Politecnico di Torino

Optimization methods for engineering problems

Machine Learning through Artificial Immune Systems (AIS)

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Outline

- Artificial Immune Systems
- Three paradigms of learning
  - Unsupervised
  - Supervised
  - Reinforcement Learning
- aiNet for data analysis
  - Overview & algorithm
  - Clustering of points in a 2D space
- Dudek's AIS
  - Overview & algorithm
  - Forecasting of electricity load profiles
- Kato and Kobayashi's IS
- Conclusions

Artificial Immune Systems

"Adaptive system[s], inspired by theoretical immunology and observed immune function, principles and models, which are applied to problem solving." de Castro and Timmis

The main motivation for building immune-inspired solutions to engineering problems arises from the following characteristics of IS:

- **self-organization** of huge numbers of immune cells;
- **distributed operation** throughout the body;
- **pattern recognition** and anomaly detection (**self/nonself discrimination**);
- **optimization** and **memory** to improve and remember immune responses.

Characteristics of the **immune system** as a **defence system**:

- **specialized cells;**
- subdivided into:
  - **innate immunity**: generic defence acting on general classes of pathogens (does not adapt in the lifetime)
  - **adaptive immunity**: ability to adapt and react to unseen pathogens based upon exposure to them (**learning mechanism)**
- **recognition mechanism**: protein molecules on the immune cell surface act as biochemical receptors.

Four main classes of AIS algorithm, inspired by different theories of the IS:

- **Clonal Selection** (affinity maturation)
- **Immune Network** (self-organization of the IS as a regulated network)
- **Negative Selection** (based on the concept of self–nonself discrimination choice)
- **Danger Theory** (danger-theory-inspired systems based on the recognition of danger zone where the intruder acts)

Three paradigms of learning

UNSUPERVISED LEARNING (UL) :

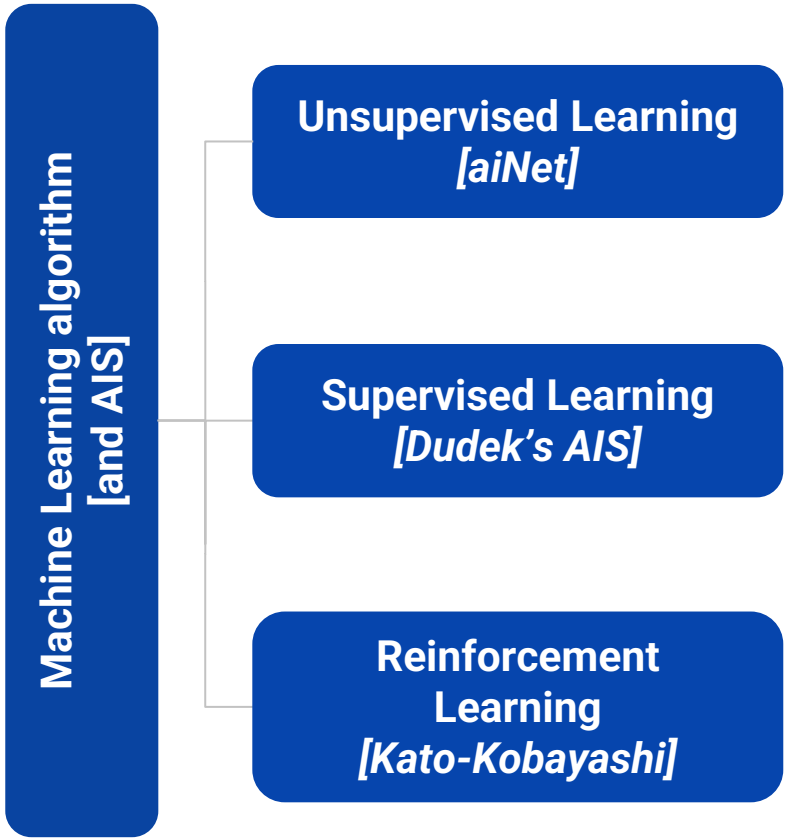
- **learning patterns from untagged data** (e.g. clustering, pattern recognition)
- **self-organization** to capture **patterns** as probability densities or a combination of neural feature preferences.
- two popular methods are **k-means** and **Self-Organizing Maps**.

