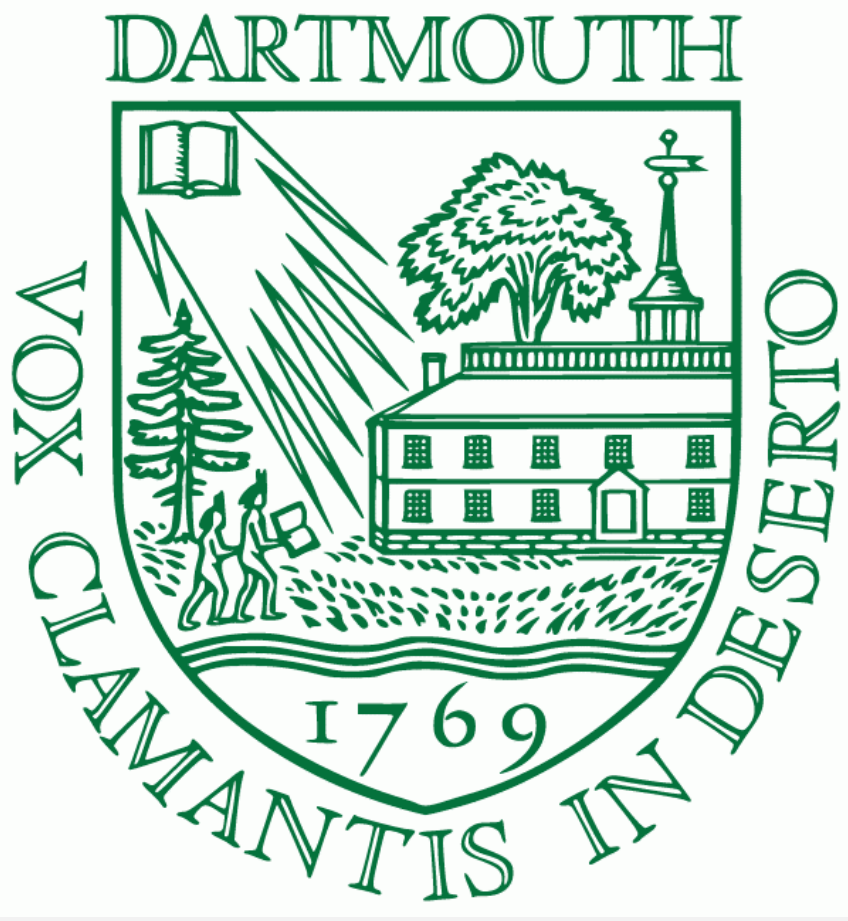


Dynamic Analysis of Synchronization in Neural Networks

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INTRODUCTION

Neurons, the cells that make up the brain, form the basis of sensory perception in humans, and perception in turn plays a large role in many important life processes and decision-making. These include both those that take place over large time scales, such as the daily circadian cycle, as well as quick, one-time actions, such as identifying objects in the field of vision. Receiving, processing, and acting on stimuli is a complex procedure, and it requires networks of neurons to work together in some kind of synchronized fashion. For a neural network, synchronization entails the neurons in the network firing or releasing chemicals in the same oscillatory phase.

An important question in the study of neural networks is whether synchronization of neurons is stable, or whether states of partial synchronization or non-synchronization are stable. Given that individual neurons undergo impulses periodically in response to a stimulus and that neurons within a network are linked in terms of phase, the neurons are modeled as phase oscillators that each have a natural frequency yet are also governed by the force of coupling with other neurons in their network; this coupling causes the neurons to tend more towards synchronization with other neurons in the network in terms of their firing while the neurons’ natural frequencies tend to resist this synchronization.

BASIC MODEL

The baseline model used here to analyze the periodic impulses of neurons as phase oscillators is the Kuramoto Model. This model is $\theta_i' = \omega_i + \sum_{j=1}^N K_{ij} \sin(\theta_j - \theta_i)$, ($i = 1, 2, \dots, N$), where θ_i represents the displacement from equilibrium of a neuron, ω_i is the neuron’s natural frequency (which may not be identical to that of other neurons), and N is the number of neurons in the network. Further, K represents the coupling matrix, so $K_{ij} \geq 0$ is some coupling constant between neurons i and j , where larger values of K_{ij} imply stronger coupling between the two neurons.

