

Ortus: an Emotion–Driven Approach to (artificial) Biological Intelligence

Andrew W.E. McDonald, Sean Grimes, and David Breen

Drexel University, Philadelphia, PA 19104
awm32@cs.drexel.edu

Abstract

Ortus is a simple virtual organism that also serves as an initial framework for investigating and developing biologically-based artificial intelligence. Born from a goal to create complex virtual intelligence and an initial attempt to model *C. elegans*, Ortus implements a number of mechanisms observed in organic nervous systems, and attempts to fill in unknowns based upon plausible biological implementations and psychological observations. Implemented mechanisms include excitatory and inhibitory chemical synapses, bidirectional gap junctions, and Hebbian learning with its Stentian extension. We present an initial experiment that showcases Ortus' fundamental principles; specifically, a cyclic respiratory circuit, and emotionally–driven associative learning with respect to an input stimulus. Finally, we discuss the implications and future directions for Ortus and similar systems.

Introduction

While much work has been done to develop artificial intelligence (AI) systems that borrow principles from organic nervous systems, far less has been done that specifically targets the intersection of biology and artificial intelligence such that adherence to biological principles is of primary concern—rather than to the specific applicability of the technology—with the main goal being a virtual system that exhibits biological intelligence (BI). As our understanding of organic nervous systems, and access to computation power are ever-growing, widespread interest in systems that do exactly this is greatly increasing. Evidence of this lies in DARPA's recent L2M project, which publicizes their search for machines that learn throughout their lives (DARPA, 2017).

Researchers in the realm of computational biology and neuroscience have made progress toward developing systems that model specific organisms or neural circuits, as seen in work done with the nematode *Caenorhabditis elegans* (*C. elegans*) (Izquierdo and Beer, 2016). These systems have the potential to require too much focus on organism–specific details in order to achieve proper functionality, shifting focus away from creating more generalized neurologically–inspired intelligent systems.

On the other hand, more traditional (application focused) AI research has started taking more inspiration from human learning, such as the development of an auto–encoder augmented by Hebbian learning, decreasing the need for an initial supervised–like learning period (Bowren et al., 2016). Further, Marblestone et al. (2016) discusses ways that artificial neural networks (ANNs) can more closely approximate neural functionality. In the context of biologically–inspired AI, the frameworks underlying these approaches may be too constraining for full exploration of the potential for the field of biologically–based artificial intelligence.

Recent work at the intersection of these two areas includes Sinapayen et al. (2016), which investigates the applicability and biological plausibility of spiking neural networks learning by “stimulation avoidance”. Perhaps the project most closely aligned to Ortus is a biologically inspired neural network modeled off of a honey bee's visual system, which merges biological mechanisms and neural networks (Roper et al., 2017).

Ortus is an initial implementation of, and framework for creating, virtual life aimed at approximating the intelligence of living organisms. Born from the study and analysis of *C. elegans*' connectome and behavior, it aims to strike a balance between biological abstraction, retention of biological fidelity, and computation scalability in order to approximate biological intelligence and learning as closely as possible. At its core, Ortus is a network of biologically–inspired, non-spiking neurons, capable of forming excitatory, inhibitory, and electrical synapses. Similar to the way the structure of *C. elegans*' 302 neuron nervous system is capable of complex behaviors including toxin avoidance, reflexively withdrawing from a “tap”, and “remembering” the temperature at which it found food (Jarrell et al., 2012), Ortus' “connectome” (neural structure) enables its inherent functionality. Once running, Ortus refines its network—similar to the way organic nervous systems adjust themselves (though far simplified, and not yet as dynamic), based upon Ortus' intrinsic “understanding” that certain things are “good” and others are “bad”, with regard to its own longevity. This understanding is derived from the structure of the nervous system

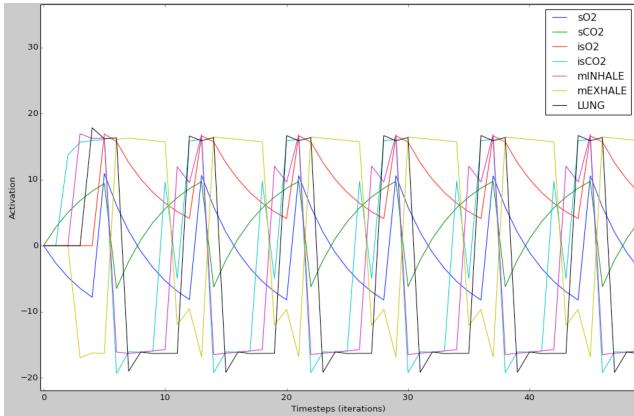


Figure 1: Activation cycle of Ortus’ respiratory circuit. It can be seen that sCO_2 “naturally” rises, while sO_2 “naturally” falls. In response to this, $isCO_2$ (an SEI) increases in activation, which excites the inhalation motor neuron ($mINHALE$), causing $LUNG$ to activate. This in turn causes O_2 to spike, which excites isO_2 (also an SEI). The excited isO_2 inhibits $mINHALE$ while exciting $mEXHALE$, thereby allowing CO_2 to rise—completing the cycle.

it generates for itself from a set of input definitions given to it.