SUPERVISED LEARNING (SL) :

- task of learning a **mapping function** between input and output
- training samples of input-output pairs are used (**instructive feedback**).
- an SL algorithm uses the training data to **optimize** its mapping function, which can be used for mapping **new** samples (unlabelled data)

REINFORCEMENT LEARNING (RL):

- an **agent** interacts with an **environment** through a sequential process: observe state, take action, get reward .
- the agent does not receive as feedback which action was best but how the action taken was good (**evaluative feedback**)
- the goal of the agent is maximising the **cumulative reward** over time



Richard S. Sutton, Incomplete Ideas. [Online]. Available at: http://www.incompleteideas.net/

aiNet for data analysis

"The aiNet is an edge-weighted graph, not necessarily fully connected, composed of a set of nodes, called antibodies, and sets of node pairs called edges with an assigned number called weight […]."

- the learning algorithm aims at **building a memory** set of antibodies that recognize and represent the **structural organization** of the input **unlabelled** data (i.e. AGs).
- memory set is a **network** composed of AB cells (nodes) interconnected by links (edges) with associated connection strengths, depending on the Ab-Ab affinity (weights)
- this network is the **internal image** of input patterns (AGs).
- **affinity** between ABs and ABs or AGs is inversely proportional to their distance in the **shape space** (L-dimensional space)
- the algorithm makes use of two concepts from AIS theory:
  1. **clonal selection** (and affinity maturation) to locally improve each antibody (**exploitation**)
  2. **network suppression** to eliminate memory cells that are too close to each other (**exploration**)
- the AIS algorithm **does not** perform clustering directly, but it uses **hierarchical clustering** techniques on the aiNet graph.

aiNet algorithm

1. load set of **antigens** $Ag_j \in S_{Ag} \subset \mathbb{R}^{n_{Ag} \times L}$
2. randomly initialize set of **antibodies** $Ab_i \in S_{Ab} \subset \mathbb{R}^{n_{Ab} \times L}$ and **empty memory** set M
3. for $t = 1, \dots, t_{max}$ do:

   3.1 for $Ag_j \in S_{Ag}$ do:

   3.1.1 compute $f_{i,j}$ **affinity** with Ag_j for each $Ab_i \in S_{Ab}$

$$D_{i,j} = \|Ab_i - Ag_j\|$$

$$f_{i,j} = 1/D_{i,j}$$

   3.1.2 **select** n_{best} antibodies with highest affinity to Ag_j

   3.1.3 generate $n_{c,i}$ **clones** $Cl_{k,i}$ for each best antibody (clonal pool)

$$n_{c,i} = n_{Ab} \left(\frac{f_{i,j}}{f_{max,j}} \right)$$

   3.1.4 **mutate** clones with rate inversely proportional to parent's affinity

$$Cl_{k,i} = Ab_i + \alpha_i \, rand(\quad)(Ag_j - Ab_i)$$

$$\alpha_i = \beta \, e^{-f_{i,j}}$$

   3.1.5 put percentage of best clones (highest affinity) into **clonal memory**

   3.1.6 perform ***apoptosis*** (remove clones distant from antigen)

