Genetically-regulated Neuromodulation Facilitates Multi-Task Learning

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ABSTRACT

In this paper, we use a gene regulatory network (GRN) to regulate the learning parameters of State-Action-Reward-State-Action (SARSA) algorithm. The GRN modulates the SARSA parameters: learning rate, discount factor, and memory depth. We have optimized GRNs with a genetic algorithm to regulate these parameters on specific problems but with no knowledge of problem structure. We show that GRN-regulated SARSA performs equally or better than the SARSA with fixed parameters. We then extend the GRN-regulated SARSA algorithm to multi-domain problem generalization, and show that GRNs trained on multiple problem domains can generalize to previously unknown problems with no further optimization.

Categories and Subject Descriptors

I.2.6 [**Learning**]:

Keywords

Reinforcement learning, Gene regulatory network, Parameter control

1. INTRODUCTION

2. SARSA ALGORITHM

Reinforcement learning is a reward-based learning algorithm that allows agents to learn from experience. More formally, reinforcement learning (RL) is a mathematical framework for learning from a reward signal that is derived from Bellman's equation for optimal control [13]. One of the most important forms of RL is temporal-difference (TD) RL. TD-RL is a method for learning optimal behavior from changes in state and reinforcement by error prediction [12]. TD-RL agents learn an expected return that will be received after taking an action in any state. Strong correlations with this type of error predictive behavior have been found in studies of dopamine neurons [10]. This line of research has continued and is now been supported by fMRI data of reward processing for tastes, money, and love [6].

TD-RL is used to solve Markov decision processes, which are an extension of Markov chains to problems framed in terms of state, action, and reward. Reward signals (such as reinforcement of dirt cleanup) are geometrically encoded in a table which associates action preferences with states. The basic TD(γ) algorithm updates one state-action association at a time which prohibits sequence learning. Eligibility traces are used to associate reward with sequences of actions by reinforcing a weighted history of most recent actions. In this study the online version of TD-RL, SARSA (short for, state-action-reward-state-action), is used. A review of the nuances of reinforcement learning can be found in [13].

We include a few of the key equations from the RL algorithm which are employed. If we are in state, s_t at time t, then we will take some action a_t which will bring us a reward r_t. This action will also cause us to transition to a new state, s_{t+1}. The SARSA algorithm learns a Q-function, which maps a value to each state-action pair, (s_t, a_t). From each state multiple actions, A_t, may be taken which may be a function of s_t (for example, an obstacle may prevent an action in a given state). Given an optimal Q-function the best action to take is

$$argmin_{a_t \in A_t} Q(s_t, a_t). \tag{1}$$

The Q-function is approximated by SARSA with the following update rule

$$Q(s_t, a_t) \leftarrow Q(s_t, a_t) + \alpha\big[r_{t+1} + \gamma Q(s_{t+1}, a_{t+1}) - Q(s_t, a_t)\big] \tag{2}$$

where α is the learning rate, and γ is the discounting factor. Given only this update rule it can be difficult to compute the Q-value for state-action pairs which indirectly contribute to obtaining a reward. This update method propagates information only to the preceeding state-action pair, for those that are very distant from the reward, such as in the case of maze solving problems, this can require a large number of repeated trials. However, this problem of reward propagation can be partially aleviated by the use of eligibility traces. Eligibility traces store an accumulating trace of state-action pairs. The "memory" of these state-action pairs can be tuned with the trace decay parameter λ. Eligibility traces are updated with

$$e_t(s,a) = \begin{cases} \gamma\lambda e_{t-1}(s,a) & \text{if } s \neq s_t \\ \gamma\lambda e_{t-1}(s,a) + 1 & \text{if } s = s_t \end{cases} \tag{3}$$

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By combining the error predictive capabilities of TD-RL with the state-action sequence memory of eligibility traces we can amplify the effects of our reward and speed up the learning process. When performing on-policy learning it is important to ensure that a sufficient amount of exploration occurs. To this end the ϵ-greedy method is used, where a random action is taken with $p(\epsilon)$, otherwise the agent's most preferred action is taken. However, the RL algorithm can still fail to capitalize on rarely experienced rewards.

3. GENE REGULATORY NETWORK

Our model uses an optimized network of abstract proteins. The inputs of the agent are translated to protein concentrations that feed the GRN. Output proteins regulate the reinforcement learning parameters previously described. This kind of controller has been used in many developmental models of the literature [7, 5, 3] and to control virtual and real robots [14, 9, 8, 4].