The remaining sections of this paper outline Ortus’ design and implementation, describe an initial experiment, discuss the implications of this framework, and analyze its shortcomings.

System Design

As Ortus aims to be a virtual analogy to intelligent life, we tried to only implement functions that either had a known analogous biological process, or which may have an analogous biological implementation that is unknown, but can be defended with anecdotal evidence. Following each Ortus design element (ODE) below, is its biological rationale (BR):

ODE 1: The underlying source of “life” for Ortus is its respiratory circuit, which maintains a balance of O_2 and CO_2 . O_2 is consumed, and CO_2 is generated, as a product of Ortus being “alive”. As activation of the CO_2 sensory neuron, sCO_2 , rises, and activation of the O_2 sensory neuron, sO_2 , falls, motor neurons responsible for excitation and inhibition of the lung muscle are excited and inhibited (respectively), and the lung increases in activation, supplying O_2 and expelling CO_2 .

BR 1: Maintaining a given system concentration of CO_2 is fundamental to mammalian life, and is very strongly linked to the mammalian fear response (e.g., you get scared if you can’t breathe). The ability to regulate one’s level of CO_2 appears to at least be part of the basis for defining what is a “good” and “bad” event/stimulus in the mam-

malian brain.

ODE 2: Currently, Ortus has two “emotions”, fear and pleasure, represented by emotional interneurons, $eFEAR$ and $ePLEASURE$, which are both tied into the respiratory circuit. When CO_2 rises, $eFEAR$ ’s activation rises, and Ortus would then be in a fearful state. As CO_2 falls, its contribution to Ortus’ fearful state falls. The interaction between O_2 and $ePLEASURE$ is the same. In this way, any stimulus presented in combination with either increased CO_2 or increased O_2 will become known as either a desirable (good) or undesirable (bad) stimulus. Thus, it is via linking new stimuli to known stimuli, that emotions are the driving force behind associative learning in Ortus. In Ortus, the idea of “emotion” is simply the rise and fall of activation levels of different neurons or groups of neurons, tied to fundamental behaviors—such as “breathing”.

BR 2: Clearly, mammals don’t normally get scared when they exhale, nor do they feel a measurable increase in pleasure upon inhalation, however the relations do exist on some level. The concept of “good” and “bad” sensations or emotions only carry meaning to us because of their associations to other neural circuits that are either fundamentally desirable or undesirable from a longevity/survival perspective. As stated by Verma et al. (2015), “emotions, motivations, and reinforcement are a closely related, evolutionarily—conserved phenomena maintaining the integrity of an individual and promoting survival in a natural environment”. Taking this idea into account, along with work by Gore et al. (2015), which suggests that associative learning is funneled through innate behavior circuits to assign positive or negative emotions to neutral sensory stimuli, it seems that building a virtual organism driven by emotional states is a fairly sound approach.

While at the human level, the emotional “part” of the brain is quite complex, it is not unreasonable to assume that as organismic complexity (and thereby intelligence) decreases, the complexity of emotions decreases. Numerous experiments done on rodents, such as those described by Weiner et al. (2015), show that the major structures of the brain can be examined by lesioning portions of the brain. One can infer then, that representing regions of the brain by single neurons would enable a rough approximation of the region’s functionality. If one follows this line of thought, the possibility emerges that organisms like *C. elegans* may, in fact, be driven by “emotions” as well. For example, *C. elegans* is capable of toxin avoidance, a tap-withdrawal response, as well as learning that it found food at a certain temperature (Wicks et al., 1996; Xu and Deng, 2012) – one must ask how this can be. There is nothing external that assists it in differentiating good from bad, yet it wants to avoid certain things, while is attracted to others. In Ortus, we make the assumption that that these behaviors are a result of a *very* simple emotional subsystem that forms the basis for *C. elegans*’ behavior.