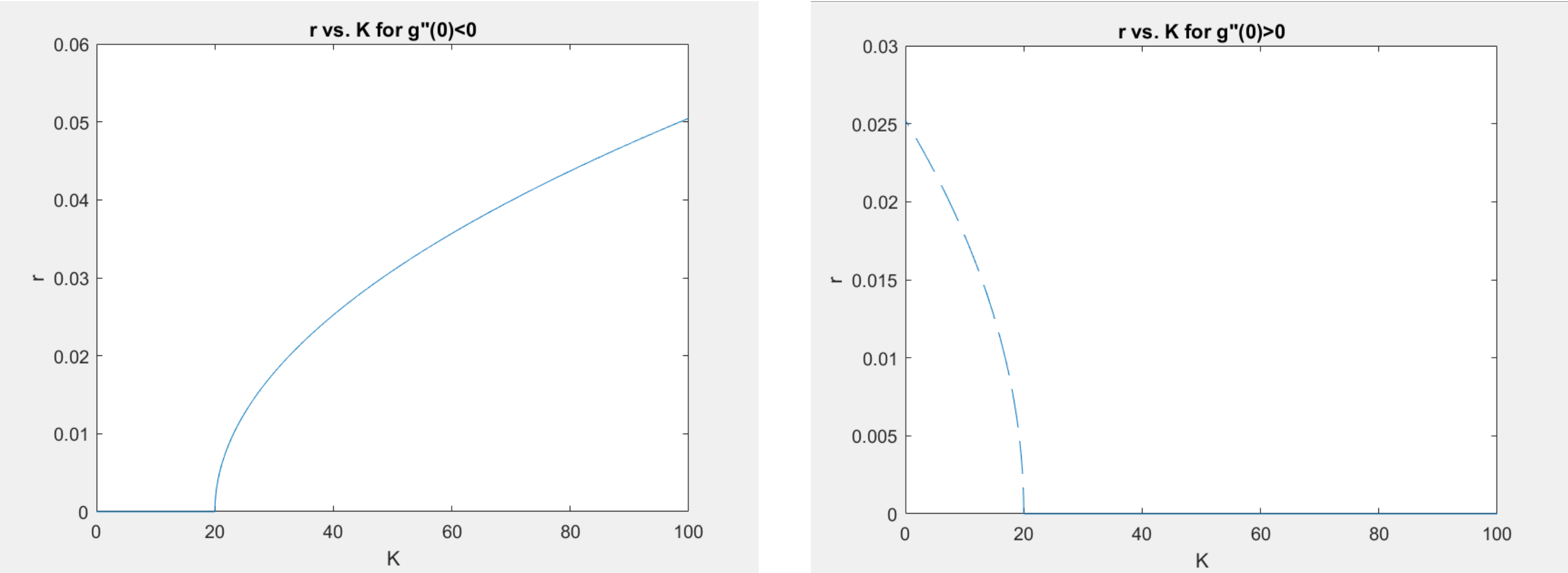
Kuramoto further developed this model by introducing an order parameter, $r(t)$, where $0 \leq r \leq 1$ and values of r closer to 1 mean that the network of neurons are more synchronized while $r=0$ means that all neurons oscillate at their individual natural frequencies, which implies that they are not synchronized. The parameter $r(t)$ satisfies the equality $re^{i\psi} = \frac{1}{N} \sum_{j=1}^N e^{i\theta_j}$. Kuramoto then used this order parameter to modify the original model into a slightly simpler form: $\theta_i' = \omega_i + Krsin(\psi - \theta_i)$; $i = 1, 2, \dots, N$. In this model, K is defined as $K = N * K_{ij}$. Though the model is still nonlinear in θ , the modifications do allow the summation term to be removed. It is important to note that defining K in this way is based on the assumption that coupling among neurons is in the form of mean-field coupling, which means that all the neurons in the network have the same degree of coupling to each other and are coupled to a common average phase, rather than each pair of neurons having a unique coupling strength and being coupled individually to each other neuron. The shared coupling strength (K) is defined to be equal to the mean coupling strength of individual pairs of neurons multiplied by N , the number of neurons in the network.

