Simulating the Principles of Chaotic Neurodynamics

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Abstract

Intelligent behavior in biological organisms is achieved through the coupled interaction of exogenously imposed environmental constraints and endogenously generated drives and goals. This view of cognition as a structured coupling of external environment and internal mechanism is certainly not new and is espoused by, for example, the ecological approach to psychology of Gibson [8] among others. However, what is new since the time of the first work in cybernetics and systems theory is a deeper understanding of the mathematics of complex nonlinear dynamical systems, including chaotic dynamics. The insights of complex nonlinear systems theory have begun to be applied to neurology [3, 14, 5, 2] and cognition [1, 9, 10, 15]. Biological organisms manifest intentional behaviors through the self-organization of goals, strategies and behaviors in pursuit of endogenous drives mediated by external constraints [6]. Chaotic neurodynamics may provide a crucial piece in the internal self-organizational mechanisms of behavior. Chaos provides a mechanism that balances flexibility with stability and allows for the creative generation of behavior to satisfy drives. In this paper we discuss these issues and present a neurobiologically inspired simulation of chaotic neurodynamics in our attempt to model truly intentional systems.

Keywords: Chaos, chaotic neurodynamics, complex adaptive systems.

Introduction

Biologically inspired control architectures for adaptive agents have begun to make use of complex spatial and temporal dynamics to try and explain cognition. Clark [1] categorizes such biologically inspired architectures as third generation connectionist models. Third generation connectionist models are characterized by more complex temporal and spatial dynamics. More complex temporal dynamics are due, in part, to the use of feedback and recurrent connections in the models. Complex spatial dynamics are seen in the variety of connectionist architectures produced, usually meant to capture some aspect of the architecture of biological brains. Such simulations are no longer strictly 3 layered, with input, hidden and output layers, but have many layers connected with specialized and complex relations. Examples of third generation connectionist models include the DARWIN series produces by Edelman's research associates [2] and the Distributed Adaptive Control (DAC) models of Verschure and Pfeifer [13].

Some researchers in dynamical cognition and neurodynamics have speculated on the possibilities that more complex, chaotic like dynamics may play in the role of adaptive behavior [14, 5, 7, 11, 12]. Chaotic dynamics have been observed in the formation of perceptual states of the olfactory sense in rabbits [14]. Skarda and Freeman have speculated that chaos may play a fundamental role in the formation of perceptual meanings. Chaos provides the right blend of stability and flexibility needed by the system. Essentially, Skarda and Freeman believe that the normal background activity of neural systems is a chaotic state. In the perceptual systems, input from the sensors perturbs the neuronal ensembles from the chaotic background, and the result is that the system transitions into a new attractor that represents the meaning of the sensory input, given the context of the state of the organism and its environment. But the normal chaotic background state is not like noise. Noise cannot be easily stopped and started, whereas chaos can essentially switch immediately from one attractor to another. This type of dynamics may be a key property in the flexible production of behavior in biological organisms.

Freeman [3, 14, 4] has developed a model of the chaotic dynamics observed in the olfactory system. Freeman's model is based on the description and solution of sets of 2nd order ordinary differential equations and are called K-sets. We next describe a version of the K-set model that we have developed for use in the creation of adaptive agent control architectures, referred to as KA-sets (K-sets for adaptive agents). We then present simulations using the KA-sets to model some of the important principles of chaotic neurodynamics.

KA Model

The purpose of the model presented here is to provide elementary units capable of the complex dynamics observed in biological organisms. These units model the dynamics of populations of neurons, rather than a single neuron. The modeled units presented here are also designed to be computationally efficient, so that they may be used to build real-time control architectures for autonomous agents.

At its heart the KA model uses a discrete time difference equation to replicate the dynamics of the original 2nd order ordinary differential equations of the K-sets. A unit in the KA model simulates the dynamics of a neuronal population. Each KA unit simulates a current, which represents an average population current density. The basic form of the difference equation

can be stated simply as shown in equation 1, which simply states that the current at time step $t+1$ depends on the current at time t plus some change that is applied to the current.

$$c_{t+1} = c_t + \Delta_t \tag{1}$$

The difference equation of the KA unit can be described as 3 differences that are combined to compute the simulated current at time $t+1$ from the current and the rate of change of the current at time t. These 3 influences on the simulated current are 1) a tendency to decay back to the baseline steady state; 2) a tendency to maintain the momentum of the current in a particular direction; and 3) the influences of external excitation or inhibition as input to the unit. In addition a saturation effect is simulated such that as the unit moves towards its theoretical maximum or minimum, it becomes increasingly difficult to continue to push the current towards those extremes.