ODE 3: Ortus employs four different classes of interneurons. First are “Sensory Extension Interneurons” (SEIs). These take input directly from sensory neurons, and pass the input along to the second class of interneurons, “Sensory Consolidatory Interneurons” (SCIs). SCIs take anywhere from 1 to the number of sensory inputs as chemical synapses (CSs), with incoming synaptic weights equal to $1 / (\# \text{ sensory inputs})$. The idea behind SCIs is to enable different types of sensors to combine their input, and trigger emotions, effectively as a “new” sensory input, thereby forming associations between two stimuli. We defer the descriptions of the last two interneurons to **ODE 5**.

BR 3: Admittedly, SEIs may not be necessary. We included these to easily give sensory neurons a functionality that was separate from interneurons if the need arose. With regard to SCIs, it is suggested by Xie et al. (2016) that neurons in the brain are organized according to the idea that if there are N neurons, then the brain has the ability to represent all $2^N - 1$ possible combinations. Clearly, for anything but the most simple organisms, having one neuron that collects the input for each of the $2^N - 1$ possibilities is unrealistic (as the authors note). However, the authors suggest there may be additional combining of neuron inputs to decrease the computational and spacial complexity (in organic brains, that is). It also possible that mammalian brains are not quite as connected as they seem to think they are, and a great deal of what the brain does amounts to interpolation. Regardless, we employed the idea of $2^N - 1$ SCIs because losing sensory resolution with such few neurons (that only have one neuron per sensory input) doesn’t make sense. This will have to be reassessed as the system develops. There is, however, a far stronger argument for the strength of the SCI inputs: it seems that synaptic strength scales inversely with the number of connections, “ K as $1 / \sqrt{K}$ ” (Barral and D Reyes, 2016). This makes intuitive sense; if neuron A synapses onto B with a weight of 1, and C, D, E all synapse onto F, the weight of each of the latter three connections must be $\frac{1}{3}$ in order to maintain equivalence between B and F. In addition, there is evidence that neighboring neurons in the same “layer” are connected in *C. elegans* (Azulay et al., 2016), which suggests that a certain amount of information consolidation may occur.

ODE 4: Ortus currently implements both Hebbian learning and the Stentian extension to Hebbian learning. Specifically, for each chemical synapse, on every timestep, if activity is sufficiently synchronous or sufficiently asynchronous, the synapse strengthens or weakens (respectively) according to its *mutability index (MI)*. The *MI* determines a synapse’s potential to be modified. Currently, *MIs* are static, though in the future we plan to vary them with synaptic age, among other things.

BR 4: Hebbian and Stentian learning, relating to the correlation (or lack thereof) between presynaptic and postsynaptic pairs is a proven learning paradigm in neuroscience,

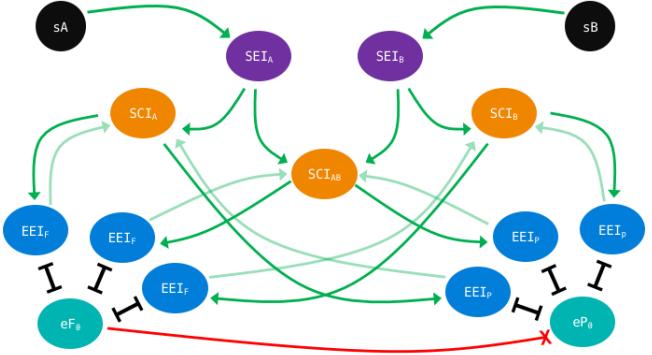


Figure 2: Diagram of Ortus’ emotional integration. Each color (other than black, which are sensory neurons) represents a specific layer of interneurons. SEIs are purple, SCIs are orange, EEIs are blue, and primary emotions (eF_0 and eP_0) are teal. Dark green arrows represent excitatory chemical synapses, while light green arrows represent weak chemical synapses. Black bars with flat caps on each end depict bidirectional gap junctions, and the red connection with an “X” at the end, symbolizes an inhibitory (“opposing”) relationship, in which not only does eF_0 inhibit eP_0 , but the group of EEIs inhibit the EEIs connected to their respective SCIs. This is the basic structure that Ortus employs. Currently there is no restriction regarding which interneurons can connect to motor neurons.

as described by Kutsarova et al. (2016).