We have based our regulatory network on Banzhaf's model [1]. It is designed to be as close as possible to a real gene regulatory network but neither to be evolved nor to control any kind of agent. However, Nicolau used an evolution strategy to evolve the GRN to control a pole-balancing cart [9]. Though this experiment behaved consistently, the evolution of the GRN has been an issue. We have decided to modify the encoding of the regulatory network and its dynamics. In our model, a gene regulatory network is defined as a set of proteins. Each protein has the following properties:

- The protein *tag* coded as an integer between 0 and p. The upper value p of the domain can be changed in order to control the precision of the GRN. In Banzhaf's work, p is equivalent to the size of a site, 16 bits.
- The *enhancer tag* coded as an integer between 0 and p. The enhancer tag is used to calculate the enhancing matching factor between two proteins.
- The *inhibitor tag* coded as an integer between 0 and p. The inhibitor tag is used to calculate the inhibiting matching factor between two proteins.
- The *type* determines if the protein is an *input* protein, the concentration of which is given by the environment of the GRN and which regulates other proteins but is not regulated, an *output* protein, the concentration of which is used as output of the network and which is regulated but does not regulate other proteins, or a *regulatory* protein, an internal protein that regulates and is regulated by other proteins.

The dynamics of the GRN is calculated as follow. First, the affinity of a protein a with another protein b is given by the enhancing factor u^+_{ab} and the inhibiting u^-_{ab}:

$$u^+_{ab} = p - |enh_a - id_b| \quad ; \quad u^-_{ab} = p - |inh_a - id_b| \qquad (4)$$

where id_x is the tag, enh_x is the enhancer tag and inh_x is the inhibiting tag of protein x.

The GRN's dynamics are calculated by comparing the proteins two by two using the enhancing and the inhibiting matching factors. For each protein in the network, the global enhancing value is given by the following equation:

$$g_i = \frac{1}{N}\sum_j^N c_j e^{\beta(u^+_{ij} - u^+_{max})} \quad ; \quad h_i = \frac{1}{N}\sum_j^N c_j e^{\beta(u^-_{ij} - u^-_{max})} \qquad (5)$$

where g_i (or h_i) is the enhancing (or inhibiting) value for a protein i, N is the number of proteins in the network, c_j is the concentration of protein j and u^+_{max} (or u^-_{max}) is the maximum enhancing (or inhibiting) matching factor observed. β is a control parameter described hereafter.

The final modification of protein i concentration is given by the following differential equation:

$$\frac{dc_i}{dt} = \frac{\delta(g_i - h_i)}{\Phi} \qquad (6)$$

where Φ is a function that keeps the sum of all protein concentrations equal to 1.

β and δ are two constants that set up the speed of reaction of the regulatory network. In other words, they modify the dynamics of the network. β affects the importance of the matching factor and δ affects the level of production of the protein in the differential equation. The lower both values, the smoother the regulation. Similarly, the higher the values, the more sudden the regulation.

4. NEUROMODULATION

In living organisms, neuromodulators are neuropeptides or small molecules, such as dopamine and serotonin. The production of these substances within the cell is controlled by gene regulatory networks. Neuromodulators change the behavior of neural networks within individual neurons, amongst neighboring neurons, or throughout the entire network. Neuromodulation has been found to be pervasive throughout the brain, and can have drastic consequences on the behavior of neurons and neuronal circuits [**?**, **?**, **?**]. A particularly applicable example in the realm of robotics is the neuromodulation of motor signals produced by central pattern generators in the brain and spinal cord [**?**]. It has been found that neuromodulators tune and synchronize neuromuscular signals [**?**].

We have already noted that the temporal difference learning algorithm for error prediction has been observed in neural substrates [**?**]. Dopamine neurons of the ventral tegmental area (VTA) and substantia nigra exhibit this error predictive behavior. The dopamine system is itself a neuromodulatory system. While the temporal difference learning algorithm extends ideas of reward processing to engineering, there are models of the dopamine system with closer ties to biology [**?**]. These models also confirm the error predictive behavior found in the brain for a variety of physiological data including reaction-time and spatial-choice tasks. Dopamine is an important neuromodulator, especially in learning, but it is but one of many neuromodulatory substances found in the brain. An extensive review of computational models of neuromodulation can be found in [**?**], and some recent models are reviewed in [**?**]. In this study we focus on the relationship between evolved neuromodulator-producing GRNs and learned behaviors.

4.1 Regulating parameters

With the intention of simplyfing the computational model, all the molecular pathway of neuromodulation has been deliberately ignored and only the consequences of neuromodu-

lation on learning behaviors are targetted. Therefore, the artificial gene regulatory network presented in the previous section directly regulates the learning parameters of the SARSA algorithm. Three learning parameters are considered in this work: the learning rate α, the discount factor γ and the memory depth λ.