In the KA model the simulated current has a tendency to return back to its baseline steady state. In our model, the current is constrained to range from -1.0 to 1.0, with a current of 0 being the resting current. The effect of decay is described by equation 2.

$$\Delta_t^1 = -c_t \times \alpha \tag{2}$$

Where α is a parameter that indicates the rate of the decay. Since the difference is proportional to the current, the effect is to cause the decay to be rapid for large values of the current, while the decay slows down as the current approaches 0.

Neural populations exhibit a certain amount of momentum in the dynamics of their current's change over time. In essence, once a population's current begins to move in a certain direction (positive or negative) it tends to keep moving in that direction even for some time after any influence pushing it has been removed.

We first define the rate of change of the current at time t (r_t). This is simply defined as the difference of the current at time t from the current at the previous time step $t-1$. The rate at time t is given in equation 3.

$$r_t = c_t - c_{t-1} \tag{3}$$

With the rate at time t defined, we can describe the momentum as shown in equation 4.

$$\Delta_t^2 = r_t \times \beta \tag{4}$$

Where β is a parameter that controls how much of an influence the momentum has on the dynamics of the model. β can be thought of again as a percentage which indicates what portion of the momentum at the current time step should continue into the next time step.

In the KA model, units may be connected together with other units to form networks. A KA unit is strictly either excitatory or inhibitory. Excitatory units cause the current level to be increased in units they are connected to while inhibitory units have the opposite effect. The effect of excitation or inhibition is scaled by a weight on the link between the units. Weights can range from 0.0 to 2.0 in value.

The effect of the input at time t is calculated by equation 5. In this equation, the unit under consideration receives input from N external units. f is a transfer function of the current, w_i is the weight of the connection between the two units and γ is a scaling factor.

$$\Delta_t^3 = \sum_{i=1}^{N} f(c_t^i) w_i \gamma \tag{5}$$

Another feature supported by the KA model is the specification of delays between the connected units. When a delay is present on a connection between units, the link is no longer simply specified by the weight w_i but it also has a delay parameter d_i associated with it. The delay indicates how far back in time to examine the output of the connected unit ($f(c_{t-d_i}^i)$). The default delay is 0.

The output of a KA unit is calculated as a function of its current. We use an asymmetric sigmoid function, shown in equation 6, to calculate the output.

$$o_t = \epsilon\{1 - exp[\frac{-(e^{c_t} - 1)}{\epsilon}]\} \tag{6}$$

o_t is used as the output if the unit is excitatory, otherwise $-o_t$ is used.

$$o_t = \begin{cases} o_t & \text{if unit is excitatory} \\ -o_t & \text{if unit is inhibitory} \end{cases} \tag{7}$$

The ϵ parameter is a scaling factor that indicates the level of arousal of the KA unit. In the KA model, we take the result of equation 6 and scale them so that the values range between 0 and 1.

The sum of the differences in equations 2, 4 and 5 represent the total difference that we are proposing to apply to the current:

$$\Delta_t' = \Delta_t^1 + \Delta_t^2 + \Delta_t^3 \tag{8}$$

However, before this difference is applied, we first check for the saturation of the unit. Saturation begins to occur when a unit goes above (or below) a saturation threshold. η is the saturation threshold parameter and λ is a parameter that determines the rate of saturation. Equation 9 shows how the saturation is applied.

$$\Delta_t = \begin{cases} \Delta_t' & \text{if } |c_t + \Delta_t'| \leq \eta \\ \Delta_t'(\frac{1-|c_t|}{1-\eta})^\lambda & \text{if } |c_t + \Delta_t'| > \eta \end{cases} \tag{9}$$

In other words, if we are still below the threshold then we simply use Δ_t' as the difference. If we are above the threshold, the Δ_t' gets scaled by the value $(\frac{1-|c_t|}{1-\eta})^\lambda$.

Given the definitions of the previous equations 2-9, the final form of the difference equation used by the KA units can then be stated simply, as shown in equation 1. In other words, the current in the next time step is simply obtained by adding the current at the present time to the difference, where the difference is a result of the 3 influences (decay to baseline, momentum and input) modified by a saturation effect. Table 1 gives a summary of all of the variables and parameters used in the KA model along with default values for the parameters that will be used in the simulations in the next section.