ODE 5: The third class of interneurons is comprised of “Emotion Extension Interneurons” (EEIs), which are essentially auxiliary emotion neurons. EEIs are specific to each emotion, and receive input from SCIs via chemical synapses, and pass that input on to their respective primary emotion neurons via gap junctions (GJs). Emotional learning is achieved by strengthening synapses (via Hebbian learning) of interneurons that form synapses between SCIs (consolidated sensory information), and EEIs. The GJ connection enables a given emotional state, incurred by any stimulus, to permeate through Ortus—resulting in an “emotional state” of the system (gap junctions in Ortus have no activation threshold). The effect of this, is that if a certain sensory input causes a given emotion to increase in activation, the introduction of another sensory input will cause the synapses at the junction of the newly introduced sensory input and elevated emotion to strengthen. Further, as each SCI synapses onto one EEI per emotional state, each EEI has a fairly weak excitatory chemical synapse to their parent SCI. The idea behind the synapse from the EEI to the SCI is to allow an emotional state to “trigger”, or cause Ortus to “remember” a stimulus that previously invoked (and thus become associated with) that emotional state. In this case, “remembering” is defined as measurably increased activation in the SCI that

the EEI synapses onto. In practice, we have observed a very slight difference in Ortus as a result of these connections; currently they exist more as a concept that doesn't break the system. A diagram of this layout may be seen in Fig. 2.

BR 5: Specifically with regard to the mammalian dopamine circuit, there exists a small locus of dopaminergic cells, that receive inputs from diverse sources, and project to diverse parts of the brain, as discussed by Beier et al. (2015). From a psychological perspective, it is clear that when one enters an emotional state, the emotion is globally encompassing rather than localized. It is also clear that a stimulus or activity is likely to be “colored” by the emotional state one was in when the stimulus occurred. The approach we took with Ortus is a simplification of the dopamine circuit, and a generalization across emotional states; it will likely need refinement.

ODE 6: Certain emotional states preside over (dominate) others. In Ortus, fear dominates pleasure.

BR 6: The idea of a hierarchical system, where certain emotional states dominate others, is supported by the inhibition of fear in mice, in favor of searching for food when hungry (blood glucose levels falling causing the release of hormones), but greater concern for safety when not hungry (Verma et al., 2015). Further, Leknes and Tracey (2008) discusses the “Motivation–Decision Model”, which suggests that anything that is more important for survival than pain should inhibit the feeling of pain.

ODE 7: Ortus builds its nervous system by reading in a *.ort* file, using our “*Ortus Development Rules*” language. First, “elements” (neurons and muscles) must be specified, along with attributes such as the type of element, its affect, and activation threshold. Once all elements have been defined, relationships between elements may be specified. Currently, there are four relationships:

- [+-]A **causes** [+-]B

Where the “+”/“-” indicates an increase/decrease in the presynaptic neuron causes an increase/decrease in the postsynaptic element. This translates into a chemical synapse.

- A **correlated** B

Where A and B are correlated. This translates into a bidirectional gap junction.

- A **opposes** B

Where A and B inhibit each other.

- A **dominates** B

Where A inhibits B.

Relationship-level attributes may be specified, such as: “mutability”, “weight”, and “polarity”. At this time, most relationship attributes need not be specified as Ortus uses default values for mutability and weight, while polarity is determined from the relationships defined. Once Ortus reads

these instructions in, it creates interneurons and connections as described by the **ODEs** above to satisfy all constraints imposed, and to allow associative learning between all emotional states and sensory stimuli (both individual and combined) as described in **ODE 5**.

BR 7: Our goal in creating the *Ortus Development Rules* was to approximate, in a greatly simplified manner, the way a brain grows itself, based upon genetic instructions that cause circuits to form in certain ways via gene expression (Weiner et al., 2015). Ortus’ neural structure is essentially wired to force it to behave in certain ways, akin to the innate behaviors observed in mammals. Further, Schröter et al. (2017) provides evidence for the existence of organizational “motifs” found in *C. elegans* that may underly more complex networks in larger brains; this also lends credence to Ortus’ rule-based development approach.

Implementation Details

Ortus is written in C++ and OpenCL. Initial development of its neural structure, and sensory stimulation, are carried out through C++, while all signal transmission between neurons and learning is done in the OpenCL kernel. Each iteration of the OpenCL kernel constitutes one timestep, during which, each neuron sums incoming (positive or negative) “activation” from presynaptic cells via chemical synapses and gap junctions. We use the term “activation” in place of “potential” or “voltage” to make it clear that we aren’t dealing with the transfer of electrical current, but have abstracted that away to simply “activation”. The chemical synapse and gap junction activation transfer equations below were adapted and simplified from those described by Wicks et al. (1996) to ignore physical properties of neurons (such as neuron length), decrease computational complexity, and reduce potential sources of hard-to-trace error.