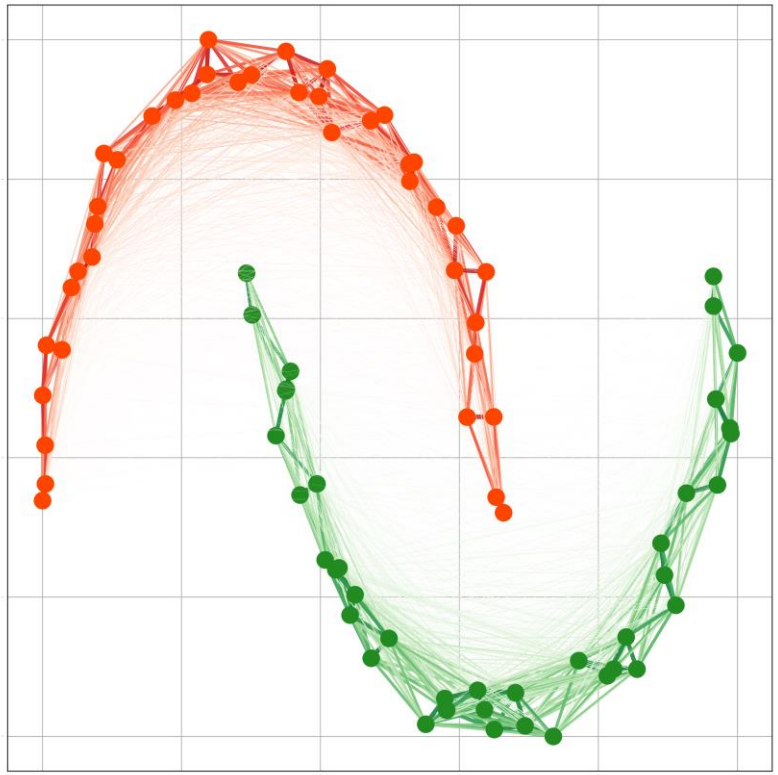
   3.1.7 perform **clonal suppression** (remove clones close to each other)

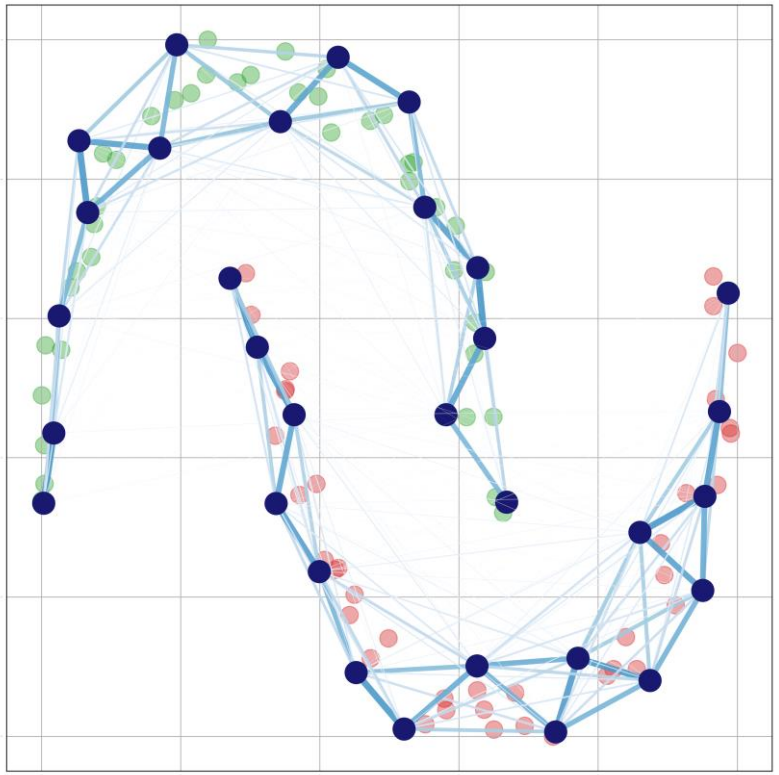
   3.1.8 put surviving clones in **memory** set M

   3.2 perform ***network suppression*** (remove antibodies in M close to each other)

   3.3 **update** S_{Ab} with M *and* n_{new} *randomly generated elements*
4. get final memory set M (**nodes**) and calculate **weights** of Ab-Ab connections (affinity)

Results on moons dataset





- data divided into **2 non-convex** clusters (moon-shaped)
- clustering performed through **hierarchical clustering** (graph analysis)
- results of clustering on the **whole** dataset (left)
- results of clustering after using aiNet to **reduce** number of samples (right)
- the algorithm is still able to find the two clusters while working on a smaller (**representative**) set

Dudek's AIS for regression (SL)

