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BRIEF REPORT

Approximating the Effect of Consciousness on Stochastic Brain Structures

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HIGHLIGHTS

A testable new idea proposes a causal connection between the randomness of physical systems and the decision-making dynamics of the human brain.

ABSTRACT

There is a large amount of evidence in the Parapsychology literature that indicates that consciousness is not an emergent property of neuronal interactions and can exist and function independently of a brain. Here we examine mathematical methods that can be used to derive dynamic equations for the mind-matter interactions occurring in the brain that these observations imply the existence of. We use the moments method to approximate the effect that consciousness has on stochastic binary decision-making neural networks , which we model using biologically realistic Wilson-Cowan equations (Deco et al., 2007). We show that small changes in the variance of the randomness (on the order of 0.1 Hz^2) consumed by the neural networks can bias the networks to select one binary decision value over the other. Using observations about the interconnectedness of relatively isolated groups of neurons, we argue that biases of the size predicted by the approximation would be sufficient to allow a brain-independent consciousness to exert significant control over the brain. The results that we present here are relevant to any theory positing that rote computations carried out by neurons are not the sole contributor to consciousness.

KEYWORDS

Consciousness, survival, parapsychology, moments method, Wilson-Cowan equations.

INTRODUCTION

Over the past century, Parapsychology has amassed an extensive collection of anomalous observations that, when viewed together, suggest that human consciousness can exist and function independently of the brain (Kelly et al., 2007). Cases of veridical near-death experiences (NDEs) suggest that under some circumstances, clinical patients experiencing extreme physiological stress (oftentimes as a result of cardiac arrest) can accurately perceive remote locations in detail– a feat that they would be unable to accomplish if they were fully conscious and not in the throes of a serious medical emergency (Kelly et al., 2007, Chapter 6). Cases of young children claiming to remember details of "previous lives," who bear birthmarks in the shape of fatal injuries they claim to have sustained in previous lives, whose detailed stories can be matched to the life stories and autopsy information of deceased people suggest that in some circumstances consciousness can reincarnate after bodily death (Tucker, 2008). While research into anomalous experiences and happenings of this sort does little to shed light on what consciousness

is directly, it makes a compelling case for what consciousness *is not*. If consciousness can exist without a brain, then it is not, as contemporary neuroscience posits, an emergent property of neuronal interactions. This would imply the existence of mind-matter interactions taking place inside the human brain. Easily observable neuronal processes drive conscious behavior requiring physical movement in humans and brain-equipped animals. For a brain-independent consciousness to exert control over the actions of its body, it must be able to influence these neuronal processes in some way.

Generally, individual neurons behave deterministically following the Hodgkin-Huxley model, so there is no room for a brain-independent consciousness to influence the activity of these cells significantly. However, *in vivo* observations of layer five pyramidal cells in awake cats have shown that some pyramidal cells deviate from Hodgkin-Huxley behavior (Naundorf et al., 2006). These neurons have an unusually wide firing threshold window, resulting in large temporal variability in spike times (Hameroff & Penrose, 2014). Empirical evidence suggests that the physical structures underpinning many brain functions associated with consciousness, such as decision-making, attention, and some types of memory recollection, need to consume random noise to function properly (Deco et al., 2009). A significant portion of this noise comes from the probabilistic spiking of pyramidal cells (Rolls & Deco, 2012, pp. 78-80). If consciousness is independent of the brain, it must *a priori* exert influence on the brain by influencing the random noise that it consumes. This suggests that certain pyramidal cells may contain some mechanism that consciousness can influence. Some have speculated that layer five pyramidal cells contain what are essentially small quantum random noise generators made of microtubules (Hameroff & Penrose, 2014). It may be the case that these structures are sensitive to micro-PK effects, as there is evidence that a combination of conscious attention and intention can influence the behavior of random systems, not limited to just those of a quantum nature (Kauffman & Radin, 2021).

Of the brain structures thought to require the consumption of random noise to function, perhaps the best understood are those that make binary decisions (Deco et al., 2007). In this paper, we will approximate how sensitive the behavior of stochastic binary decision-making neural networks is to small changes in the underlying randomness that they consume. We will show that small changes in the variance of the randomness consumed (on the order of 0.1 Hz^2) by the networks bias the networks to select one choice over the other. Our analysis will use the moment method, and we will derive an analytical approximation for the size of the decision bias. We will then discuss how biases of the kind and size shown to exist when applied to all stochastic brain processes could feasibly allow a brain-independent consciousness to exert substantial control over its associated body. The methods presented here can be used to perform similar analyses on other brain structures that exhibit stochastic behavior. The results that we present here are relevant to any theory positing that consciousness is not the sole product of rote computations carried out by neurons.