Table 1: KA Model Variables and Parameters

Variable	Description	
c_t	Simulated current at time t	
r_t	Rate of change of current at time t.	
Δ_t	Difference to be applied to current at time t	
Δ_t^1	Difference at time t due to decay to baseline	
Δ_t^2	Difference at time t due to momentum	
Δ_t^3	Difference at time t due to external input	
Parameter	Description	Default
α	Rate of decay to baseline	0.03
β	Rate of momentum	0.81
γ	Input scaling parameter	0.018
ϵ	Transfer function arousal level	5
η	Saturation threshold	0.75
λ	Saturation scaling ratio	0.5

KA Simulations of Neurodynamic Principles

The units in the KA model are discrete time difference equations that capture some of the properties of the time-varying dynamics of neural populations. These properties are essential in generating important behavioral principles that may lie at the heart of the flexible generation of behavior by biological brains.

KA-I Single Unit Dynamics

Figure 1 shows a simulation of a single KA unit in response to a period of excitation. In this simulation, the unit received external stimulation for the first 5 time steps. The unit shows a typical population response in the slope of the curve as it returns back to its baseline state, as well as a slight overshoot of the current before coming back to rest, due to the inherent momentum of the changing current. In all of our simulations, time is calculated in discrete steps, where each step represents 1 millisecond.

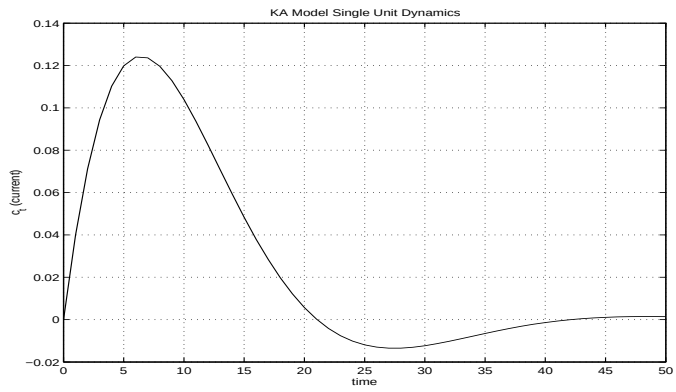

Figure 1: KA simulation of single isolated population dynamics.

Feedback Dynamics

Freeman [5, pg. 37] postulates 10 building blocks of neurodynamics that help to explain how neural populations create the chaotic dynamics of intentionality. These principles are given in the appendix.

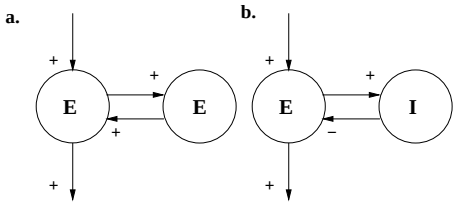

Figure 2: (a) Excitatory-Excitatory feedback (b) Excitatory-Inhibitory feedback.

We can demonstrate the first three principles, which deal with the formation of point and limit cycle attractors, using the KA-I units by connecting them together with mutual feedback. Figure 2a illustrates a simulation of two excitatory units connected with mutual feedback to demonstrate principle 1. Figure 2b illustrates the same but with an excitatory-inhibitory pair to demonstrate principles 2 and 3.

In Figure 3 we demonstrate principle 1 where excitatory-excitatory feedback transitions to a non-zero steady state. The two excitatory units stimulate one another but their activation is constrained from running away because of the saturation effects of the individual units and the less than unity gain between them. These units eventually obtain a non-zero steady state (point attractor). In the figure we show three different steady state levels that are achieved by varying the weights between the excitatory units.

In Figure 4 we demonstrate principle 2. In this simulation, an excitatory unit is connected with an inhibitory unit. When the weight of the links between the units is below a certain threshold, the units will respond to a stimulation by oscillating up and down but gradually damping back and returning to their steady state.