ASSUMPTIONS

As mentioned above, it is assumed that coupling among neurons takes the form of mean-field coupling in particular, as opposed to other forms of coupling such as short-range couplings and hierarchical couplings. It makes sense to assume mean-field coupling because neural functions are undertaken by relatively large regions of the brain or even disparate regions of the brain, and it is most efficient for the firing of the neurons that play a role in a given function to be coupled to some average value. In addition, it is assumed that the number of neurons $N \rightarrow \infty$. Again, this makes sense as an assumption because there are approximately 100 billion neurons in the human brain, so even if only a small portion of the brain is considered as part of a network, there are still likely tens of millions of neurons in that network. A final assumption is that the natural frequencies ω_i of the neurons are distributed by a probability density function with a mean of ω_0 , and the natural frequency of any given neuron is random according to that probability density function.

BIFURCATION ANALYSIS

Acebron et al. determined that $r \sim \sqrt{\frac{-16(K - K_c)}{\pi K_c^4 g''(0)}}$, where $g(\omega)$ is a general frequency distribution for the neurons’ natural frequencies and K_c is some critical value of K at which the system bifurcates between coherence (synchronization) and incoherence of neuronal oscillations. Thus, it is clear that if $K = K_c$, then $r = 0$, which by definition means a state of non-synchronization (incoherence), in which all neurons oscillate at their natural frequencies. Since the value of r is of greatest interest here (since r is the determinant of the state of coherence of the network) and K is the bifurcating parameter, it will be most useful to analyze graphs of r vs. K for various values of K and $g''(0)$. For simplicity, we assume that $|g''(0)| = 1$ even though this may not necessarily always be the case; the assumption just makes use of the fact that only the sign of $g''(0)$ matters for the nature of the bifurcation. The magnitude of $g''(0)$ merely affects the magnitude of r . In addition, an arbitrary value is taken for K_c (in this case, $K_c = 20$); similarly, this does not affect the nature of the bifurcation, but rather merely shifts its location to higher or lower values of K .



Note that the negative case ($r < 0$) of the asymptotic value of r can be ignored since r is defined to be in the range $0 \leq r \leq 1$. As expected, the state of a neural network, in terms of synchronization of neurons, bifurcates at K_c . Specifically, for a fixed value of K_c , the bifurcation is supercritical for $g''(0) < 0$ and subcritical for $g''(0) > 0$. In the supercritical case ($g''(0) < 0$), the corresponding graph shows an equilibrium state of partial synchronization ($0 < r < 1$) is stable for $K > K_c$, while in the subcritical case ($g''(0) > 0$), the graph demonstrates that the partially synchronized state is an equilibrium for $K < K_c$. On the other hand, when $K < K_c$ in the supercritical case, the incoherent state ($r = 0$) is an equilibrium; in the subcritical case, the incoherent state is an equilibrium when $K > K_c$. In both cases, this is due to the fact that the value of the fraction inside the square root that is asymptotically equal to r is negative, so r is equal to some complex number with $\text{Re}(r) = 0$.

Regarding the stability of these states, as Acebron et al. point out, the incoherent state is stable for $K < K_c$ in the supercritical case, as is the partially synchronized state in the supercritical case (for $K > K_c$). However, in the subcritical case, the non-synchronized state, where $K > K_c$, is unstable, and the state of partial synchronization is also unstable ($K > K_c$).

It is important to note that partial synchronization refers to a state in which some of the neurons are oscillating at the same phase, which is the average phase of the entire network, while other neurons are oscillating incoherently. In particular, the neurons with phases that do not become locked to the average phase of the network are those that are on the extremes of the tails of the distribution ($g(\omega)$) of natural frequencies. Of course, the stronger the coherence (the more neurons that are synchronized), the closer the value of r will be to 1, which reflects the fact that more neurons are becoming phase locked to the average phase.