STOCHASTIC WILSON-COWAN MODEL OF BINARY DECISION MAKING ATTRACTOR NETWORKS

The Basic Stochastic Wilson-Cowan Model

Empirical data collected from animal experiments have shown that binary decision-making attractor networks are composed of two competing populations of excitatory and inhibitory neurons whose interaction is mediated by inhibition (Deco & Martí, 2007). The brain uses each of the two populations to represent one of two possible binary decisions. In general, after interacting for some time, one population ends up with a firing rate significantly higher than the other, signifying that its associated decision value has been reached. As the populations interact, sensory input fed to each population biases the attractor network to favor one decision over the other if significant evidence for one decision exists. In cases where there is no evidence supporting either of the binary decisions, random noise alone is responsible for causing the system to favor one decision over the other (Deco et al., 2007). Neurons in the same population are connected to each other with excitatory connections of dimensionless weight w_+. The two populations are connected with inhibitory connections of dimensionless weight w_I. The time evolution of the firing rates of the two groups of neurons, $v_i(t)$ where i = 1,2, is driven by competing inhibitory and excitatory interactions—a sort of "push and pull".

This interaction can be accurately modeled using a system of stochastic first-order Wilson-Cowan differential equations (Deco et al., 2007). We will consider Gustavo Deco's 2007 model:

$$\tau \frac{dv_i(t)}{dt} = -v_i(t) + \phi\left(\lambda_i + \sum_{j=1}^{2} w_{ij} v_j(t)\right) + \sqrt{\tau}\xi_i(t), i = 1,2 \qquad (1)$$

where w_{ij} is the (dimensionless) total synaptic strength between populations i and j, λ_i is the external sensory input received by population i measured in Hertz, τ is a time constant describing the rate at which the system responds to sensory input change measured in milliseconds

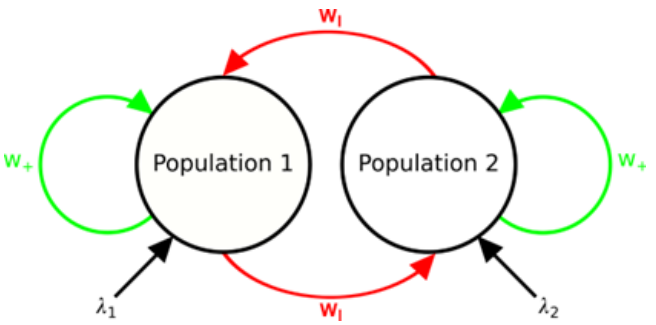

Figure 1. Two populations of neurons, each corresponding to one of two binary decision outcomes, interact with inhibitory connections between populations (red) and excitatory connections within populations (green). The inhibitory connections between the two populations have dimensionless weight w_I, and the excitatory connections among neurons in the same population have dimensionless weight w_+.

$$\phi(x) = \frac{v_c}{1 + exp(-\alpha[x/v_c - 1])}$$

,and φ(x) is the sigmoidal activation function (Deco & Martí, 2007). Here v_c is both the maximal activity rate of the two populations and the input frequency needed to cause each population to assume one-half of its maximal activity rate, and α is a constant. Random fluctuations in the attractor network are modeled by independent additive Gaussian noise terms $\xi_i(t)$ measured in Hertz satisfying $< \xi_i(t) > = 0$ and $< \xi_i(t)\xi_j(s) >= \beta^2\delta(t - s)\delta_{ij}$ where the angle brackets denote expected value β and is the variance of $\xi_i(t)$.