Ortus’ neurons are based upon *C. elegans*’ neurons, and are non-spiking, but do not transmit any “activation” below a given threshold, as described by Graubard (1978). The equations governing activation transmission between Ortus’ neurons are as follows (unless otherwise specified, A refers to the postsynaptic neuron, *i*):

$$A[m + 1] = (A[m] - A_{decay}) + A_{GJ_{in}} + A_{CS_{in}} \quad (1)$$

Where $A[m]$ and $A[m + 1]$ are the activations of neuron *i* we are working on for the current and next timestep, A_{decay} is the amount of activation lost by neuron *i* during the current timestep, and $A_{GJ_{in}}$ and $A_{CS_{in}}$ are the “incoming” activations to neuron *i* from gap junctions and chemical synapses (respectively).

$$A_{GJ_{in}} = GJ_{pre,post} \frac{(A_{pre}[m] - A[m])}{2} \quad (2)$$

Where $GJ_{pre,post}$ is the weight of the chemical synapse from the pre-synaptic to post-synaptic neuron, $A_{pre}[m]$ is

the presynaptic neuron's activation. We divide the activation difference by 2 because only half of the difference should move into the i^{th} neuron; pre will lose that half to i , and will keep the other half. This is analogous to pouring water into one of two water tanks joined at the bottom.

$$A_{CS_{in}} = CS_{pre,post}S_g(A_{rev} - A[m]) \quad (3)$$

Where $CS_{pre,post}$ is the weight of the chemical synapse from the pre -synaptic to $post$ -synaptic neuron, S_g is the synaptic conductance of synapse $CS_{pre,post}$, and A_{rev} is the reversal activation for the synapse (will be negative if an inhibitory synapse, and positive if excitatory).

$$S_g = \frac{1}{1 + e^{(-5\frac{A_{pre}[m]}{A_{range}})}} \quad (4)$$

Where A_{range} is the activation range of the presynaptic neuron (which is the difference between the excitatory and inhibitory reversal potentials). Eq. 4 uses -5 to vary the conductance from a little less than 10% to a little more than 90%. It should be noted that generally, we would subtract the equilibrium activation, A_{eq} , from $A_{pre}[m]$ prior to dividing by A_{range} , but this is not necessary because $A_{eq} = 0$. We use the presynaptic neuron's activation to determine the synaptic conductance because our neurons are non-spiking, and as such, activate their synapses with magnitude proportionate to the neuron's activation level (Wicks et al., 1996).

$$A_{decay} = (C_D A[m]) - A_{GJ_{out}}[m] \quad (5)$$

Where C_D is a constant decay percentage (set to 20%), and $A_{GJ_{out}}[m]$ is the amount of activation neuron i will lose from outgoing gap junctions.

$$A_{GJ_{out}} = \sum_{j=1}^N (GJ_{out,j} \frac{(A[m] - A_j[m])}{2}) \quad (6)$$

Where $GJ_{out,j}$ is the weight of neuron i 's j^{th} outgoing gap junction, N is the number of neurons, and $A_j[m]$ is the j^{th} neuron's activation. Note that to compute the activation that neuron i will lose due to its outgoing gap junctions, we treat it as the presynaptic neuron.

Learning

In order to implement biologically plausible Hebbian and Stentian learning, synchronous activity had to be locally determined, on a per-neuron basis. Each postsynaptic neuron computes a cross-correlation between its previous four activations, and all other neurons from the current time, to four timesteps back, as seen in Eq. 7. This allows a postsynaptic neuron A to determine how every other neuron is moving with respect to itself at the current time ($t = 0$), and how their activity may have influenced it, by assessing the level

of correlation between progressively earlier historical activations of the other neurons relative to itself. A high correlation at $XCorr[3]$, for example, would indicate that the $post$ neuron acted similarly to neuron j , three timesteps after neuron j acted that way.

Neurons also compute the slopes of the activations of all neurons, as shown in Eq. 8, which allows detection of a constant, or rapidly changing neuron's activation.

$$XCorr[t] = xcorr(H_{post}[0..3], H_j[t..t+4]) \quad (7)$$

Where $XCorr[t]$ is the t^{th} cross-correlation computation between $post$ and presynaptic neuron j , H is the activation history indexed by most recent activation at $H[0]$, and $xcorr$ is the cross-correlation computation.

$$Slope_j[t] = slope_u(H_j[t..t+u]) \quad (8)$$

Where $Slope_j[t]$ is the t^{th} slope computation for neuron j , u is the number of timesteps to use when computing the slope (set to 2), $slope$ computes the slope.

