PERCEPTION THROUGH INTERACTION OF CELL ASSEMBLIES

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Abstract. We compare theories spanning more than 50 years to look for similarities and differences, and we find that the theories have much in common. We then look at a model of cell assemblies based on a system of ordinary differential equations and modified it to be more realistic. We run the model on a computer and show the phase portraits using a range of biologically significant parameters. The biological aspects are used as criterion to make the simplified mathematical models more realistic in terms of what might actually take place in the brain.

1. Introduction

Many believe cell assemblies are the fundamental structures in the brain through which we represent concepts, store and recall information, and form associations between concepts. A cell assembly is a group of neurons which are repeatedly active together and as a result of this repeated activation become associated [3]. Over time synaptic plasticity strengthens the synaptic weights so that the threshold, the number of connections to other excited neurons required for a cell to become excited, is lowered, making it easier for these associated neurons to excite each other. This synaptic plasticity is a result of learning, and it produces associative memory.

Hebb's original evidence for cell assemblies came from the discovery that perception involves areas of the brain other than just the visual cortex, which takes sensory input [3]. He studied patients who had been born blind but received surgery to correct their vision. After the surgery, the patients found it so difficult to learn how to process what they were seeing that many gave up [11]. Despite his argument that this study was evidence that cell assemblies form over time due to sensory input, the theory of cell assemblies was not accepted by the neuroscience community until many decades later. The main reason it was not accepted is that at the time that Hebb proposed it, scientists had different priorities in their research and did not deem it an important theory [11]. Neuroscientists were focused on single cell experiments and psychologists were focused on behavioral experiments.

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It was not until 2003, more than fifty years after Hebb proposed cell assemblies, that György Buzsáki discovered evidence of cell assemblies in the hippocampus by observing repeated synchronous firing of neurons beyond normal cell assembly expectations [1]. In the experiment, six male rats were implanted with either a 64-site silicon probe or several movable tetrodes, and they either collected food pellets in an open environment or walked on an elevated square track for a food reward. An LED was used to track the position of each rat. Buzsáki used a peer prediction method to ensure that the synchronous spiking was not due to coincidence. In the peer prediction method, each cell was assigned a weight, which increased or decreased the probability of synchronous spiking in the target cell. The results of the experiment showed a 25-ms timescale of synchrony, which matches the membrane time constant of pyramidal neurons in the hippocampal region as well as the period of the hippocampal gamma oscillation. Gamma oscillation is a pattern of brain waves around 40 Hz. Since that experiment, cell assemblies have become generally accepted [4].

The involvement of inhibition within and between cell assemblies began with the work of Hebb's colleague Milner, because Hebb accepted the belief of his time that there were only excitatory interactions between neurons [11]. By the time Milner wrote his paper in 1957, cortical inhibition had been observed, so he explained this phenomenon with the property that cells subjected to a constant source of excitation experience a decrease in frequency of discharge. Each cell will become fatigued, therefore contributing fewer impulses to the other neurons in the cell assembly. The inhibitory cells will also fire less, allowing cells outside the assembly to begin firing, and eventually the firing of one assembly will stop altogether and another assembly will take over [9].

In 2002 Scott used Milner's explanation of inhibition to derive a system of ODEs that could model interaction of cell assemblies for ambiguous perceptions. A program was written in MATLAB to model these ODEs while simulating the interaction between two competing cell assemblies of different perceptions of an optical illusion, the Necker cube. The objective of the work done this summer was an analysis of the interaction between multiple cell assemblies, which is an important step in better understanding the brain and how it stores and recalls memory.

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2. The Mathematics of Cell Assemblies

2.1. **Background: Palm.** In 1981, Palm applied a quantitative mathematical definition to Hebb's cell assemblies by considering them as graphs with the neurons as nodes of a graph and the connections between neurons as edges of a graph [10]. With this simple graph theoretical model, Palm defined a cell assembly to be a collection of neurons such that a sufficiently large subset of the collection will excite the whole collection. He based his model of neurons exciting each other on the idea that the neurons have a threshold that must be met or exceeded in order for the neuron to fire. Palm laid out a groundwork for how to find cell assemblies within the brain by looking at the connections between neurons, but few if any neuroscientists have studied and extended his work. Therefore, the first objective of the summer was to understand Palm's work on graph theoretical cell assemblies and see how it could be implemented in MATLAB.

2.2. **Definitions.** Let us define $G(V, E)$ to be a graph with a set of vertices and edges. Let $A(V') \subset G(V, E)$ s.t. $V' \subset V$. Let $\mathcal{P}(G)$ be the power set of $G(V, E)$, i.e. the set of all subsets of $G(V, E)$. Let $c(u, v)$ be a binary function for the connection between two vertices $u, v \epsilon V$: $c(u, v) = 1$ if $\exists$ an edge between them, and $c(u, v) = 0$ if $\nexists$ an edge between them. Let k be the threshold of excited inputs a neuron needs in order to become excited.

- excitation mapping $e : \mathcal{P}(G) \to \mathcal{P}(G)$ s.t.
  the image of A is $e(A) = \{v \epsilon V : \sum_{u \epsilon V'} c(u, v) \geq k\}$

This mapping reflects that when a certain subgraph A of graph G is excited, it will cause another subset to become excited. For our purposes, a neuron (vertex) will become excited if it receives input from 2 excited neurons. The subgraph induced by the j^{th} iteration of the mapping e will be denoted as $e^j(A)$.

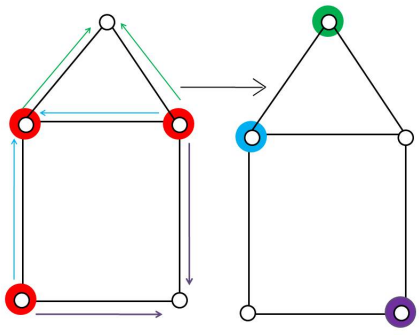


Figure 1. Excitation mapping

In Figure 1 set G is the entire connected figure of 5 vertices and 6 edges, A is the set of 3 vertices circled in the figure on the left, and the arrows point to which vertices will be excited by the mapping e because they are receiving 2 or more excited inputs. The figure on the right shows the set $e(A)$ as the 3 vertices that are now circled.

Figures 2-6 show examples of the following 5 definitions.

- persistent: $A \subseteq e(A) \subseteq \ldots \subseteq e^j(A)$

This means the excited portion re-excites itself, plus possibly other cells in the brain.

- invariant: $A = e(A) = e^2(A) = \ldots = e^j(A)$

The excited portion excites only itself over and over.

- weak: $\exists n$ s.t. $e^n(A) = \emptyset$

The excited portion was not sufficient to keep any portion of the graph excited after a finite number of iterations, so the activity would die out completely.

- tight: a persistent set s.t. for each persistent subset either the complement is weak or the subset excites the entire set