The Phase Plane of the Non-Stochastc Wilson-Cowan Model

Figure 2 depicts the phase plane of what we will refer to as the non-stochastic version of Equation (1), which we will formally define as the case where β = 0 . The case where β = 0 need not be viewed as a stochastic differential equation, as if the variance of $\xi_i(t)$ is zero then the noise term in Equation (1) vanishes because $< \xi_i(t) > = 0$. The time evolution of the system initialized at two initial states $v_1 = 1.25$, $v_2 = 1$ and $v_1 = 1$, $v_2 = 1.25$ is shown as blue lines. The three fixed points of the system are displayed as red dots. The phase plane contains two basins of attraction separated by the line $v_1=v_2$, which have been labeled P_1 and P_2. For initial states in P_1, where $v_1 > v_2$ (or equivalently $v^{def} = (v_1, v_2) \in P_1$, the system eventually falls into the fixed point in P_1, which we will refer to as F_1. For initial states in P_2, where $v_2 > v_1$, the system eventually falls into the fixed point in P_2, which we will refer to as F_2. We will refer to the fixed point on $v_1=v_2$ as F_0. In the non-stochastic version of Equation (1), the side of the $v_1=v_2$ line that the system's initial state resides on completely determines its final state. This observation will later play a crucial role in deriving the approximation of the behavior of Equation (1) in our moment method analysis. F_1 and F_2 correspond to "decision states" that are assumed by the attractor network to represent that one of two possible decisions has been made (Deco & Martí, 2007). F_0 corresponds to the "spontaneous state", which is assumed by the attractor network to represent that a decision has not yet been made. In the presence of noise, the attractor network will tend to settle near one fixed point, each of which carries meaning in the context of the binary decision being made. For all numerical simulations in this paper we will let $w_{11} = w_{22} = w_+ - w_I$ and $w_{12} = w_{21} = w_- - w_I$ where $w_+ = 2.45$,$w_I = 1.9$, $w_- = 0.43(w_+ - 1)$, and $v_c = 20$ Hz, α = 4, τ = 10 ms, and $\lambda_1 = \lambda_2 = 15$ Hz . These parameters were motivated by the model in Deco's paper and chosen so that F_1 and F_2 are stable.

Applying the Moments Method to the Stochastic Wilson-Cowan Model

The behavior of Equation (1) is fundamentally probabilistic. Its behavior can be studied via repeated numerical simulations, but these are resource-intensive and do

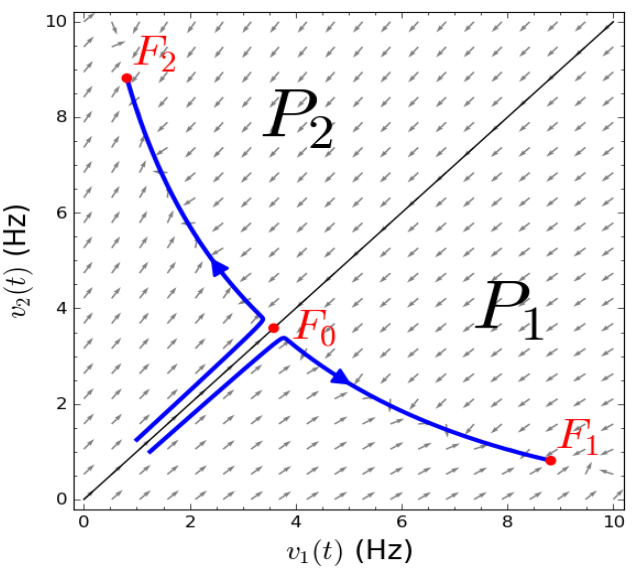

Figure 2. The phase plane of the non-stochastic version of Equation (1). The time evolution of the system is shown in blue for two example starting points: (1.25, 1) and (1, 1.25). The three fixed points of the system are shown as red dots. The arrows show the normalized direction field. Regions to either side of the $v_1=v_2$ line are labeled as P_1 and P_2.

not directly yield results that can be used to describe the dynamics of the neural network analytically. It is possible to compute the first and second moments of the random variable $v_i(t)$ at time t using the moments method (see Figure 3 below) where $\mu_i(t)$ is the statistical mean of $v_i(t)$ at time t for i=1,2, γ_{ij} are elements of the covariance matrix, and $\mu_i = \lambda_i + \sum^2_{j=1} w_{ij}u_j(t)$ (Deco & Martí, 2007). These calculations assume that the bivariate distribution of the vector $\mathbf{v}(t)$ is Gaussian, and this fact will limit the predictive power of the analysis that we will perform in section 2. Notice that the variance β of $\xi_i(t)$ shows up in Equations (2) through (6). While the time evolution of the means of random variables v_1 and v_2 is not affected by β, the time evolution of the variance of the random variables is.