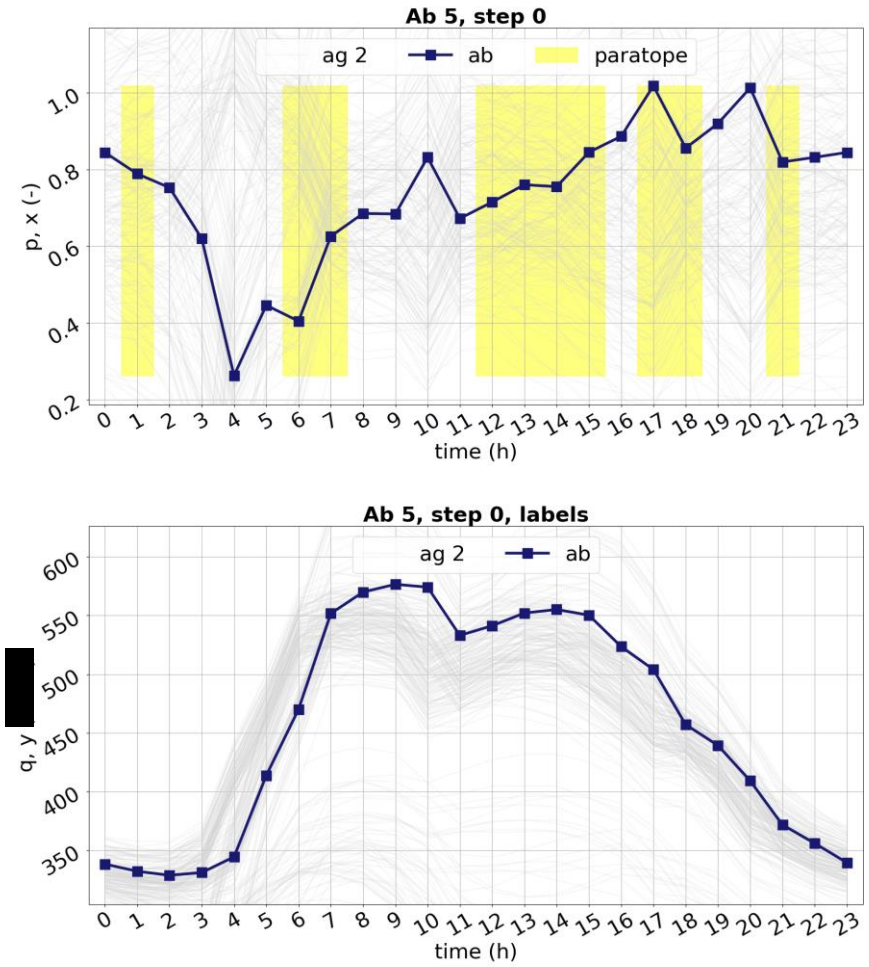
The Dudek's model is based on AIS for **forecasting time series** (e.g. short-term electrical load). The main feature is the **embedded** property of **local selection**: each antibody learns its optimal subset of features (a paratope) to improve its recognition and prediction abilities.

- AGs are input-output pairs that represent the **training samples**:
    1. vector of **features**, **x**, i.e. input variables, (e.g. day-ahed load profile)
    2. label, **y**, containing the related **ouput** variables (e.g. time-series of the load of the following day)

- ABs are expected to **cover** regions of the input space and to provide an output, hence they are composed of:
    1. pattern that **matches** the antigens features (**p**)
    2. label, **q**, containing the **predicted** output values
    3. paratope, **Ω**, i.e. a subset of the active features (**feature selection**)
    4. radius of the **recognition region**, which is an hyper-ball in the paratope sub-space
    5. power, i.e. **number** of antigens with the antibody is able to recognize

- **AB mutation** acts on paratopes.
- System goes on alert whenever an AG is not recognized (nonself), thus allowing further analysis.