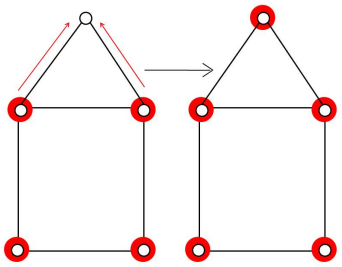


FIGURE 2. Persistent

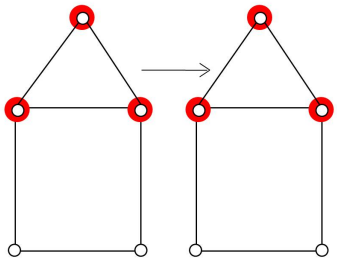


FIGURE 3. Invariant

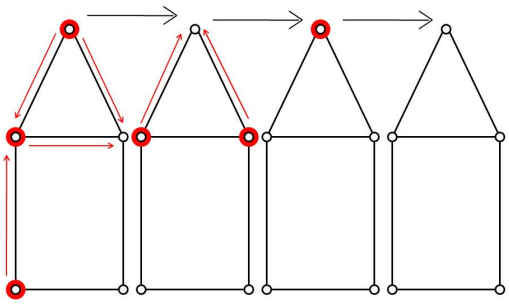


FIGURE 4. Weak set

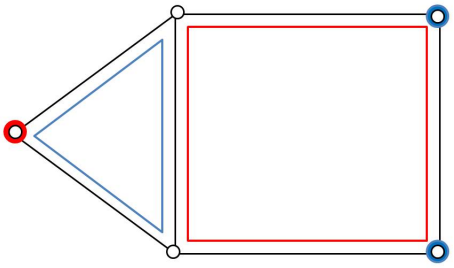


FIGURE 5. Tight set; each color shows a persistent subset and its complement

- closure: $\exists n$ s.t. $e^n(A) = e^{n+1}(A) = \dots$

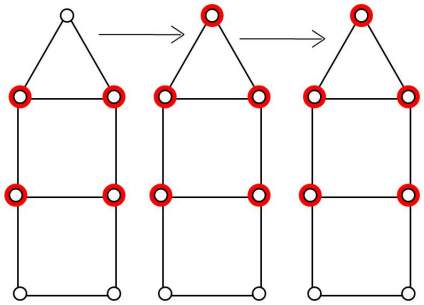


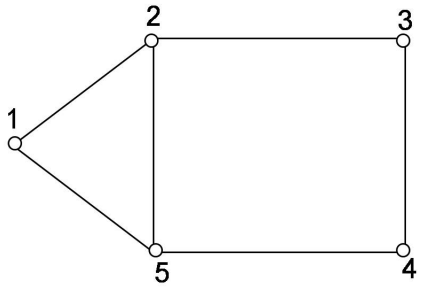
FIGURE 6. Closure of a set

In order to find one of Palm's cell assemblies in a given random network of interconnected neurons, one must find a tight set and then find its closure. Being able to find cell assemblies in the brain could give further insight into how memory works.

2.3. **Pseudocode to determine if a set is tight.**

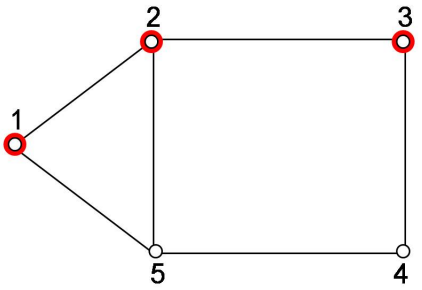
```
is set persistent?
    if no, set is not tight, stop
    if yes, find all persistent subsets
        for each persistent subset, is complement weak?
            if yes, set is tight, stop
            if no, does persistent subset excite entire set?
                if no, set is not tight, stop
                if yes, set is tight, stop
```

2.4. **Further work on Palm.** We wrote MATLAB code which applies the excitation mapping to a given network. It takes as input an adjacency matrix describing which neurons are connected to which, an excitation vector describing which neurons are excited initially, and a threshold to determine which neurons would be excited once the mapping was applied. The program output is a vector describing which neurons belong to $e(A)$.
The following figure:



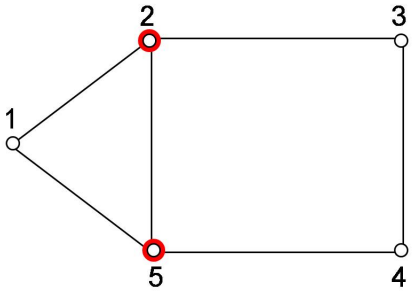
corresponds to the adjacency matrix: $A = \begin{pmatrix} 0 & 1 & 0 & 0 & 1 \\ 1 & 0 & 1 & 0 & 1 \\ 0 & 1 & 0 & 1 & 0 \\ 0 & 0 & 1 & 0 & 1 \\ 1 & 1 & 0 & 1 & 0 \end{pmatrix}$

The following figure:



corresponds to the excitation vector: $v = \begin{pmatrix} 1 \\ 1 \\ 1 \\ 0 \\ 0 \end{pmatrix}$. The output will be $w = \begin{pmatrix} 0 \\ 1 \\ 0 \\ 0 \\ 1 \end{pmatrix}$

which corresponds to the figure:



For the full code, see Appendix A.

2.5. **Importance of Palm.** Palm's translation of cell assemblies into graphs with nodes and edges is a very useful tool for understanding cell assemblies through visual representations. With this system of drawing cell assemblies, one can see precisely which cells have sufficient inputs to become excited and which subsets of a system will excited which other subsets. Hebb drew his own crude representation of a simplified cell assembly, shown in Figure 7, but it was much more difficult to interpret.

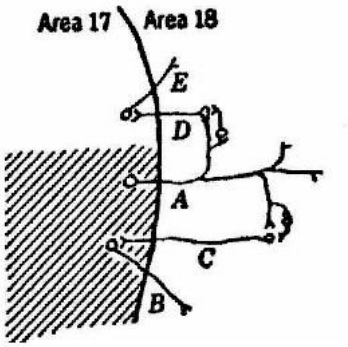


FIGURE 7. Hebb's cell assembly

3. Physiological Relevance of Cell Assemblies

3.1. **Cell Assemblies according to Hebb.** Hebb describes many ways in which cell assemblies might be utilized in our brains. First is pattern completion; that is, if one has repeatedly seen some whole figure and then one sees a part of it, the cell assembly that has been created to store that figure through synaptic plasticity will call to mind the entire figure.

Example:

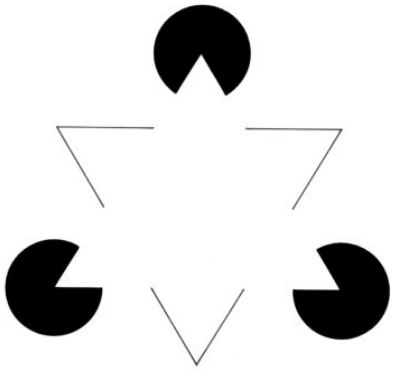


FIGURE 8. Perception of a triangle

There is no triangle in Figure 8, but we perceive there to be one because we have seen so many triangles in our lives that the repeated stimulation of triangles has caused synaptic plasticity to occur, forming a cell assembly that is recalled by seeing only the suggestion of corners. Pattern completion due to our triangle cell assembly fills in the rest of the figure for us.

He also uses cell assemblies to explain the fact that the feeling of discomfort we experience when we are hungry gives us motivation to seek food. Initially this discomfort only causes restlessness and purposeless movement, and the association between the discomfort and putting food in one's mouth must be learned over time from being fed by someone who has already learned this association. Further support for hunger as a cell assembly comes from empirical evidence that when one is deprived of food for a sufficient period of time, one loses the desire to eat. This could be explained with cell assemblies by the fact that lack of repeated stimulation to reinforce the association between the discomfort and the seeking of food could cause that association to break down. This same phenomenon is seen with stored memories: if a memory is not recalled for a long period of time, it is much harder to remember exactly what happened than a memory of which one is reminded often.

3.2. **Connection to Marr's Codon Formation.** Two decades after Hebb proposed cell assemblies, Marr introduced the codon representation concept [7], in which a subset of activated mossy fibers in the cerebellum may excite a granular cell upon exceeding a threshold of the number of mossy fibers required to excite a granular cell. Mossy fibers are axons of neurons in the cerebellum whose cell bodies lie in the nuclei in the spinal cord and brain stem and which carry sensory information to the granule cells in the granular layer [5]. Granule cells are neurons that take many mossy fibers as input and have very long axons

which synapse with many Purkinje cells. Purkinje cells are very large relative to the other neurons in the cerebellum and provide inhibitory output from the cerebellum to regulate movement. Marr also extended the concept of codon representation to codon formation in the hippocampus, where simple memory is stored [8]. Codon formation in the hippocampus is the idea that the codons that form according to which granule cell they excite provides evidence for whether a cell has previously been excited by a subevent similar to the current one.

Up to now, there has been no connection between Marr's codon concept and Hebb's cell assemblies. At first glance there appear to be many parallels between Marr's theory and Hebb's theory. Marr died at a young age without linking his theories to cell assemblies, so it is unknown whether he ever intended to do so, but an attempt at synthesis between the two theories may yield a better understanding of the structure of the brain. In our project we closely studied the two theories to determine whether there exists a close correspondence between them in order to further our analysis of the working of cell assemblies. The motivation for this connection comes from the observation of these similarities and the desire to learn as much as possible about cell assemblies.