An Analytical Approximation of Binary Decision Bias Under Varied Variance

In the non-stochastic version of Equation (1), as we have seen, the attractor network's initial state determines if it will end up at F_1 or F_2 as time progresses. Initial states in P_1 will cause the system to end up at F_1, and initial states in P_2 will cause the system to end up at F_2. As soon as noise is introduced to Equation (1), this changes.

As the variance of $\xi_i(t)$ increases, the state of the attractor network gets more "jittery" as it evolves in time and moves about the phase plane. The more "jittery" the state vector becomes, the more likely it is to jump from one side to the other of the v_1=v_2 line and thus switch basins of attraction in some fixed time interval. This can be observed via numerical simulation.

Figure 4 depicts a histogram[1] of the component of the result of one thousand simulations of Equation (1) for each of β = 0.1 and β = 0.6 for 2000ms starting at the point F_0+(-½, ½). The simulations were done using the Euler–Maruyama method with a step size of 0.01ms and the variable values given in section 1. Table 1 summarizes the results of seven batches of one thousand simulations conducted in the same way. The v_1 value of F_2, denoted $F_2^{(v_1)}$, along with the v_1 values of F_1, F_0, and the starting point F_0+(-½, ½) are shown on the histogram as vertical lines. Over two seconds of simulated time, the neural network tends to settle down near either F_1 or F_2, corresponding to each of the two possible binary decision states. It can be seen that varying the variance of the randomness consumed by the binary decision-making neural network biases its decisions and that the size of the bias increases as the variance increases.

We will construct an analytical approximation of binary decision bias under varied variance motivated by these observations and justify the approximation using previous observations about the geometry of the phase plane of the non-stochastic version of Equation (1). Equations (2) through (6) completely define the bivariate normal joint density of v_1 and v_2 at time t, which we will denote as $N(\boldsymbol{\mu}(t),\boldsymbol{\gamma}(t))$ where $\boldsymbol{\mu}(t)$ is the mean vector and $\boldsymbol{\gamma}(t)$ is the covariance matrix. It follows that:

$$P(\boldsymbol{v} \in P_i, t = t_f) \approx \int_{P_i} N\left(\boldsymbol{\mu}(t_f), \boldsymbol{\gamma}(t_f)\right) dA \stackrel{\text{def}}{=} P_{approx}(\boldsymbol{v} \in P_i, t = t_f) \quad (7)$$

holds when the final time t_f is small enough that the distribution of final states is approximately normal. Here we introduce the notation $P_{approx}(\mathbf{v} \in P_i, t = t_f)$ which is used

$$\tau \frac{d\mu_1(t)}{dt} = -\mu_1(t) + \phi(u_1) + \frac{1}{2}\phi''(u_1)\sum_{j=1}^{2}\sum_{k=1}^{2} w_{1j}w_{1k}\gamma_{jk}(t) \quad (2)$$

$$\tau \frac{d\mu_2(t)}{dt} = -\mu_2(t) + \phi(u_2) + \frac{1}{2}\phi''(u_2)\sum_{j=1}^{2}\sum_{k=1}^{2} w_{2j}w_{2k}\gamma_{jk}(t) \quad (3)$$

$$\tau \frac{d\gamma_{11}(t)}{dt} = -2\gamma_{11}(t) + \sum_{l=1}^{2}[w_{1l}\gamma_{1l}(t)\phi'(u_1) + w_{1l}\gamma_{1l}(t)\phi'(u_1)] + \beta^2 \quad (4)$$

$$\tau \frac{d\gamma_{22}(t)}{dt} = -2\gamma_{22}(t) + \sum_{l=1}^{2}[w_{2l}\gamma_{2l}(t)\phi'(u_2) + w_{2l}\gamma_{2l}(t)\phi'(u_2)] + \beta^2 \quad (5)$$

$$\tau \frac{d\gamma_{12}(t)}{dt} = -2\gamma_{12}(t) + \sum_{l=1}^{2}[w_{2l}\gamma_{1l}(t)\phi'(u_2) + w_{1l}\gamma_{2l}(t)\phi'(u_1)] \quad (6)$$

Figure.3 Formulas (2) through (6).