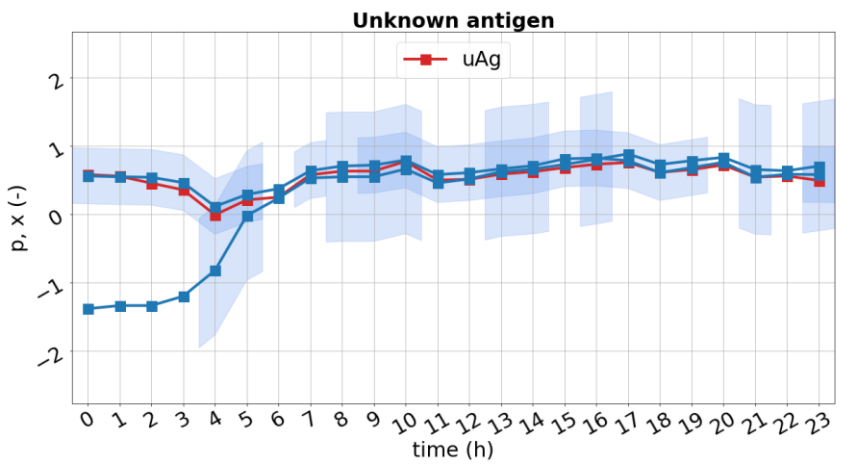
Dudek's algorithm

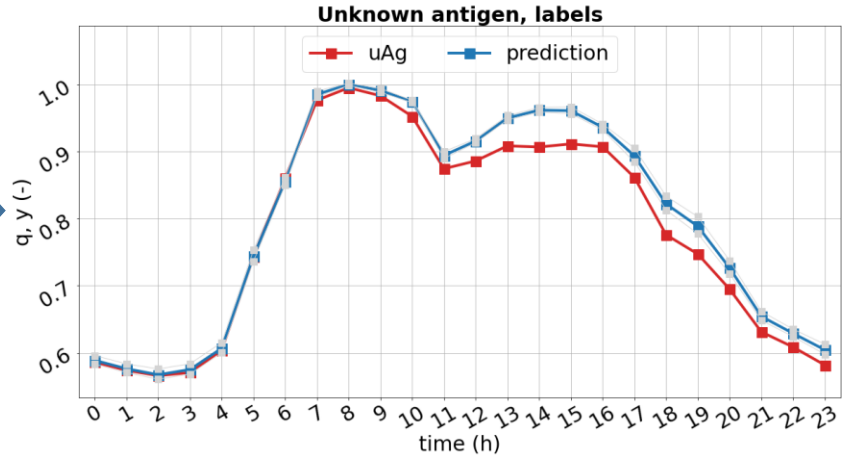


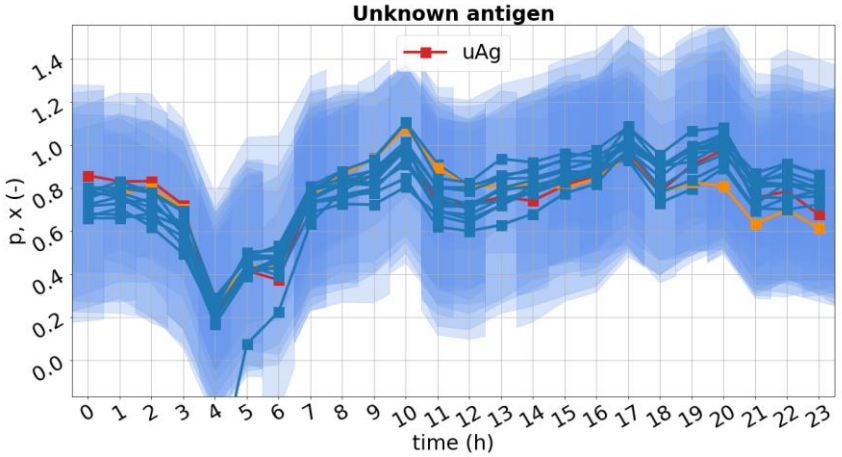
1. load **antigens** population $S_{Ag} = \{Ag_j = (\boldsymbol{x}_j, \boldsymbol{y}_j), \boldsymbol{x}_j \in \mathbb{R}^{L_x}, \boldsymbol{y}_j \in \mathbb{R}^{L_y}\}_{j=1,..,N}$
2. initialize set of **antibodies** $S_{Ag} = \{Ab_i = (\boldsymbol{p}_i, \boldsymbol{q}_i), \boldsymbol{p}_i = \boldsymbol{x}_j, \boldsymbol{q}_i = \boldsymbol{y}_j, \boldsymbol{\Omega}_i, r_i, P_i\}_{i=1,..,N}$
3. for $k = 1, \dots, N$ **do**:
   - 3.1 **select** current antibody $Ab = Ab_k$ (parent)
   - 3.2 while not stop condition **do**:
     - 3.2.1 for $l = 1, \dots, Z$ **do**:
       - 3.2.1.1 create **clone** $Cl_l = Ab$
       - 3.2.1.2 perform hypermutation on **paratope** $\boldsymbol{\Omega}_l = hyp(\boldsymbol{\Omega}_l)$
       - 3.2.1.2 compute **cross-reactivity** threshold $\mathrm{r}_l = cr(\mathrm{r}_l)$
         - **divide** antigens into class1 and class2 (error on labels)
         - identify **closest** antigen of class2, B, ($d_{l,B}$*)
         - select **farthest** antigen of class1, A, ($d_{l,A}$*) s.t. $d_{l,A} \leq d_{l,B}$
         - **evaluate** $r_l = d_{l,A} + C\left(d_{l,B} - d_{lA}\right), \mathrm{C} \in [0,1] \subset \mathbb{R}$