EXAMPLE OF A FREQUENCY DISTRIBUTION

The main illustrative example of a frequency distribution that Kuramoto used was $g(\omega) = \frac{\gamma}{\pi(\gamma^2 + \omega^2)}$. For that case, he was able to find an exact result for r , $r = \sqrt{1 - \frac{K_c}{K}}$. In addition, using the fact that $K_c = \frac{2}{\pi g(0)}$, it can be determined that $K_c = 2\gamma$ (γ represents the “width” of the frequency distribution). This particular $g(\omega)$ falls under the supercritical case ($g''(\omega) < 0$ because $g''(\omega) = \frac{-2}{\pi\gamma^3}$ and $\gamma > 0$), so it makes sense that $\text{Re}(r) = 0$ for $K < K_c$, and for $K > K_c$, $0 < r < 1$, indicating partial synchronization.

CONNECTING BACK TO THE KURAMOTO MODEL

Returning to the basic Kuramoto model with mean-field coupling ($\theta_i' = \omega_i + Krsin(\psi - \theta_i)$; where $i = 1, 2, 3, \dots, N$), the results of the bifurcation analyses clearly fit with this model. It is trivial to see that when $K = 0$ and the frequency distribution of natural frequencies is unimodal, which implies that $g''(0) < 0$, $\theta_i' = \omega_i$ and each neuron will oscillate at its own natural frequency. However, this is not the case when $g''(0) > 0$ (a bimodal or multimodal distribution), but the frequency distribution of neurons’ natural frequencies is commonly thought to be unimodal. In addition, when $r = 0$, reflecting an incoherent state, again $\theta_i' = \omega_i$ and neurons will oscillate at their natural frequencies, with coupling not being a factor.

On the other hand, as K becomes greater than K_c (if $g''(0) < 0$) or less than K_c (if $g''(0) > 0$) and the value of K moves further from K_c , the bifurcation demonstrated that the partially synchronized state becomes more synchronized. This makes sense in terms of the differential equation because as the magnitudes of K and r become larger, the coupling term ($Krsin(\psi - \theta_i)$) tends to dominate over the natural frequency term, meaning that the neurons will tend to be more synchronized to the average phase of the network.

Lastly, it should be pointed out that the nonlinear sine term in the Kuramoto equation gives rise to the potential for many local critical points. In fact, values of θ_i that are critical points are those that satisfy the equation $-\omega_i/(Kr) = \sin(\psi - \theta_i)$, so the system will have many critical points as long as the ratio $-\omega_i/(Kr)$ satisfies $|- \omega_i/(Kr)| < 1$.

CONCLUSIONS AND FURTHER QUESTIONS

The main conclusion from the studies undertaken here as they apply to the oscillations of neurons in humans is that for frequency distributions $g(\omega)$ of neurons’ natural frequencies that satisfy $g''(0) < 0$ (unimodal distributions), coupling strengths that are greater than some critical coupling strength will produce at least partial synchronization in the oscillations of neurons. If the mean-field coupling strength is sufficiently weak, then neurons will tend to oscillate at their natural frequencies, resulting in incoherence. In short, stronger coherence is produced by stronger coupling among neurons in a network. In neural networks with unimodal natural frequency distributions, the partially synchronized state is stable, meaning that for neurons with natural frequencies sufficiently close to the average, the average phase of neurons in the network is an attractor. Note that the qualifier “sufficient” depends on the network’s overall coupling strength.

As discussed in the introduction, synchronization is crucial in numerous neural functions that involve receiving and processing stimuli, as well as associative memory. A major question then becomes precisely how, on a chemical level, neurons create and maintain these coupling connections. In addition, further advancements could be made by performing experiments to determine an accepted range of values for K_c (which likely depends on the number of neurons actively firing at once), a precise idea of what the natural frequency distribution, $g(\omega)$, of neurons is, and a model for the average phase of a network of neurons as a function of time. The latter will potentially be inseparable from the Kuramoto model in that the average phase changes in response to changes in the phase of individual neurons, which in turn change in response to coupling interactions.

In terms of further work in the mathematical modeling of neuronal oscillations, one shortcoming of Kuramoto’s model is that it seems to only take into account innate coupling strength, as opposed to coupling connections that may be strengthened due to some sort of neurological memory. This could occur if repeated synchronized firing of groups of neurons tends to strengthen coupling among those neurons.

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