Marr's cortical theory states that the brain is able to track of large populations of synchronously active, excitatory, neocortical pyramidal cells such that when a subset of those cells fires later on, the entire population will activate [8]. He uses this theory to explain associative memory in the hippocampus. Keeping track of which cells are active would require a pre-existing structure in which the neurons must already be placed correctly since storage of associations would rely heavily on connections between the neurons. This keeping track could be done by assigning a binary function of discrete time to each neuron, the value depending on whether the cell is excited or not. He calls such an assignment an "event."

Marr also discusses a process called codon representation. Codons are subsets of populations of active mossy fibers in the cerebellum. Codon representation is defined as the representation of a mossy fiber input by a collection of codons. In other words, the entire set of mossy fibers could be described as a collection of subsets of mossy fibers, each of which fires a granule cell [7]. Codon representations allow mossy fiber collections to be understood in smaller components with a more concrete biological significance. That is, each codon excites a granule cell, which in turn excites a Purkinje cell once enough granule cells are excited. He also extends the idea of codon representation to apply to any subset of cells which, when active, excites another neuron [8].

There are many similarities to Hebb's idea of cell assemblies: the general idea of both theories is that a collection of interconnected neurons is used to store associative memories and when exciting a subset of these neurons the rest are excited. However, Marr did not explicitly link the two concepts despite referencing Hebb's work. So is Marr's theory related to cell assemblies, and if so, to what extent?

3.3. **Examination of the two theories.** The first piece of evidence in favor of a relationship between cell assemblies and codons is the fact that both require thresholds to be traversed in order for firing to occur. In the case of cell assemblies, the threshold required is a minimum number of incoming connections each neuron must receive in order to fire; in the case of codons, the threshold is how many neurons are needed in one codon to fire one codon cell, i.e. the minimum size of the codon. Marr gives a very rigorous algorithm for determining how many granule cells will synapse with one Purkinje cell given a codon of size L, granule cells with C claws, and threshold R. Here threshold can be different from the size of the codon because it is a minimum requirement, and it will generally be much smaller than the size of the codon because it depends on the number of claws a granule cell has. A claw of a granule cell is a short dendrite (input) which has a claw-shaped end; on average a granule cell has four or five claws.

In addition, both ideas rely on synaptic plasticity, the ability to modify the connection strength over time. According to Hebb's theory, synaptic weights gradually change such that the cells can excite each other at a lower threshold, thus creating the associative memory. Marr's theory relies on plastic synapses as well, although codons require a Brindley synapse. A Hebb synapse is initially ineffective but is facilitated by simultaneous pre- and post-synaptic activity. A Brindley synapse is a type of Hebb synapse that contains an unmodifiable excitatory component. That is, there is a baseline that the weight cannot go below, as opposed to the Hebbian which begins with a weight of zero. The Brindley synapse has a variable post-synaptic threshold in order for modification to be possible. Although Marr has come up with a new kind of synapse since Hebb wrote his theory, the idea of synaptic plasticity is the same, so this does not appear to refute an argument that cell assemblies and codons could be the same structures.

Strong evidence connecting the two theories comes in Marr's assertion that recalling an "event" is initiated by recalling a "subevent." An event is a particular assignment of which neurons are active and which are inactive simultaneously within a given set of neurons. A subevent is such an assignment within a subset of the given set of neurons. Likewise, in Palm's refinement of Hebb's cell assemblies a set of neurons is a cell assembly if every

sufficiently large subset of the set excites the whole set. From the biological point of view, this means that if a person is reminded of one or a few aspects of a concept, everything he knows about the concept will be recalled.

Codon formation in the hippocampus, which provides evidence that the same cell has fired for similar events, is similar to Hebb's idea that cell assemblies can form pattern completion so that if an event occurs that is similar to one already stored in a cell assembly, the same assembly will become excited. Hebb used this idea to explain holistic visual perception of a figure [6]. One of Hebb's main examples of holistic perception is learning to identify a triangle by looking at each of its three corners individually, as seen in the above example in Figure 8.

Both theories deal with the idea that the cells required for the respective processes are divided into several different populations of cells, and one population is connected to the next through its axons, which in turn connects to the next with its axons. Hebb specifies that the first population in this process is in Area 17, the visual cortex (see Figure 7). He noticed that from the visual cortex there is a diffusion of conduction to other areas so that neurons that are close together in one area may connect to neurons that are very far apart in another area. Marr does not specify what the different regions are that connect to each other, only that they are all pyramidal cells.

There is a potentially significant difference between the two theories. In Marr's theory, the set of input pyramidal cells excites a set of codon cells which is entirely disjoint from the set of input cells, and the set of codon cells in turn excites the set of input cells. This seems similar to Palm's graph theoretical idea of a mapping e from one subset of neurons to another subset within a graph of interconnected neurons, but according to Palm's theory of cell assemblies, a cell assembly must excite itself, and possibly other neighboring neurons depending on the threshold. If the input set is exciting the codon set and these two sets are disjoint, then is seems that this is not a cell assembly according to Palm's theory. However, since Palm's graph theoretical approach does not take any sense of timing into consideration, it is possible that Marr's codon cells looping back to excite the codons is the same concept as Palm's persistent set, a collection of neurons which when excited, excites all cells in itself plus possible other cells outside of the collection. When codons are excited, they get excited again as well as exciting the codon cells. Therefore, it is possible that these two concepts are not so conflicting after all.

From this examination it would seem that codon formation corresponds well with cell assemblies. Therefore, we can conclude that they are in fact slightly different treatments of the same concept within the hippocampus. This will help to incorporate Marr's biological

knowledge of how many and what type of neurons are involved in codons into Hebb's cell assembly for a more accurate model of what goes on within the brain.

4. Phase sequences, inhibition, and simulation

4.1. **Phase sequences.** One important extension Hebb made of his own cell assembly theory is the theory of phase sequences. A phase sequence is a series of cell assemblies such that the activity of one facilitates the activity of others. A phase sequence corresponds to a complex perception such as use of a tool or interpreting spoken language, or it could be used to explain a train of thought connecting one memory of concept. Hebb knew that phase sequences would require some mechanism by which to suppress one another from firing, but as mentioned previously, he did not know about inhibition.

4.2. **Scott's differential equations.** Scott proposed a system of differential equations to model the interactions between two cell assemblies similar to a predator-prey model [11]. He used the model of a neuron, which gives an output signal when its receives input greater than or equal to a threshold, to derive the probability that a given neuron will fire in the next increment of time:

$$P(F) = \sum_{j=\theta}^{I} \binom{I}{j} F^j (1-F)^{I-j}$$

where F =fraction of neurons firing within a cell assembly, I =number of input connections to each neuron, which may or may not be excited, and θ =threshold for neuron to give output. He made assumptions that $I = 2$ and $\theta = 1$, which yields the equation:

$$P(F) = \sum_{j=1}^{2} \binom{2}{j} F^j (1-F)^{2-j}$$

$$\Rightarrow P(F) = 2F(1-F) + F^2(1-F)^0$$

$$\Rightarrow P(F) = 2F - F^2$$

Since $P(F)$ shows the level of activity at a time $t + \tau$, we can approximate

$$\frac{dF}{dt} = \frac{P(F) - F}{\tau}$$

$$\Rightarrow \frac{dF}{dt} = 2F - F^2 - F = F - F^2$$

$$\Rightarrow \frac{dF}{dt} = F(1-F)$$

So the system of differential equations for two cell assemblies interacting with each other is:

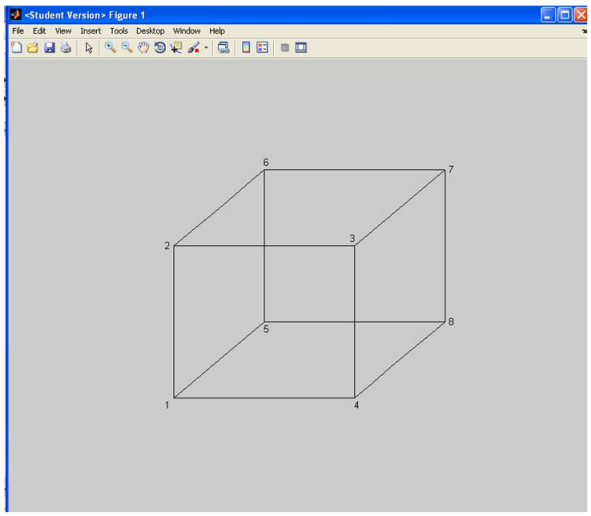
$$\frac{dF_1}{dt} = F_1(1-F_1) - \alpha F_2$$

$$\frac{dF_2}{dt} = F_2(1 - F_2) - \alpha F_1$$

where α is the inhibition constant. Several phase planes were plotted for different values of α to determine an optimal value that would ensure that one of the cell assemblies always went to 0 percent ignited within a reasonable amount of time to model the interaction between two cell assemblies as realistically as possible with the given resources and knowledge. Figures 10-13 in Appendix B show the phase planes graphed for different values of α.