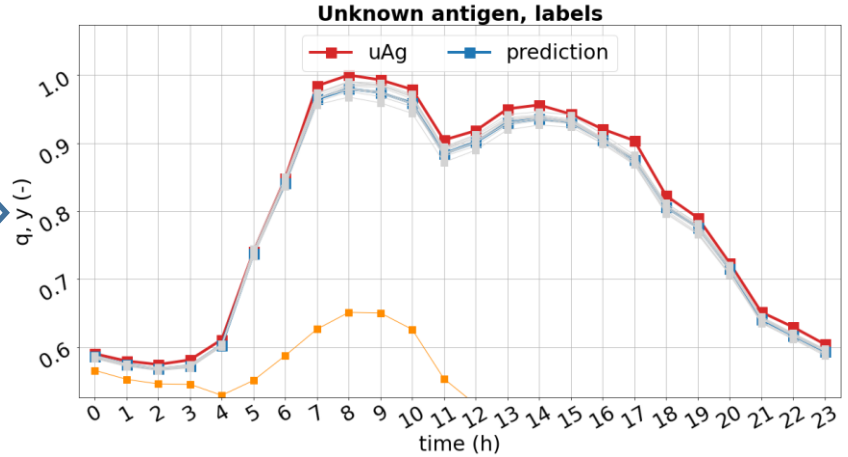
         * $d_{l,j} = \left(\sum_{t \in \Omega_i} \left|p_{l,t} - x_{j,t}\right|^e\right)^{\frac{1}{e}}, \; e = 1 \; or \; 2$
       - 3.2.1.2 compute **affinity** of antigens in class1 (linear function of $d_{l,j}$)
       - 3.2.1.3 compute **power** (number of antigens with affinity larger than 0)
     - 3.2.2 **rank** clones based on: 1. Power, 2. Paratope size, 3. Random choice
     - 3.2.3 select winner to **replace** the parent $Ab_k \leftarrow winner\left(\{Cl_l\}_{l=1,..Z}\right)$
     - 3.2.4 update **label** based on labels of antigens (weighted on the affinity)