Combining the $XCorr$ and $Slope$ computation results, assuming both neurons involved have activations above the activation threshold (note that the maximum $XCorr$ value is 1):

- rapid strengthening occurs if there is a highly synchronous pair of signals nearly constant signals:

$$\sum_{i=1}^4 XCorr[i] \geq 3.92, \text{ and}$$

$$\sum_{i=1}^4 Slope[i] \leq .02$$

- synaptic strengthening occurs slowly if:

$$\sum_{i=1}^4 XCorr[i] < .05$$

- synaptic weakening occurs slowly if:

$$\sum_{i=1}^4 XCorr[i] > 3.5$$

Experimental Design and Results

Experiment. To test our initial implementation of Ortus, we implemented the configuration described above, with the addition of an H_2O sensory neuron, sH_2O . We then “exposed” Ortus to water via sH_2O stimulation four times in bursts spaced 100 timesteps apart, in order to allow injected activation to naturally decay. During each burst of H_2O exposure, we prevented Ortus from exhaling CO_2 or inhaling O_2 , thereby inducing an enhanced state of fear. After repeating the conditioning exposure four times, we allowed Ortus 200 timesteps to let any injected activation to decay. Finally, we exposed Ortus to water, without inducing a fearful state, to determine if it had learned to be fearful of water.

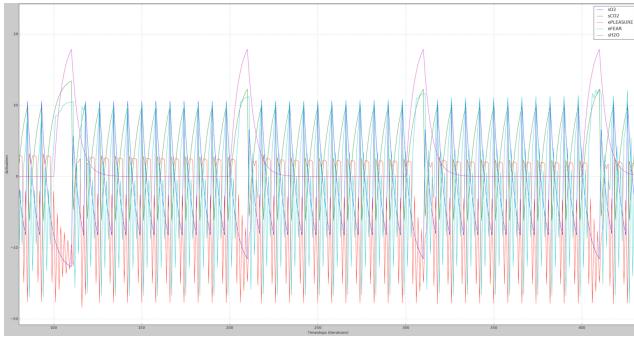


Figure 3: Ortus’ fear conditioning with CO_2 exhalation prevention and O_2 deprivation. Each time H_2O is introduced under these conditions, Ortus’ fear response grows, as evidenced by a strengthening response of eFEAR during each of the four rounds of conditioning.

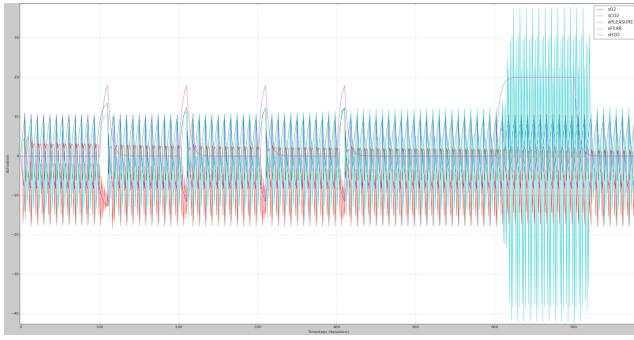


Figure 4: The final result of conditioning Ortus to fear H_2O . After the four rounds of conditioning, simply exposing Ortus to H_2O is enough to induce a state of fear.

Results. Since sCO_2 activation causes fear, and H_2O was presented (sH_2O was stimulated) during this time, we expected Ortus to become progressively more and more “scared” of water each time it was presented while it was in a state of elevated fear. As can be seen in Fig. 3, during each exposure to H_2O , activation of eFEAR increased when compared to the previous exposure. Further, after the four rounds of conditioning, we expected to observe an increase in activation in eFEAR when we presented Ortus with H_2O , in the absence of exhalation and inhalation restriction. Fig. 4 confirms this increase in fear activation as a result of sH_2O stimulation alone. Compared to simply exposing Ortus to sH_2O stimulation without prior fear conditioning, in which a very slight raise was observed—as may be seen in FIGURE, it is clear that we were able to successfully use classical conditioning to instill a fear of water in Ortus that was previously almost non-existent.

