resolve a task since they maximise the aggregation of information received from the members of the collective [14]. However, this kind of network is very costly as it necessitates $n(n - 1)$ connections and is probably not biologically realistic. Indeed, in cell networks, cells are only connected to their neighbours. The stress system that can be seen as another communication system when stress is instructive could have the role to change the network structure to flat (fully-connected) structure, at least for some part of the network and may help information aggregation.

The communication system is also fundamental for collective intelligence. In our scheme, communication is mediated via the gap junctions and two types of molecules can be exchanged, the morphogen, the stress molecule and its counterpart, the “anxiolytics”. In our simulation, stress plays a key role. Stress is an ambiguous concept. Stress encompass different meaning, certain stimuli can be stressors, the emergency responses to the stressor is defined as the stress response, and the chronic stress is the over-stimulation of the emergency responses [77]. In addition, the definition of these 3 concepts is circular. A stimulus that initiates a stress response is a stressor, but the physiological or behavioral response considered is stress response if it is initiated in response to a stressor. One solution has been to define stressors as stimuli that disrupt or threaten to disrupt homeostasis [15], but the concept of homeostasis has its own limitations [80]. A more general definition is that stressors are unpredictable and/or uncontrollable stimuli [44]. Interestingly, this last definition fits with the use of the stress system in our simulation. Stress is increasing concurrently with a decrease in local active information storage in the tissue as seen in Figure 7B. In our scheme, evolution use the communication system as a stress system that is activated when the memory in the past is not effective to predict the future. This relates to the active inference framework and the free-energy principle that has been recently developed to integrate homeostasis and allostasis [18] and applied to morphogenetic systems [37, 73].

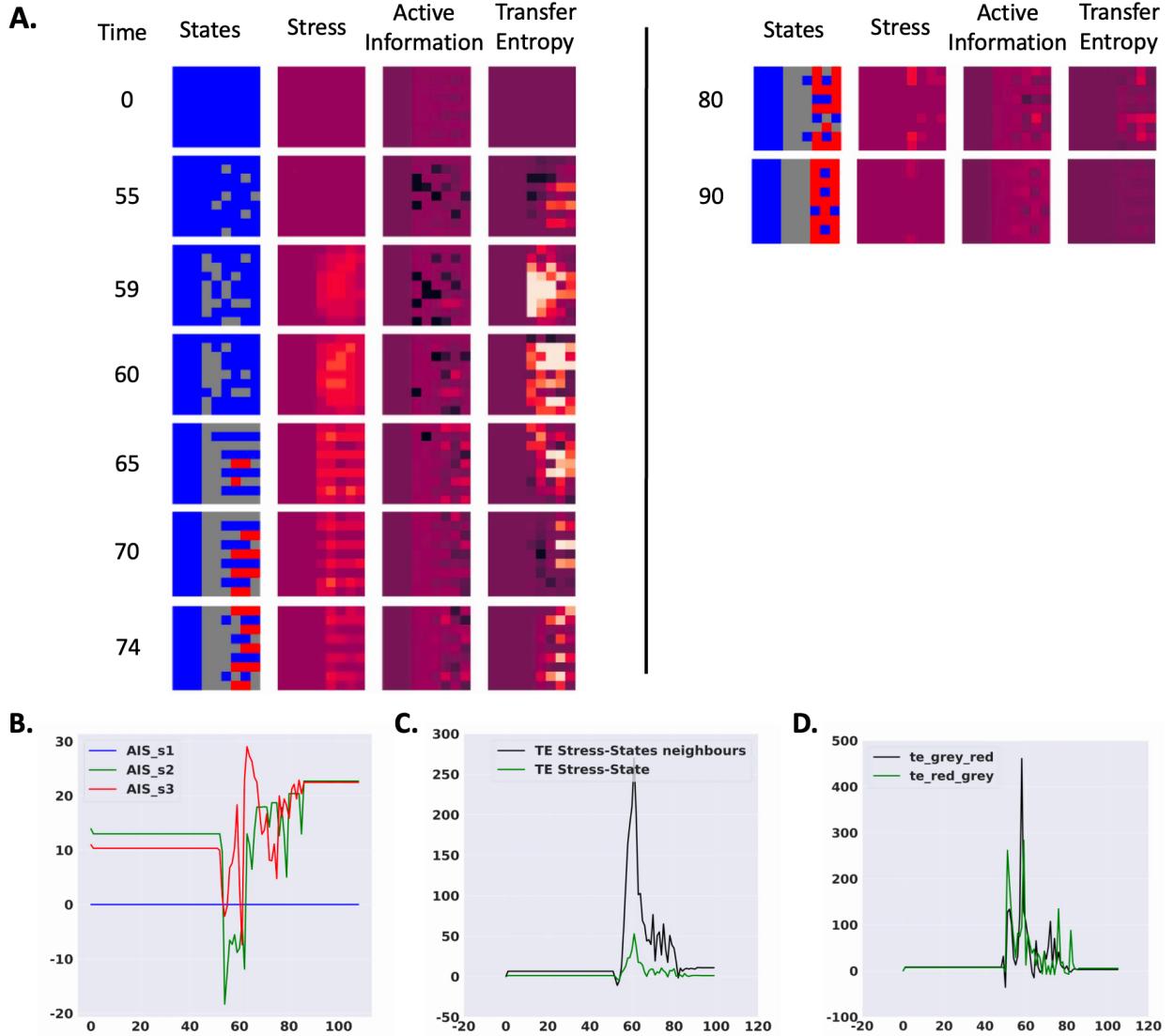


Figure 7: **A.** Time-lapses of the dynamics of resolution of the French flag problem by the tissue. The dynamics of the tissue is represented by 5 timesteps taken at 0, 30, 60, 90 and 120 steps of the dynamics of the tissue. The first column represent these steps, the second column the states of the cells in the tissue (blue, grey, red). The second column represent the use of the stress system on a red scale **B.** This figure represents the sum of active information storage by stripes, s1 is the blue stripe (blue), s2 is the white stripe (green), s3 is the red stripe (red). **C.** This figure represents the sum of the averaged transfer entropy from stress time series to the state time series of the neighbors (black) and the transfer entropy from stress states to the internal states of the cells (green) **D.** This figure represents the transfer entropy from one stripe to another, grey to red stripe (black), red to grey (red).

In this study, we focused on the scaling of goals from the scale-free cognition and multi-scale homeostasis perspective. In artificial intelligence, the fact that all intelligences are collective intelligences is rarely the main approach [42]. There are recent attempts to develop new methods based on collective intelligence [29,60]. Deep learning started to integrate the approach with adversarial networks with two networks working together [28]. Artificial collective intelligence is usually studied from the swarm intelligence or human society point of view [34, 63] but morphogenetic system present several characteristics that swarm lacks: a (growing) grid network architecture, the informational wiping property or a different communication system closer to the information processing of a cellular automata that can only communicate with its direct neighbours. This work is one step to integrate the Basal cognition finding into artificial intelligence

Some things to discuss: synchronization, low frequencies,

Ok but the choice of which collective gets more, this is self-driven (we don't force it), so I guess they decide whom to talk to as part of the development? ↴ Completely self-driven, constrained by the French flag problem.

add PCA figure of the navigation in morphospace

compute the transfer entropy from energy (controlled by the anatomical loop) to the states (seen as behaviour and single-cell level)

stats

the "organizer region" change over time, see 7D

do we add experimental data

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