Results on electric load forecasting







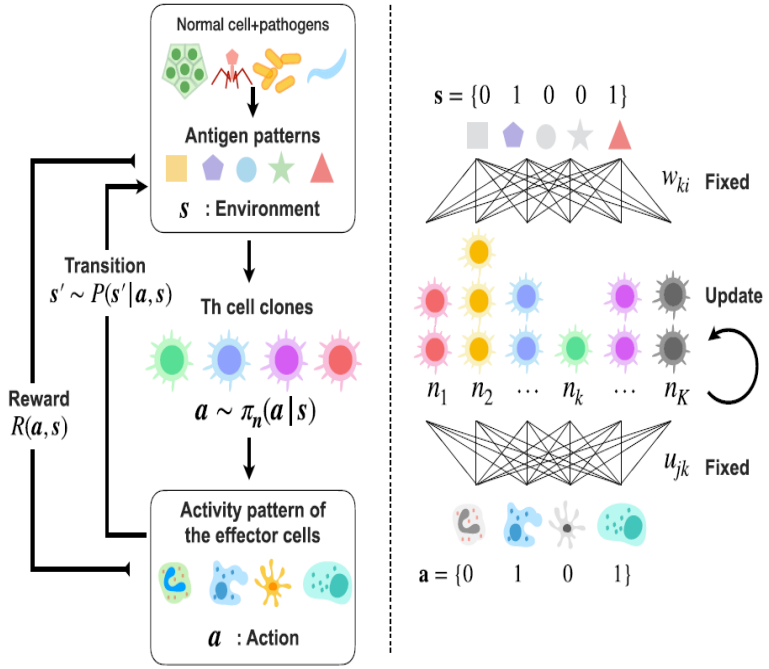


- algorithm trained on 1-years EGEA data of day-next day pairs
- train/test ratio equal to 0.8
- 4.3% MAPE on recognized Ags (38%).
- the figures show how the recognition of **new antigens** works (left plots)
- trained antibodies that **recognise** the new antigen provide their **response** (label, i.e. prediction)
- a **global** response is obtained using weights (affinity of each antibody)
- the **predicted** value is compared with the **actual** output from the test set.