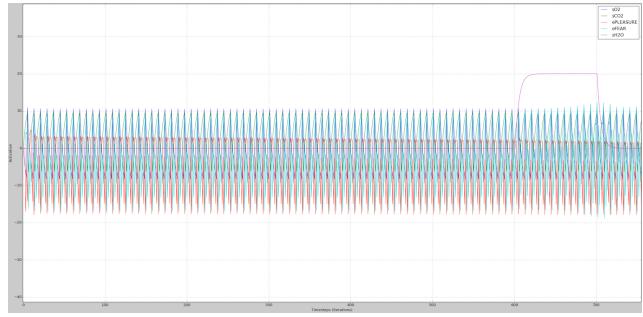


Figure 5: In the absence of fear conditioning, H_2O introduction causes a very subtle, slowly growing association with fear. This is likely due to instability in Ortus, and is an indication Ortus’ learning needs more nuance.

Discussion

Our initial experiments with Ortus show that it has great potential as a biologically-based framework for developing virtual life that exhibits complex intelligence. That said, there are a number of shortcomings that must be addressed in order for Ortus to grow much more complex than it currently is.

First, there is instability in the system. Further development, analysis, and experimentation of the emotional circuit(s) is necessary. The current approach is quite naive, and seems to play a role in the observable instability. A number of additional things could play a role in its instability as well; at this point, it is quite easy to throw Ortus out of balance. Small changes to the weights can have vast implications for the functionality of the system because an initial small change can multiply causing the system to become unstable. Johansen et al. (2014) suggests that in mice, while synchronous activity (correlation) is necessary for synaptic plasticity, it is not sufficient; neuromodulatory signaling is also required. This could be a way to control some of the unstable behavior seen as a result of the purely correlation-based learning rules implemented. In general, implementing more biologically-based constraints should help solve this, though refinement of the neural structuring rules is also likely necessary.

In fact, the neural structure needs refinement for combinatoric reasons anyway; in order to implement a sensor array (e.g., any sort of topographically ordered sensory inputs, like vision), using a “fully-wired” approach is completely unfeasible. Even 32 sensors would require over 4 billion neurons, if each possible combination of sensory inputs mapped to a different neuron. Schröter et al. (2017) suggests that C. elegans’ neurons may have multiplexed functions, meaning that one neuron may contribute to more than one behavior. This is another possible way to decrease the number of necessary neurons in Ortus.

Lastly, we would like to make Ortus’ learning far more

nuanced, and capable of picking up on more subtle changes than it currently can; though working on resolving the aforementioned issues should help the learning aspect as well. Additionally, Kutsarova et al. (2016) indicates that, relative to synaptic strengthening and weakening, axonal branch tips emerge to form new synapses, and that synchronous activity stabilizes synapses and prolongs axonal branches. In addition to refining Ortus' method for altering its synaptic weights, this is one more way for Ortus to modify its approach to learning. It also suggests there is biological precedent to implement a "floor" so that as Ortus learns new things, they can't be unlearned beyond a certain point.

Shortcomings aside, the results of our classical conditioning experiment with Ortus show that we successfully created a biologically-based virtual system that has a "self-sustaining" respiratory circuit, and is capable of Hebbian and Stentian learning. To suggest Ortus was virtually "alive" at any point during our experimentation seems as though it would be quite a stretch, however, once Ortus and similar systems increase in complexity, we must pose the question: when does something cross the boundary from computer program to sentient virtual being? When it can "feel"? What defines a "feeling" then? We have made the case that Ortus is emotion-driven, and have shown that we can both induce fear in it by interrupting its respiration cycle, and teach it to fear things it hadn't previously feared. If Ortus *can* feel, and isn't alive, what would it need to do in order to be alive? Alternatively, If Ortus *can't* feel, what would it take for a virtual organism to be able to "feel"?

Conclusion

We have presented Ortus, an initial approach to a framework that exhibits basic emotionally-based learning (fear conditioning), self-sustaining inherent behavior (cyclic breathing via a respiratory circuit), and approach to sensory processing that enables inter-sensory associative learning, with or without an emotional component. While the implementation of Ortus presented is quite simple, the design principles are solidly based in biology, and with further development and refinement, the potential exists for Ortus' development of (artificial) biological intelligence to quickly increase.

Acknowledgements

We thank Denisa Qori for her helpful thoughts, and Rachel Greenstadt for her prior guidance. This work was supported by the NSF GRFP.