When $\alpha < 0.333$ (Figures 10,11) there is a sink in the phase plane, which will cause the solutions to the two ODEs to converge instead of diverge, corresponding to the two cell assemblies becoming equally excited instead of one winning out over the other. In order to have α sufficiently large for one assembly to become completely excited while the second assembly completely dies out within a reasonable window of time, we use $\alpha = 0.75$ (Figure 13) for the MATLAB simulation.

The simulation shows the following image when the program is run:



The Necker cube is a common optical illusion designed by Louis Albert Necker in 1832. The command prompt then directs the user: "Type the number of the first corner that caught your eye." The user's input corresponds to certain initial conditions which the program uses to approximate solutions to the differential equations, and it outputs a plot of what fraction of cells in each assembly are ignited versus time. Some output is shown in Figure 9.

4.3. **Problems with Scott's differential equations.** A significant problem with Scott's differential equations is that his assumptions for the number of inputs per neuron ($I = 2$)

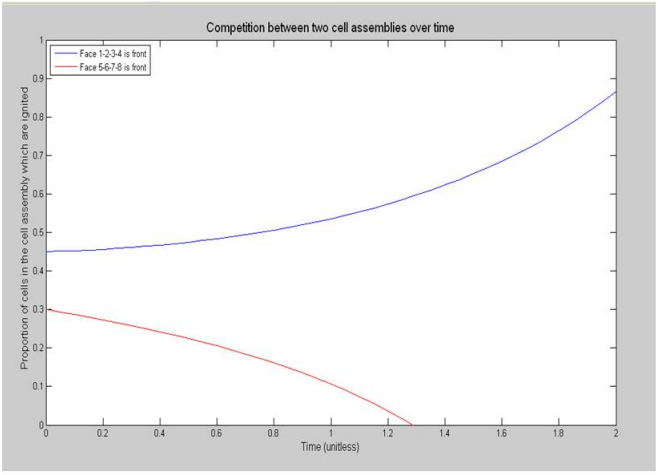


FIGURE 9. Graph outputted if user chooses Corner 2

and the threshold for the neurons to fire ($\theta = 1$) are unrealistic. Palm's graphs generally use $I = 3$ and $\theta = 2$, and even these numbers are too low [5]. Using these parameters from Palm, we obtain the equations:

$$P(F) = -2F^3 + 3F^2$$

$$\frac{dF}{dt} = -2F^3 + 3F^2 - F$$

$$\frac{dF_1}{dt} = -2F_1^3 + 3F_1^2 - F_1 - \alpha F_2; \frac{dF_2}{dt} = -2F_2^3 + 3F_2 - F_2 - \alpha F_1$$

Figures 14-16 in Appendix B show the phase planes for different values of α. Note that for any $\alpha > 0$ (Figures 15,16) the solutions of $F_i(t)$ go outside the biologically possible range of values of 0 to 1. This same problem is encountered with Scott's original equations.

To improve the equations, it is sufficient to look to the predator-prey equations, which will be bounded from below by 0 because a population cannot become negative. The term in which the predator-prey model differs from this cell assembly interaction model is an "interaction" term $-\alpha F_1 F_2$ in both equations instead of $-\alpha F_2$ in one equation and $-\alpha F_1$ in the other. Therefore, a new system of equations was developed with an interaction term:

$$\frac{dF_1}{dt} = F_1(1 - F_1) - \alpha F_1 F_2$$

$$\frac{dF_2}{dt} = F_2(1 - F_2) - \alpha F_1 F_2$$

Figures 17-24 in Appendix B show the phase plane and particular solutions with initial conditions $F_1(0) = 0.3$ and $F_2(0) = 0.45$ for each value of α. Note that for $\alpha < 1.0$ the solutions converge instead of diverging, which is equivalent to the two cell assemblies equalizing their levels of excitation instead of competing until one becomes completely excited and the other completely dies out. Note also the time scale for $\alpha = 1.1$ versus $\alpha = 1.9$. The former does not level out until $t = 68$ whereas the latter levels out at $t = 10$. From $\alpha = 1.9$ to $\alpha = 5.0$ there is not as significant a decrease in the amount of time. It is to be expected that the shorter amount of time would make for a more realistic model.

One final adjustment to these equations was made to incorporate a "run-down" term, which accounts for the fatigue a cell assembly experiences after it has been excited for a period of time. So the final system of equations with an interaction term and a run-down term is:

$$\frac{dF_1}{dt} = F_1(1 - F_1) - \alpha F_1 F_2 - \beta F_1$$

$$\frac{dF_2}{dt} = F_2(1 - F_2) - \alpha F_1 F_2 - \beta F_2$$

However, this $-\beta F_i$ term lengthens the time required for one cell assembly to become completely excited and the other to completely die out. Further work will be needed to determine a good balance of the parameters.

5. Conclusion

Perception is a major aspect of our lives and determines how we react to our environment in any situation. Cell assemblies may provide the key to understanding perception, and their interactions may explain how a single perception is favored when a figure is ambiguous. This summer we read several theories on cell assemblies and storage of memory. We then compared their similarities and differences and argued that their differences are far more minor than their similarities. We found an ODE model that built on a few of the theories and improved it to incorporate biological factors that Scott had not incorporated. Cell assemblies connect neurons in the visual cortex to other areas of the brain and build phase sequences to comprehend complex concepts such as using tools and understanding spoken language. Cell assemblies can be explained to an extent using graph theory as sets of nodes and edges. This graph theoretical approach helps to visualize cell assemblies in a way that is useful when discussing them, and to identify cell assemblies, an important step to further understanding the brain. Cell assemblies can be more accurately described with mathematics when inhibition is taken into account, as with Scotts system of differential

equation for two competing cell assemblies. Although his model has been improved with this work, further work is needed to make it as close to actual activity in the brain as possible.