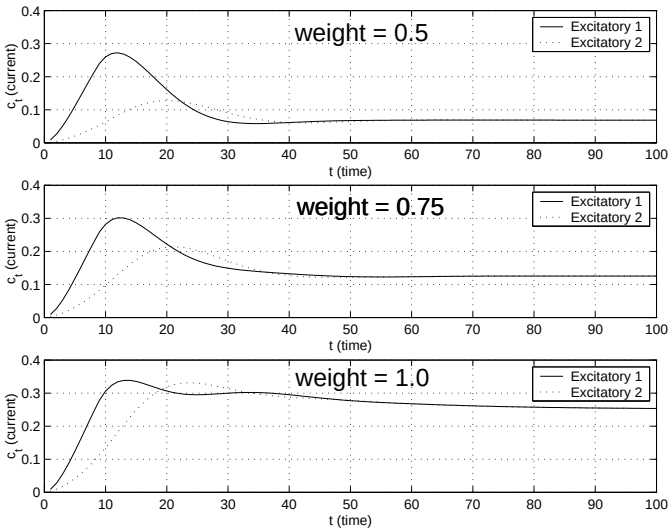

Figure 3: KA simulation excitatory-excitatory.

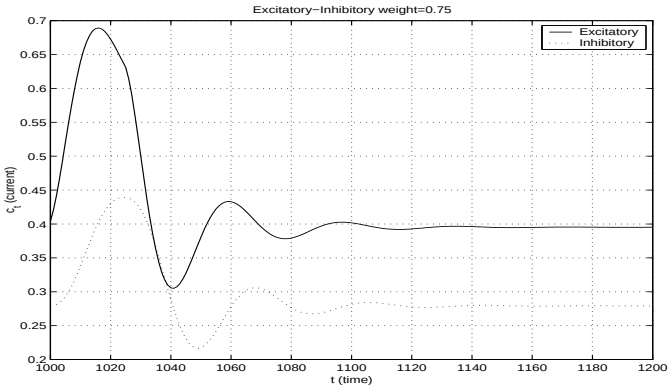

Figure 4: KA simulation excitatory-inhibitory, weight=0.75.

Figure 5 demonstrates principle 3. We again are using an excitatory and an inhibitory unit connected together with mutual feedback. In this simulation, however, the weight between the units is greater than the critical threshold value. When the connection weight between units exceeds this threshold, the excitatory-inhibitory pair's oscillation no longer simply dies out but continues to oscillate in response to a stimulation.

KA-II Mixed Excitatory-Inhibitory Populations

Figure 6 illustrates the configuration of the original K-II sets, and also our KA-II sets. In this configuration two excitatory and two inhibitory units are connected with mutual feedback connections. KA-II sets are capable of the oscillation and limit cycle dynamics of the first three principles shown previously. A KA-II set will tend to oscillate at a characteristic frequency. Figure 7 displays a typical time series of the 4 units in a KA-II simulation. An analysis of the power spectrum distribution of the first excitatory unit is displayed in figure 8. Notice that the power

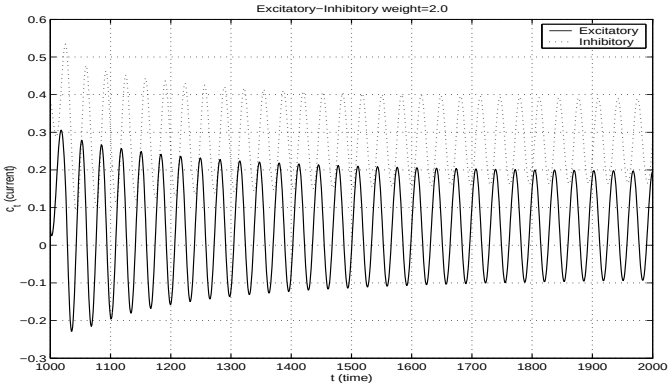

Figure 5: KA simulation excitatory-inhibitory, weight=2.0.

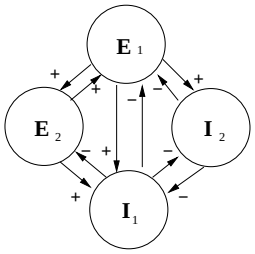

Figure 6: KA-II mixed population simulation.

spectrum shows a peak frequency occurring at about 27 Hz, which is the characteristic frequency for a KA-II with its particular weight settings. A KA-II set will tend to oscillate at a characteristic frequency in the so called Gamma band range, which is typical of EEG measurements from biological brains.