To do so, the GRN uses three inputs that describes the current performance of the agent in the environment. They have been chosen to be problem-independent: one of our goal is to reduce the configuration of our neuromodulation architecture to have minimum changes to applied when the problem changes. The first input describes the duration since the beginning of current episode: the concentration of this first input protein increases when the agent takes too much time to solve the problem or is getting closer to the end of the episode. The concentration $C_{I_1}(t)$ of this input protein at time step t is calculated as follows:

$$C_{I_1}(t) = e^{-\frac{t^2}{t^2_{max}}} \tag{7}$$

where t_max is the expected duration of an episode. The second output protein's concentration $C_{I_2}(t)$ describes the quality of the current sequence of actions in term of rewards, smoothed on 25 steps:

$$C_{I_2}(t) = \frac{\sum_{s=1}^{25} \Big((25-s) \times q_s(t-1)\Big)}{\sum_{s=1}^{25} s} \tag{8}$$

$$\text{with } q_s(t) = \begin{cases} 1 + 1000\frac{q.e}{q.q*e.e} & \text{if} \frac{q.e}{q.q*e.e} > 0.4995 \\ 0 & \text{otherwise} \end{cases}$$

where e is a vector that contains the current possible action rewards and q is a matrix that contains all the state/action rewards encountered by the agent at time step t. The aim of this input is to capture the quality of the current state according to the past states the agent has visited. Finally, the third input protein's concentration $C_{I_3}(t)$ informs the GRN about the 25-step smoothed average reward the agent can obtain in its current state:

$$C_{I_3}(t) = \frac{\sum_{s=1}^{24} \Big((25-s) \times C_{I_3}(t-s)\Big) + 25 \times \sum_{r_e \in e} r_e}{\sum_{s=1} 25s} \tag{9}$$

where e is the vector of current possible rewards. This input informs the GRN whether or not the agent is stranded within a long-term decreasing reward.

In addition to these inputs, the GRN uses four output proteins to regulate the learning parameters:

- the output protein O_n, which concentration C_n normalizes the concentration of other outputs[1],
- the output protein O_α of concentration C_α, which provides the value for α to the SARSA algorithm with $\alpha = C_\alpha/(C_\alpha + C_n)$,
- the output protein O_γ of concentration C_γ, which provides the value for γ to the SARSA algorithm with $\gamma = C_\gamma/(C_\gamma + C_n)$,

[1]this is generally used in regulatory networks to obtain output values in $[0, 1]$

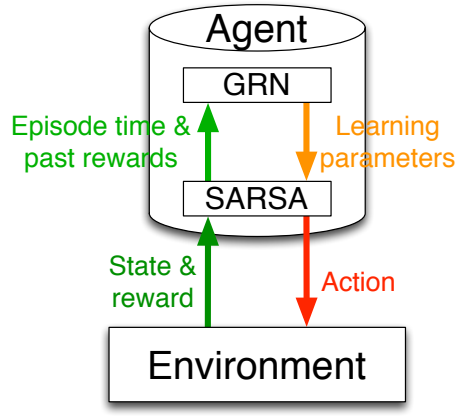

Figure 1: At every time step, SARSA updates the GRN inputs. The GRN returns updated learning parameters that will be used by the SARSA algorithm.

- the output protein O_λ of concentration c_λ, which provides the value for λ to the SARSA algorithm with $\lambda = C_\lambda/(C_\lambda + C_n)$.

As depicted by figure 1, the GRN updates SARSA's learning parameters at every time step, before SARSA updates its internal variables and prediction. The GRN returns the learning parameters SARSA uses for its own decision step.

4.2 GRN Optimization

Before using a gene regulatory network for neuromodulation, the network of proteins needs to be optimized. In this paper, we use an adapted genetic algorithm inspired from the NEAT algorithm [11]. Three features are modified in comparison to a standard genetic algorithm:

- the *initialization* of the algorithm - as opposed to initializing with individuals randomly sampled from the complete distribution, only small networks are used in the initial population so as to allow for a more progressive complexification,
- the *speciation* protects newly appeared solutions by giving them some time to optimize their structures before competing with the whole population, and
- the *alignment crossover* with the use of a distance metric between proteins to keep subnetwork architecture during the crossover operation.

This modified algorithm has been proven to converge faster to better solutions. More details can be find in [2].

During its optimization, each GRN is evaluated independently on a given problem with 25 reruns in order to reduce stochasticity impact of the problems on the fitness. The fitness being problem dependent, more explanations will be provided in the experience section. To avoid any memory biais, SARSA is completely resetted before each evaluation.

5. EXPERIENCES

5.1 Problems

5.1.1 Mountain Car

5.1.2 Maze

5.1.3 Puddle World

5.1.4 Actor critic pendulum

5.2 Training GRN on one specific problem

5.3 Generalization of the regulation

6. CONCLUSION

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