Appendix A: MATLAB Code

```
% Name: lightprog.m
% Example: lightprog([0 1 1;1 0 1;1 1 0],[1;1;0],2)
% This function applies 'e' to the given network
% input:A, the adjacency matrix, v, the vector of what is excited, t, the
% threshold
% output: w, the vector of what is excited after "f" is applied

function lightprog(A,v,t)
w=ones(length(A),1);
mult=transpose(A)*v;

for i=1:length(A)
    if mult(i)>=t
        w(i)=1;
    else w(i)=0;
    end
end
if w==ones(length(A),1)
    disp(['You excited everything!'])
elseif w==zeros(length(A),1)
    disp(['Your set is weak.'])
end

disp(w)
a=input('Type 1 to apply f again.  Type any other number to quit. ');
ct
if a==1
    v=w;
    lightprog(A,v,t);
    ct=ct+1;
end



% Name: neuron.m
% example: neuron(0.5,[-1 1],.75)
% this program solves the system of ODEs for a particular time t
% arguments: t=particular time you want, j=row vector of the initial values,
% a=inhibition constant (must be >1/3)

function djdt=neuron(t,j,a)
```

```
djdt(1,1)=j(1)*(1-j(1))-a*j(2);
djdt(2,1)=j(2)*(1-j(2))-a*j(1);



% Name: necker_cube.m
% this program shows the necker cube, takes an input of which corner
% the subject observed first, and based on the initial conditions
% specific to that corner, solves a system of 2 ODEs to predict which
% necker cube cell assembly is "stronger"
x=[0:.1:1]; y=x*0; z=x*0+1;
a=[.5:.1:1.5]; b=a*0+0.5; c=a*0+1.5;
d=[0:.1:.5]; e=d; f=d+1;
g=(1:.1:1.5); h=g-1; i=g;

plot(x,y,'k-',y,x,'k-',x,z,'k-',z,x,'k-',a,b,'k-',b,a,'k-',a,c,'k-',c,a,
        'k-',d,e,'k-',d,f,'k-',g,h,'k-',g,i,'k-')
axis([-0.5 2 -0.5 2])
axis off

text(x(1,1)-.05,x(1,1)-.05,'1')
text(x(1,1)-.05,x(1,11),'2')
text(x(1,11)-.025,x(1,11)+.05,'3')
text(x(1,11),x(1,1)-.05,'4')
text(x(1,6),x(1,6)-.05,'5')
text(x(1,6),g(1,6)+.05,'6')
text(g(1,6)+.025,g(1,6),'7')
text(g(1,6)+.025,x(1,6),'8')
node=input('Type the number of the first corner that caught your eye: ');

if node==1
    ICLeft=0.3;
    ICRight=0.2;
elseif node==2
    ICLeft=0.45;
    ICRight=0.3;
elseif node==3
    ICLeft=0.75;
    ICRight=0.5;
elseif node==4
    ICLeft=0.6;
    ICRight=0.4;
elseif node==5
    ICLeft=0.5;
    ICRight=0.75;
```

```
elseif node==6
    ICLeft=0.3;
    ICRight=0.45;
elseif node==7
    ICLeft=0.2;
    ICRight=0.3;
elseif node==8
    ICLeft=0.4;
    ICRight=0.6;
end

[t j]=ode45(@neuron,[0 2],[ICLeft ICRight],[],.75);
figure(1)
plot(t,j(:,1),'b-',t,j(:,2),'r-')
axis([0 2 0 1])
axis on
xlabel('Time (unitless)','FontSize',12)
ylabel('Proportion of cells in the cell assembly which are ignited','FontSize',12)
title('Competition between two cell assemblies over time','FontSize',14)
m = legend('Face 1-2-3-4 is front','Face 5-6-7-8 is front',2);
set(m,'Interpreter','none')



% Name: neuron_revised.m
% this program solves the system of ODEs for a particular time t
% arguments: t=particular time you want, y=row vector of the initial values,
% a=inhibition constant (must be >1/3)
% this program differs from neuron.m in that it uses different ODEs which
% correspond to different biological parameters (number of afferent inputs
% and threshold required to give output)

function djdt=neuron(t,j,a)
djdt(1,1)=-2*(j(1))^3+3*(j(1))^2-j(1)-a*j(2);
djdt(2,1)=-2*(j(2))^3+3*(j(2))^2-j(2)-a*j(1);
% djdt(1,1)=3*(j(1))^4-8*(j(1))^3+6*(j(1))^2-j(1)-a*j(2);
% djdt(2,1)=3*(j(2))^4-8*(j(2))^3+6*(j(2))^2-j(2)-a*j(1);
% djdt(1,1)=j(2);
% djdt(2,1)=j(1);



% necker_revised.m
% this program shows the necker cube, takes an input of which corner
% the subject observed first, and based on the initial conditions
```

```
% specific to that corner, solves a system of 2 ODEs to predict which
% necker cube cell assembly is "stronger"
% this program differs from necker_cube in that it uses the ODEs from
% neuron_revised

x=[0:.1:1]; y=x*0; z=x*0+1;
a=[.5:.1:1.5]; b=a*0+0.5; c=a*0+1.5;
d=[0:.1:.5]; e=d; f=d+1;
g=(1:.1:1.5); h=g-1; i=g;

plot(x,y,'k-',y,x,'k-',x,z,'k-',z,x,'k-',a,b,'k-',b,a,'k-',a,c,'k-',c,a,
        'k-',d,e,'k-',d,f,'k-',g,h,'k-',g,i,'k-')
axis([-0.5 2 -0.5 2])
axis off

text(x(1,1)-.05,x(1,1)-.05,'1')
text(x(1,1)-.05,x(1,11),'2')
text(x(1,11)-.025,x(1,11)+.05,'3')
text(x(1,11),x(1,1)-.05,'4')
text(x(1,6),x(1,6)-.05,'5')
text(x(1,6),g(1,6)+.05,'6')
text(g(1,6)+.025,g(1,6),'7')
text(g(1,6)+.025,x(1,6),'8')
node=input('Type the number of the first corner that caught your eye: ');

if node==1
    ICLeft=0.3;
    ICRight=0.2;
elseif node==2
    ICLeft=0.45;
    ICRight=0.3;
elseif node==3
    ICLeft=0.75;
    ICRight=0.5;
elseif node==4
    ICLeft=0.6;
    ICRight=0.4;
elseif node==5
    ICLeft=0.5;
    ICRight=0.75;
elseif node==6
    ICLeft=0.3;
    ICRight=0.45;
elseif node==7
    ICLeft=0.2;
```

```
    ICRight=0.3;
elseif node==8
    ICLeft=0.4;
    ICRight=0.6;
end

[t j]=ode45(@neuron_revised,[0 2],[ICLeft ICRight],[],.5);
figure(1)
plot(t,j(:,1),'b-',t,j(:,2),'r-')
axis([0 2 0 1])
axis on
xlabel('Time (unitless)','FontSize',12)
ylabel('Proportion of cells in the cell assembly which are ignited','FontSize',12)
title('Competition between two cell assemblies over time','FontSize',14)
m = legend('Face 1-2-3-4 is front','Face 5-6-7-8 is front',2);
set(m,'Interpreter','none')
```