References

- Azulay, A., Itskovits, E., and Zaslaver, A. (2016). The *C. elegans* Connectome Consists of Homogenous Circuits with Defined Functional Roles. *PLOS Computational Biology*, 12(9):e1005021.
- Barral, J. and D Reyes, A. (2016). Synaptic scaling rule preserves excitatoryinhibitory balance and salient neuronal network dynamics. *Nature Neuroscience*, 19(12):1690–1696.
- Beier, K. T., Steinberg, E. E., Deloach, K. E., Xie, S., Miyamichi, K., Schwarz, L., Gao, X. J., Kremer, E. J., Malenka, R. C., and Luo, L. (2015). Circuit Architecture of VTA Dopamine Neurons Revealed by Systematic Input-Output Mapping. *Cell*, 162(3):622–634.
- Bowren, J., Pugh, J., and Stanley, K. (2016). Fully Autonomous Real-Time Autoencoder-Augmented Hebbian Learning through the Collection of Novel Experiences. *Proceedings of the Artificial Life Conference 2016*, (Alife 15):382–389.
- DARPA (2017). Toward machines that improve with experience.
- Gore, F., Schwartz, E. C., Brangers, B. C., Aladi, S., Stujenske, J. M., Likhik, E., Russo, M. J., Gordon, J. A., Salzman, C. D., and Axel, R. (2015). Neural Representations of Unconditioned Stimuli in Basolateral Amygdala Mediate Innate and Learned Responses. *Cell*, 162(1):134–145.
- Graubard, K. (1978). Synaptic transmission without action potentials: input-output properties of a nonspiking presynaptic neuron. *Journal of Neurophysiology*, 41(4):1014–1025.
- Izquierdo, E. J. and Beer, R. D. (2016). The whole worm: Brain-body-environment models of *C. elegans*. *Current Opinion in Neurobiology*, 40:23–30.
- Jarrell, T. A., Wang, Y., Bloniarz, A. E., Brittin, C. A., Xu, M., Thomson, J. N., Albertson, D. G., Hall, D. H., and Emmons, S. W. (2012). The Connectome of a Decision-Making Neural Network. *Science*, 337(July):437–444.
- Johansen, J. P., Diaz-Mataix, L., Hamanaka, H., Ozawa, T., Ycu, E., Koivumaa, J., Kumar, A., Hou, M., Deisseroth, K., Boyden, E. S., and LeDoux, J. E. (2014). Hebbian and neuro-modulatory mechanisms interact to trigger associative memory formation. *Proceedings of the National Academy of Sciences*, 111(51):201421304.
- Kutsarova, E., Munz, M., and Ruthazer, E. S. (2016). Rules for shaping neural connections in the developing brain. *Frontiers in Neural Circuits*, 10(January):111.
- Leknes, S. and Tracey, I. (2008). A common neurobiology for pain and pleasure. *Nature Reviews Neuroscience*, 9(4):314–320.
- Marblestone, A. H., Wayne, G., and Kording, K. P. (2016). Towards an integration of deep learning and neuroscience. *bioRxiv*, 10(September):1–61.
- Roper, M., Fernando, C., and Chittka, L. (2017). Insect Bio-inspired Neural Network Provides New Evidence on How Simple Feature Detectors Can Enable Complex Visual Generalization and Stimulus Location Invariance in the Miniature Brain of Honeybees. *PLOS Computational Biology*, 13(2):e1005333.
- Schröter, M., Paulsen, O., and Bullmore, E. T. (2017). Micro-connectomics: probing the organization of neuronal networks at the cellular scale. *Nature Reviews Neuroscience*, 18(3):131–146.
- Sinapayen, L., Masumori, A., and Ikegami, T. (2016). Learning by Stimulation Avoidance: A Principle to Control Spiking Neural Networks Dynamics. page 17.

Verma, D., Wood, J., Lach, G., Herzog, H., Sperk, G., and Tasan, R. (2015). Hunger Promotes Fear Extinction by Activation of an Amygdala Microcircuit. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 41(2):431–439.

Weiner, J. A., Burgess, R. W., and Jontes, J. (2015). *Mechanisms of Neural Circuit Formation*.

Wicks, S. R., Roehrig, C. J., and Rankin, C. H. (1996). A dynamic network simulation of the nematode tap withdrawal circuit: predictions concerning synaptic function using behavioral criteria. *The Journal of neuroscience : the official journal of the Society for Neuroscience*, 16(12):4017–4031.

Xie, K., Fox, G. E., Liu, J., Lyu, C., Lee, J. C., Kuang, H., Jacobs, S., Li, M., Liu, T., Song, S., and Tsien, J. Z. (2016). Brain Computation Is Organized via Power-of-Two-Based Permutation Logic. *Frontiers in Systems Neuroscience*, 10(November):95.

Xu, J. X. and Deng, X. (2012). Complex chemotaxis behaviors of *C. elegans* with speed regulation achieved by dynamic neural networks. *Proceedings of the International Joint Conference on Neural Networks*, pages 10–15.