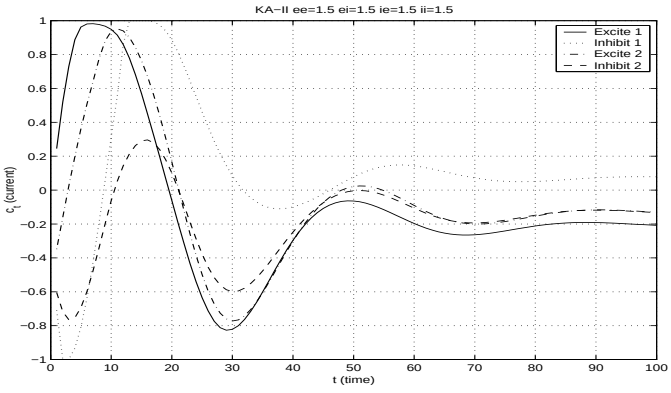

Figure 7: A typical simulated run of a KA-II.

KA-III Sets and Chaotic Dynamics

Freeman's fourth principle building block of neurodynamics, as shown in the appendix, concerns the formation of chaotic background activity.

Connecting 3 KA-II populations together as shown in Figure 9 creates a KA-III set which is capable of satisfying principle 4. In this configuration, each of the

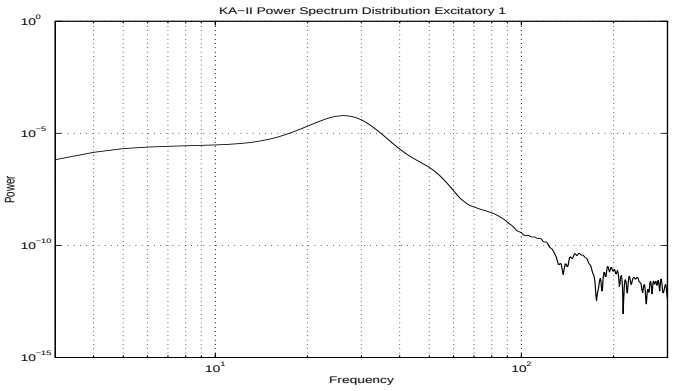

Figure 8: KA-II power spectrum distribution of Excitatory 1.

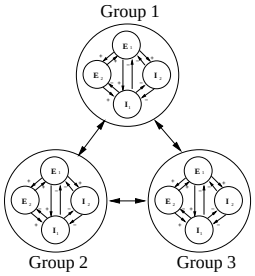

Figure 9: KA-III, 3 populations generate chaotic dynamics.

KA-II populations has different internal connection weights and therefore oscillates at a different characteristic frequency. The connections between KA-II populations are from the first excitatory unit in each population. The groups are fully connected to each other and weights and delays between the groups have been found through experimentation that produce chaotic behavior.

Figure 10 shows a portion of the time series of the first excitatory unit from each of the three groups and Figure 11 is a state space representation of the activity of group 2 versus group 3. The state space plot shows a typical tangle of the activity of the two groups, with no noticeable periodicity. However the behavior between the groups is definitely not random, the activity is confined to a subspace of the total possible state space. Figure 12 is a power spectrum analysis of the time series of the first excitatory unit from group 2. Notice now there are many frequency peaks detected in the signal, not a simple single characteristic frequency. This is due to the interaction of the three incommensurate frequencies of the KA-II groups.

A calculation of the Lyapunov exponent of the three time series using Wolf's method [16] shows a strictly positive exponent above 0.04 for all of them. This is an indication that the trajectories are in the chaotic region. KA-III sets, like the original K-III sets, are capable of producing and modeling chaotic background activity.

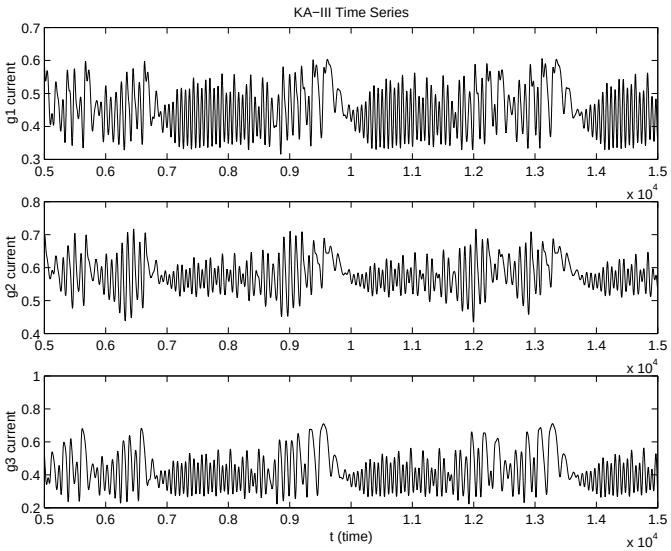

Figure 10: KA-III Time Series of Group 1, 2 and 3.