Appendix B: Phase Planes and Particular Solutions

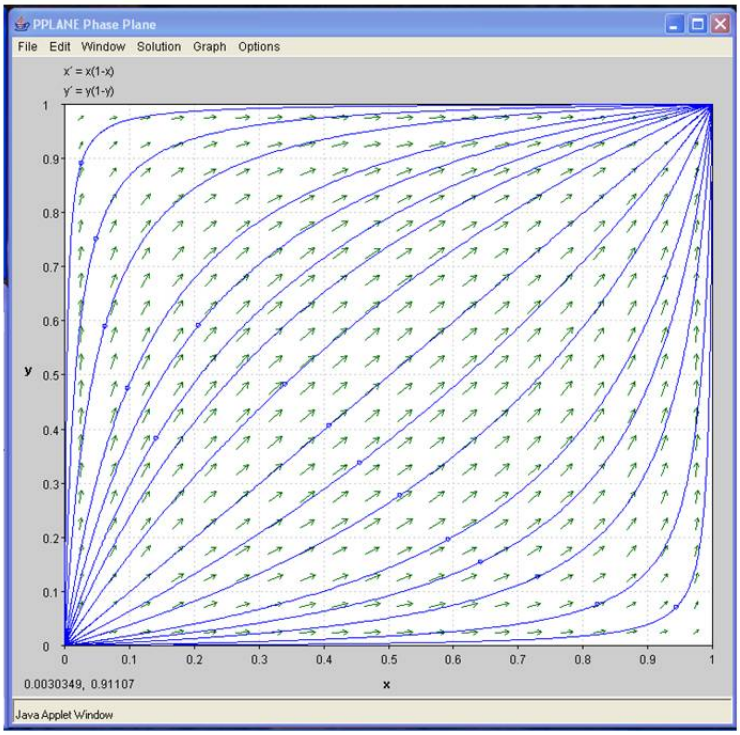


FIGURE 10. $\alpha = 0$

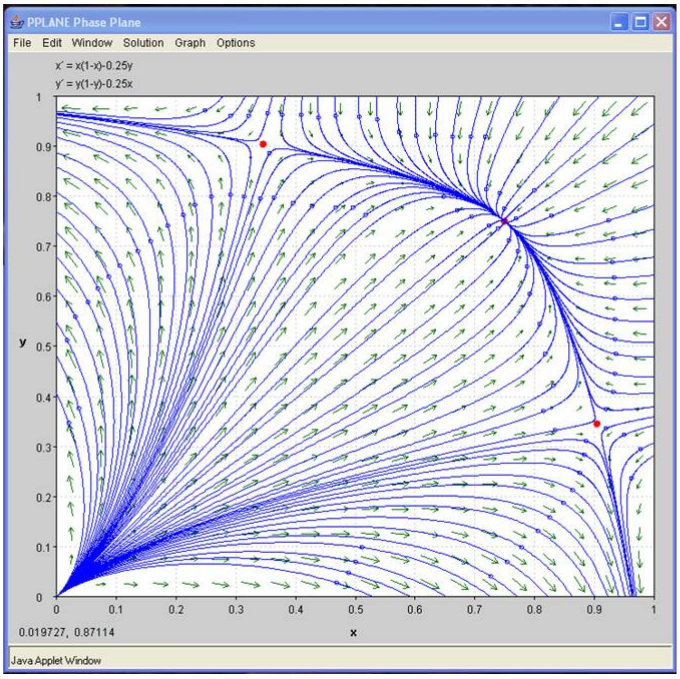


FIGURE 11. $\alpha = 0.25$

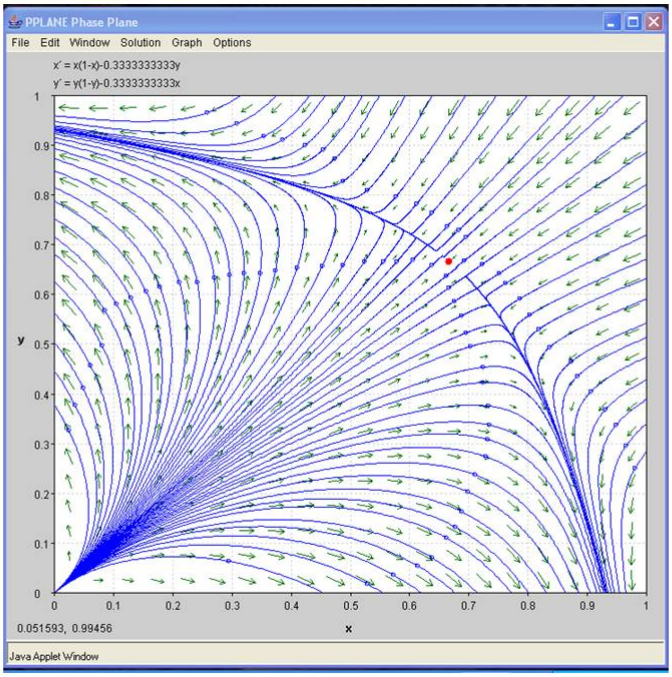


FIGURE 12. $\alpha = 0.333$

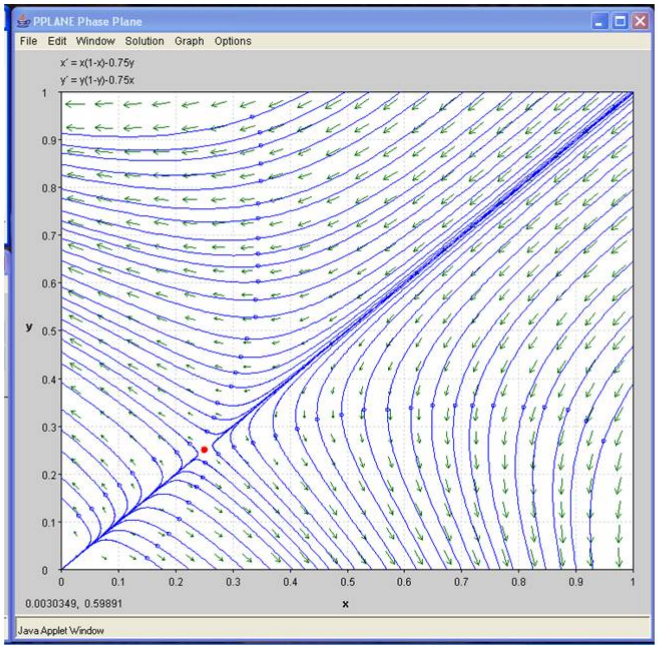


FIGURE 13. $\alpha = 0.75$

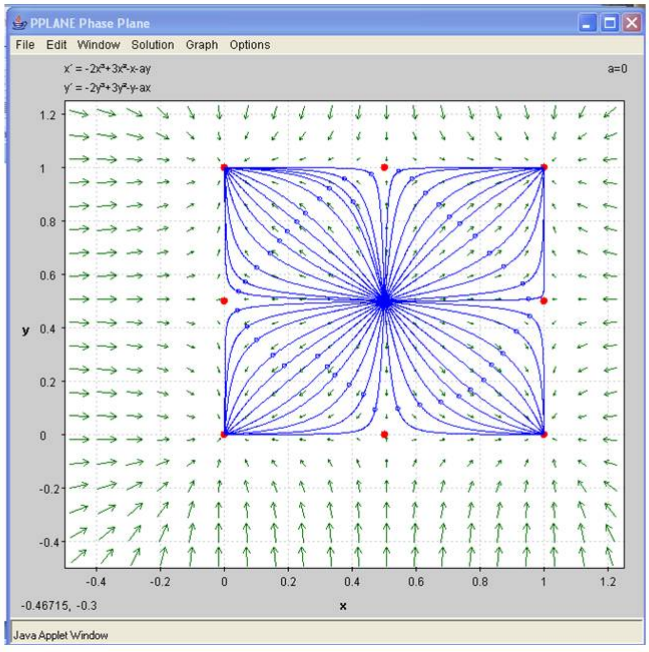


FIGURE 14. $\alpha = 0$

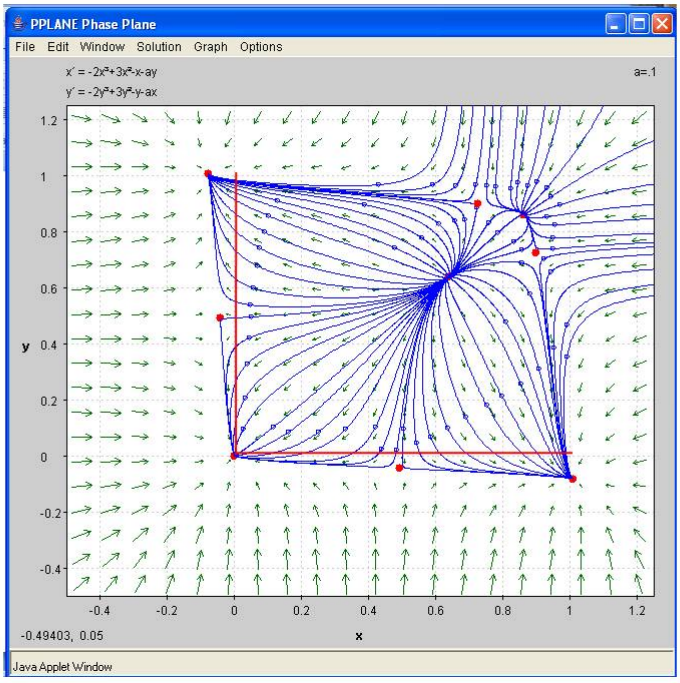


FIGURE 15. $\alpha = 0.1$

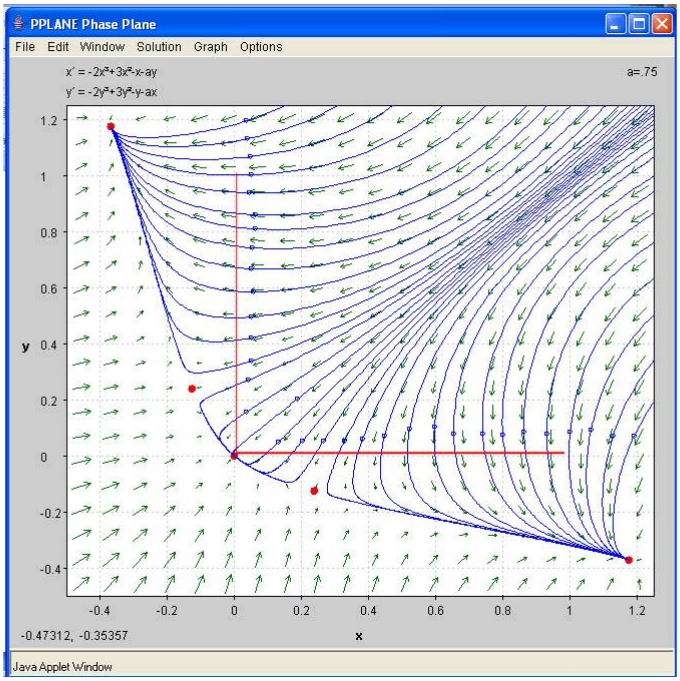


FIGURE 16. $\alpha = 0.75$

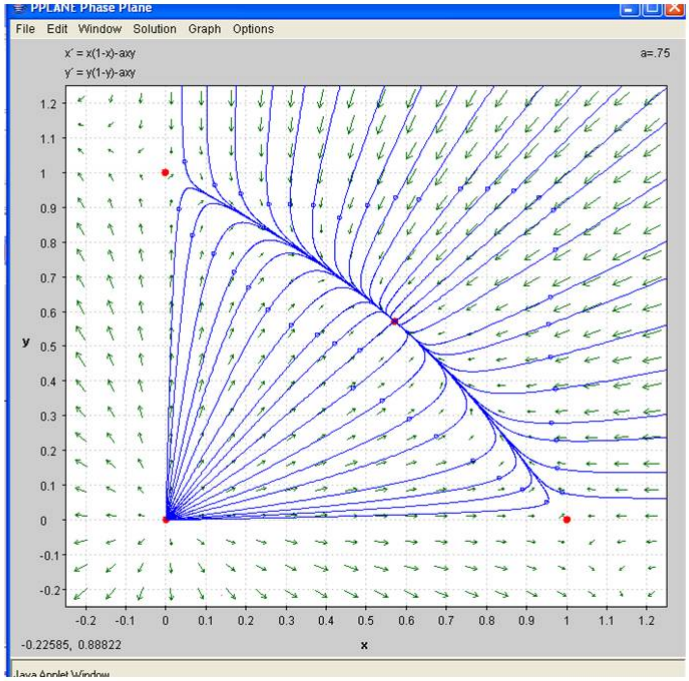


FIGURE 17. $\alpha = 0.75$

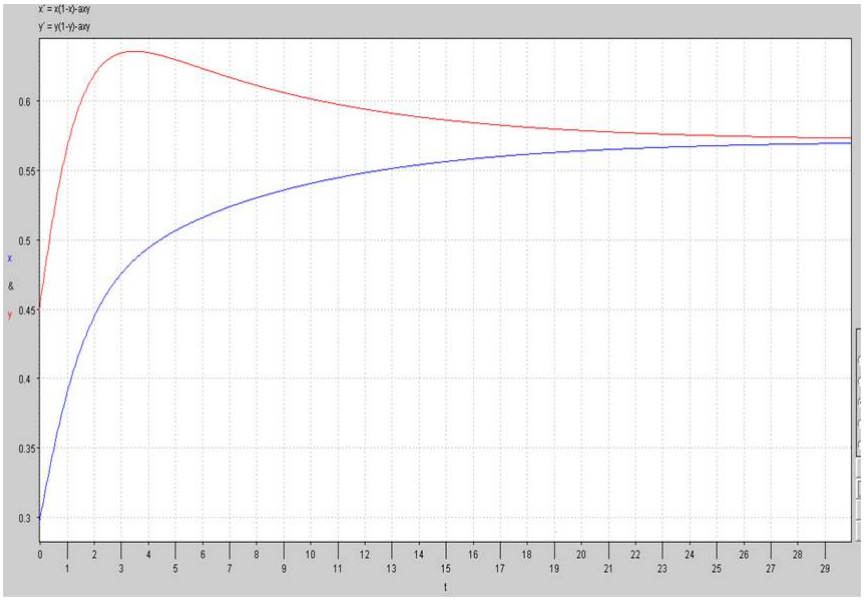


FIGURE 18. A particular solution of F_1 and F_2 over time

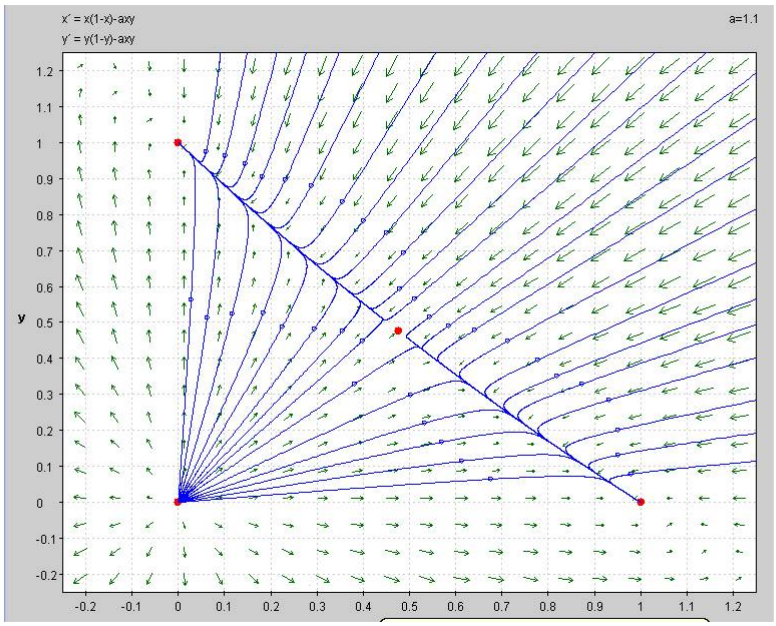


FIGURE 19. $\alpha = 1.1$