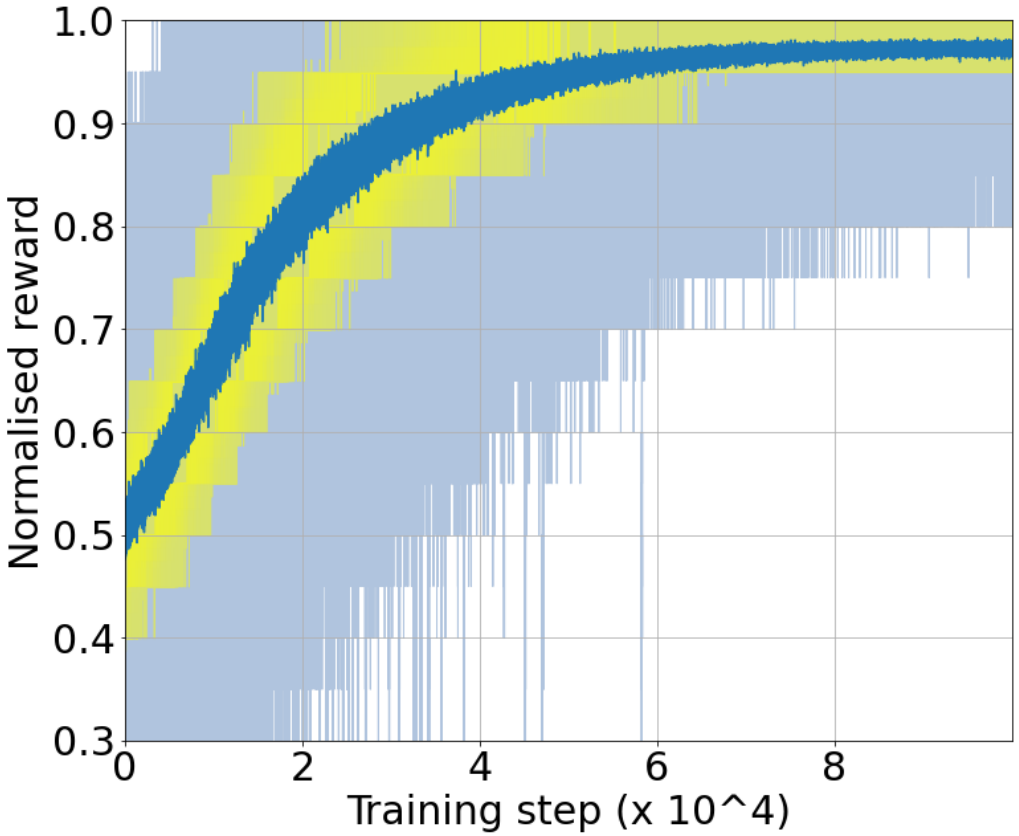
Kato-Kobayashi's model of IS

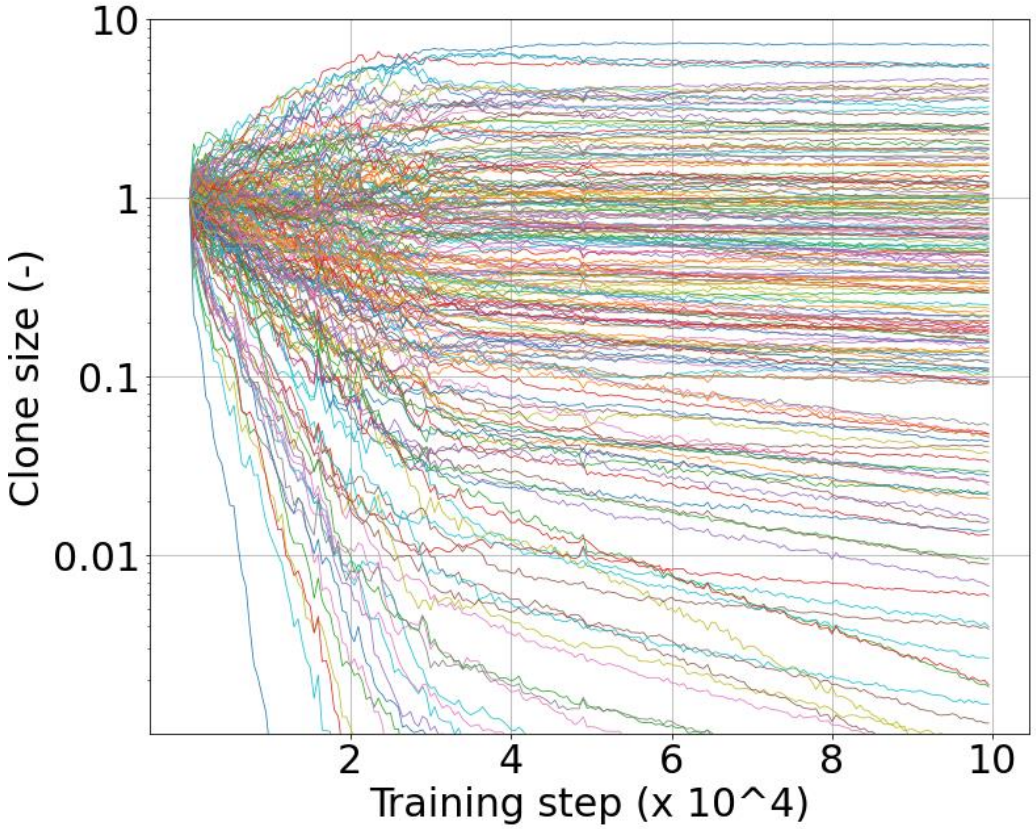
The vast majority of AIS operate at the **individual** level: a clonal selection loop evolves Abs so that offspring tend to posses higher recognition capability. But what if focus on the **population** level?

- Kato and Kobayashi attempt to model **population dynamics** of AIS using RL, thus highlighting several parallels between the two fields.
- based on this assumption, they aim at modelling the **learning dynamics** of the IS which is exposed and has to respond to a set of AG patterns (binary vectors).
- the system's response works on **two** levels:
    1. Different types of **Th cells** recognize the AGs (states) and produce cytokines.
    2. **Effector Cells** understand cytokine production and consequently induce immune response activity (action).
- the system learns how to bias the activity of Th-cells only by adjusting the **distribution** of the sizes of each type: this is realized with the help of **SARSA** algorit
- The RL model induces optimal neural behavior in the cell population: overall net activity provides an estimate of the **Q-function**.
- The body (environment) responds to the IS (agent) with a **reward** and a **transition** depends probabilistically on the action induced by the IS



Results of training





Conclusion

- As stated by Timmis in several works, the application of AIS often failed (or proved ineffective) because of unproper treatment and misunderstanding.
- The immune metaphor still has a lot of potential to express in the domain of **Computational Intelligence** and, w.l.o.g., in optimization settings that can be different from traditional multiobjective problems.
- Statistical Machine Learning seems to us the enabling formal framework to properly analyze, treat and exploit the features of immune systems.
- We believe the most promising paradigms for immune-ispired learning are SL and RL. Particular attention should be given to the investigation of Transfer and Incremental Learning methods.

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Machine Learning through Artificial Immune Systems

Thanks for your attention !

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