Conclusion

KA units are based upon Freeman's original K-sets and are capable of reproducing the first four principles of chaotic neurodynamics. KA sets are built using difference equations (rather than solving sets of ordinary differential equations) and are fast enough to build modest architectures capable of controlling an autonomous agent in real-time.

We have begun work on using the KA model to implement the remaining principles of chaotic neurodynamics. Our goal is to demonstrate the importance of chaotic neurodynamics by building autonomous agents based on these principles. We are developing complete agent architectures for perception, memory and action selection, using insights from this work and previous results on the importance of chaos in perception and memory [5, 14, 10]. We believe that architectures based on these principles may show some of the flexibility and complexity displayed by biological organisms performing cognitive tasks.

Acknowledgment

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Appendix 1 - Principles

Freeman's ten principles of chaotic neurodynamics [5, pg. 37]:

1. The **state transition** of an excitatory population from a point attractor with zero activity to a **non-zero point attractor** with steady-state activity by **positive feedback**.
2. The emergence of **oscillation** through **negative feedback** between excitatory and inhibitory neural populations.
3. The state transition from a point attractor to a **limit cycle attractor** that regulates steady-state oscillation of a mixed excitatory-inhibitory cortical population.
4. The genesis of **chaos** as **background activity** by combined negative and positive feedback among three or more mixed excitatory-inhibitory populations.

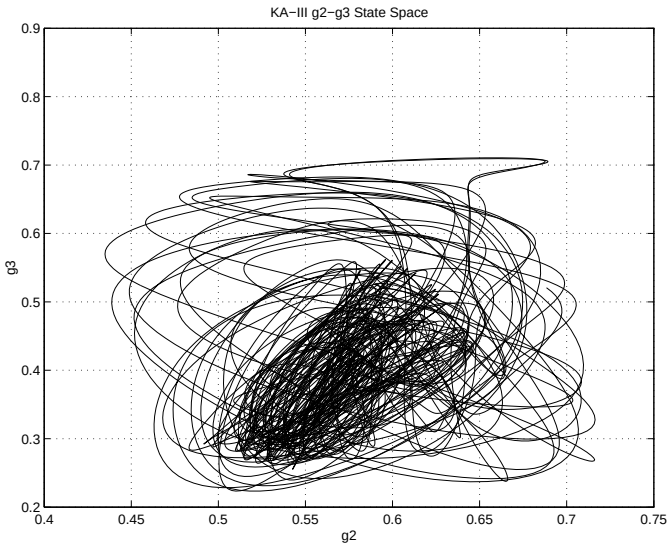

Figure 11: KA-III State Space of Group 2 vs. Group 3.

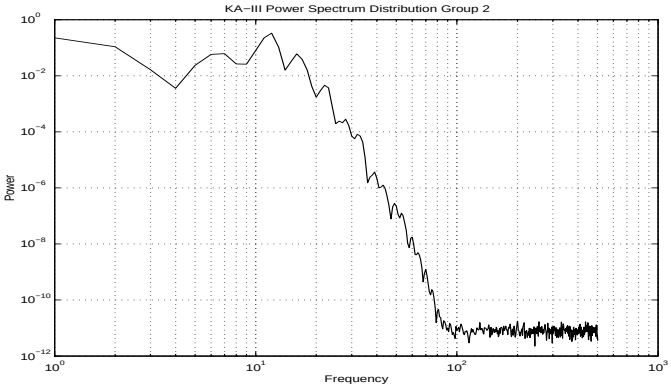

Figure 12: KA-III Power Spectrum Distribution of Group 3.

5. The distributed wave of chaotic dendritic activity that carries a spatial pattern of **amplitude modulation** made by the local heights of the wave.
6. The increase in **nonlinear feedback gain** that is driven by input to a mixed population, which results in construction of an amplitude-modulation pattern as the first step in perception.
7. The **embodiment of meaning** in amplitude-modulation patterns of neural activity, which are shaped by synaptic interactions that have been modified through learning.
8. **Attentuation** of microscopic sensory-driven activity and **enhancement** of macroscopic amplitude-modulation patterns by **divergent-convergent** cortical projections underlying solipsism.
9. The divergence of corollary discharges in **preafference** followed by **multisensory convergence** into the entorhinal cortex as the basis for Gestalt formation.
10. The formation of a sequence of **global amplitude-modulation patterns** of chaotic activity that integrates and directs the intentional state of an entire hemisphere.

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