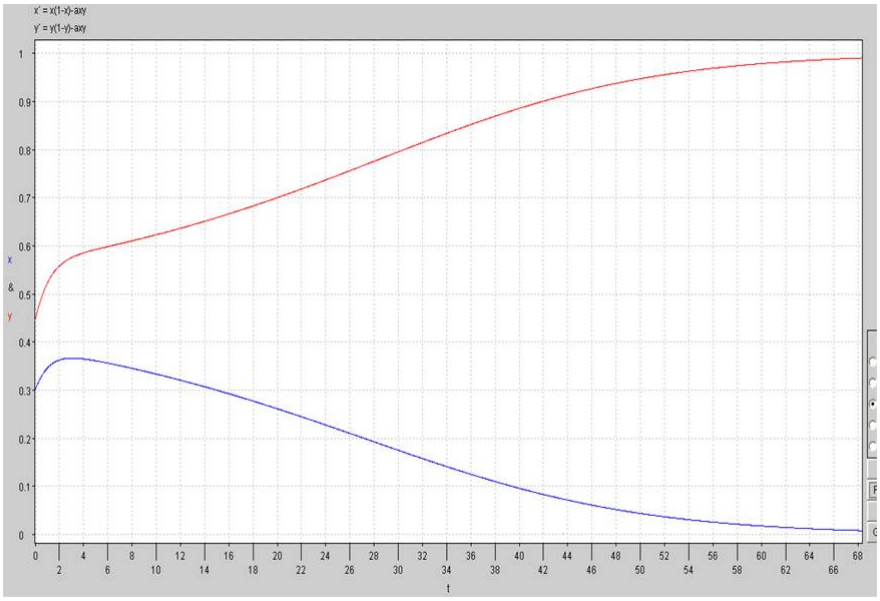


FIGURE 20. A particular solution; $0 \leq t \leq 68$

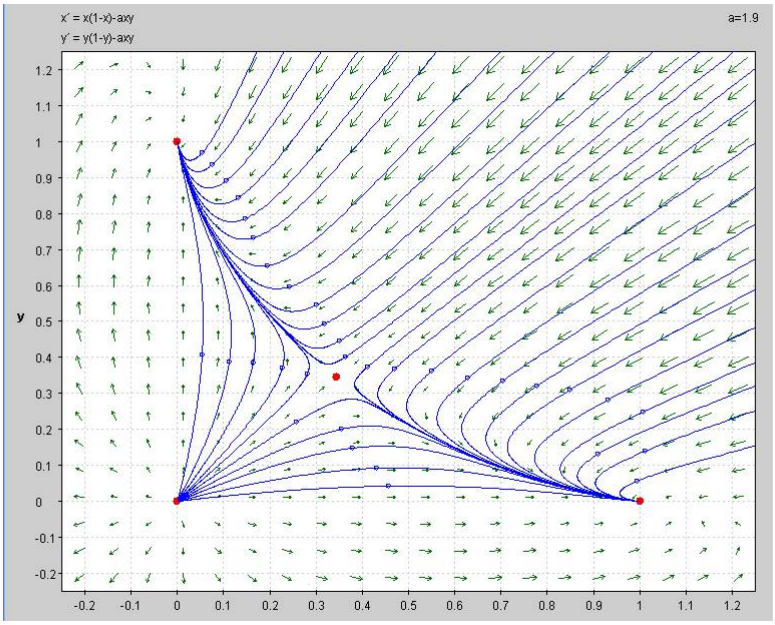


FIGURE 21. $\alpha = 1.9$

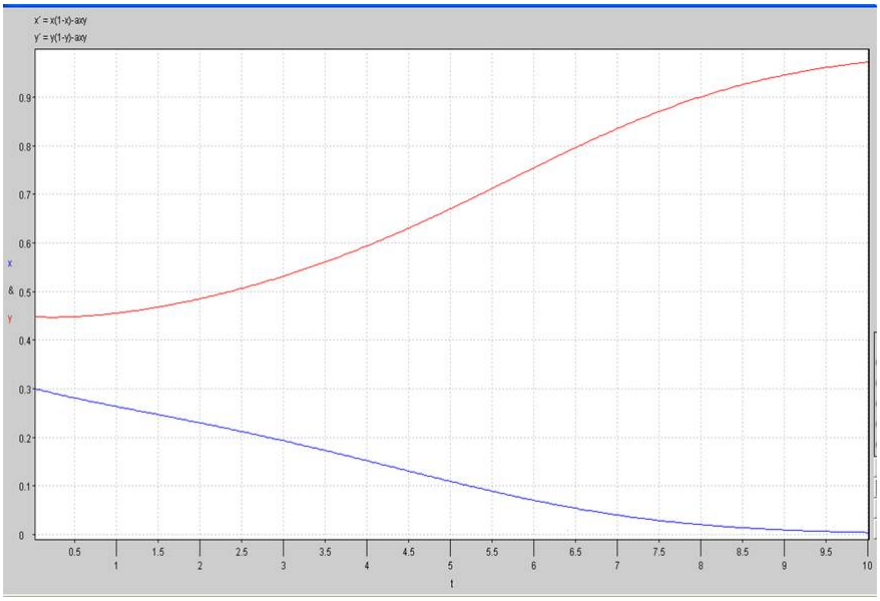


FIGURE 22. A particular solution; $0 \leq t \leq 10$

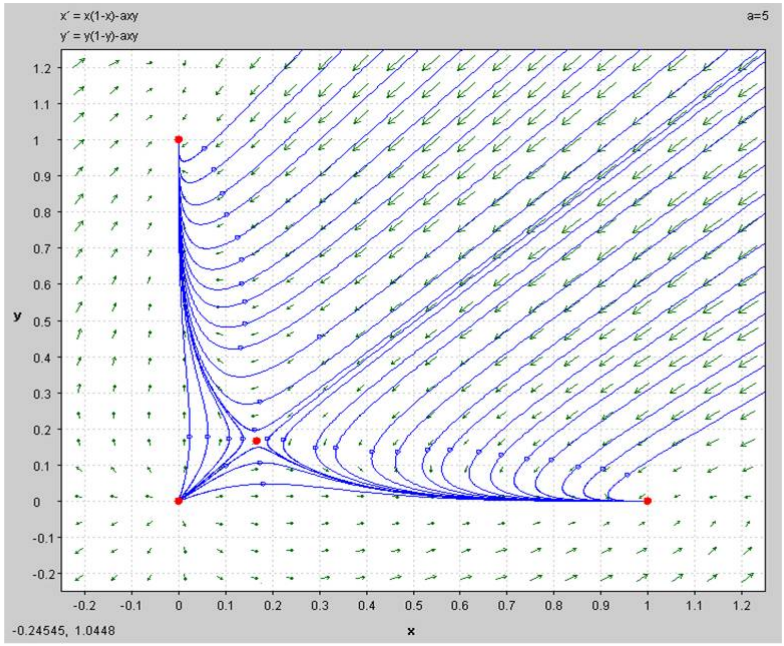


FIGURE 23. $\alpha = 5.0$

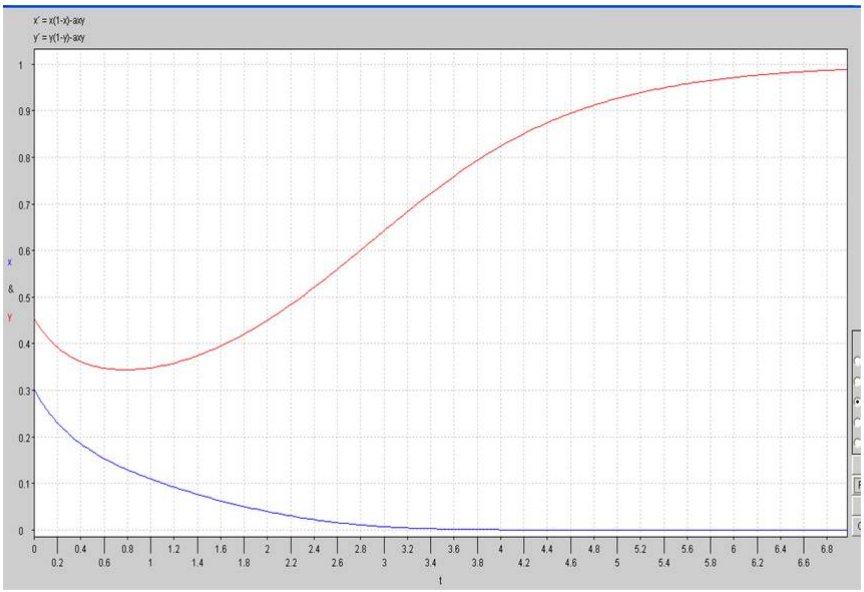


FIGURE 24. A particular solution; $0 \leq t \leq 7$

References

[1] Buzsáki, G (2003), Organization of cell assemblies in the hippocampus, Nature, 424, 552 –556.
[2] Buzsáki, G (2006) Rhythms of the Brain, Oxford University Press
[3] Hebb, D O (1949) The Organization of Behavior: a neuropsychological theory, Wiley and Sons
[4] Huyck, C (2003), Cell Assemblies http://www.cwa.mdx.ac.uk/chris/hebb/hebb.html
[5] Kandel, E et al. (2000) Principles of Neural Science: Fourth Edition, McGraw-Hill
[6] Lansner, A (2009), Associative memory models: from the cell-assembly theory to biophysically detailed cortex simulations, Trends in Neurosciences, 32, 178 –184.
[7] Marr, D (1969), A theory of cerebellar cortex, The Journal of Physiology, 202, 437 –470.
[8] Marr, D (1971), Simple Memory: A Theory for Archicortex, Philosophical Transactions of the Royal Society of London, 262, 23 –81.
[9] Milner, P M (1957), The Cell Assembly: Mark II, Psychological Review, Vol 64, 242 252.
[10] Palm, G (1981), Towards a Theory of Cell Assemblies, Biological Cybernetics, 39, 181 –194.
[11] Scott, A (2002) Neuroscience: a mathematical primer, Springer-Verlag New York, Inc.

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