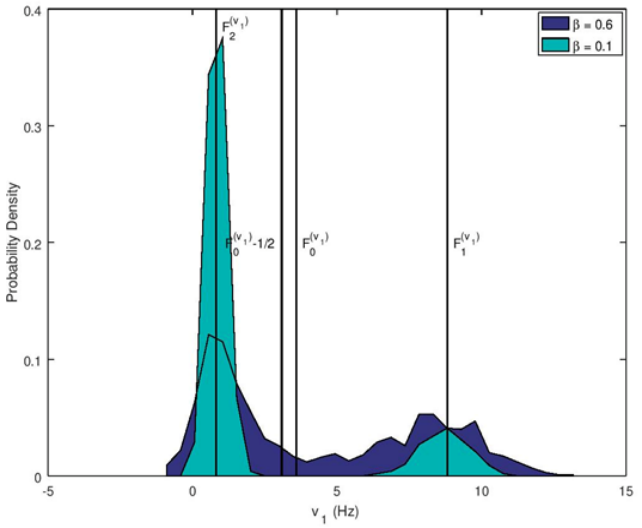

Figure 4 . A histogram of the v_1 component of the result of one thousand simulations of equation (1) for two variances initialized at $\mathbf{v} = F_0+(-½, ½)$ and ran for 2000ms with a step size of 0.01ms.

β (Hz^2)	$P(\boldsymbol{v} \in P_2, t = 2000)$
0	1.000
0.1	0.819
0.2	0.722
0.3	0.648
0.4	0.605
0.5	0.576
0.6	0.532

Table 1. For each variance value, Equation (1) was initialized at $\mathbf{v} = F_0+(-½, ½)$ and executed 1000 times for 2000ms of simulated time with time steps of 0.01ms. Each final state was classified as being in either P_1 or P_2, and the ratio of P_2 states to P_1 states are shown beside each variance value.

to denote the value of our approximation of $P(\mathbf{v} \in P_i, t = t_f)$, which we have previously only computed via repeated and tedious numerical simulation. Note that the distributions of final states are not normal over long time intervals, as shown in Figure 4. However, since in the long term, $\mathbf{v} \in P_i$ implies that the decision corresponding to F_i has been reached, we can take Equation (7) to be an analytical approximation of the influence of varied variance on decision bias over short periods of time[2]. The integral can be numerically approximated using a Monte-Carlo method. It is sufficient to programmatically sample a large number of points from $N(\boldsymbol{\mu}(t),\boldsymbol{\gamma}(t))$ and then count the cases where, without loss of generality, $v_i > v_j$ and compute a point-count ratio between points in P_i and the total point count to approximate $P(v \in P_i, t = t_f)$. This kind of Monte-Carlo integration is a standard technique for numerically evaluating integrals and has applications beyond probability theory (Weinzierl, 2000, p. 11).

In Figure 5, Equation (7) has been evaluated at many variances for 30ms starting at initial state $F_0+(-½, ½)$ with every element of the covariance matrix initialized to zero using the aforementioned Monte-Carlo method. In Figure 6, simulation data used to create Table 1 is visualized to show that in this particular case, the probability distribution of **v** values is roughly normal at t =30. As the variance of the randomness consumed by the binary decision-making neural network is increased, the probability that the state vector will still reside in P_2 at the end of the 30ms time window decreases. There are regions in Figure 5 where small changes in variance can induce a significant decision bias. For example, according to the approximation, a shift in variance from 0.05 Hz^2 to 0.1 Hz^2 will induce a 14.6% increase in the probability that the state vector escapes P_2. While the parameters of the simulation in Figure 5 were chosen somewhat arbitrarily, the same sort of behavior can be observed for a wide variety of parameters when the state vector is close to the $v_1=v_2$ line at t = 0.

DISCUSSION

Here we have derived an approximation that describes how changes in the variance of the randomness consumed by a binary decision-making neural network bias the network towards one decision or the other. The approximation assumes that given a fixed initial state, the probability distribution of final states at t_f is roughly normal. The "moments method" that the approximation is based on is applicable to a wide range of neuronal structures and was originally created as a generic tool to work with equations similar to Equation (1) (Deco & Martí, 2007; Rodriguez & Tuckwell, 1996). Additionally, we have shown that under some circumstances, changes in variance of approximately 0.05 Hz^2 can induce a decision bias on the order of 10%. These results come as no surprise, given Deco's results showing that for simulations of Equation (1) initialized at decision states (the points we have labeled F_1 and F_2), larger variances make it more likely that the simulated attractor network settles down near the decision state that it was not initialized at (Deco & Martí, 2007, p. 9). When viewed as a computational device, a function (one of several) of the stochastic binary decision-making neural networks stud-

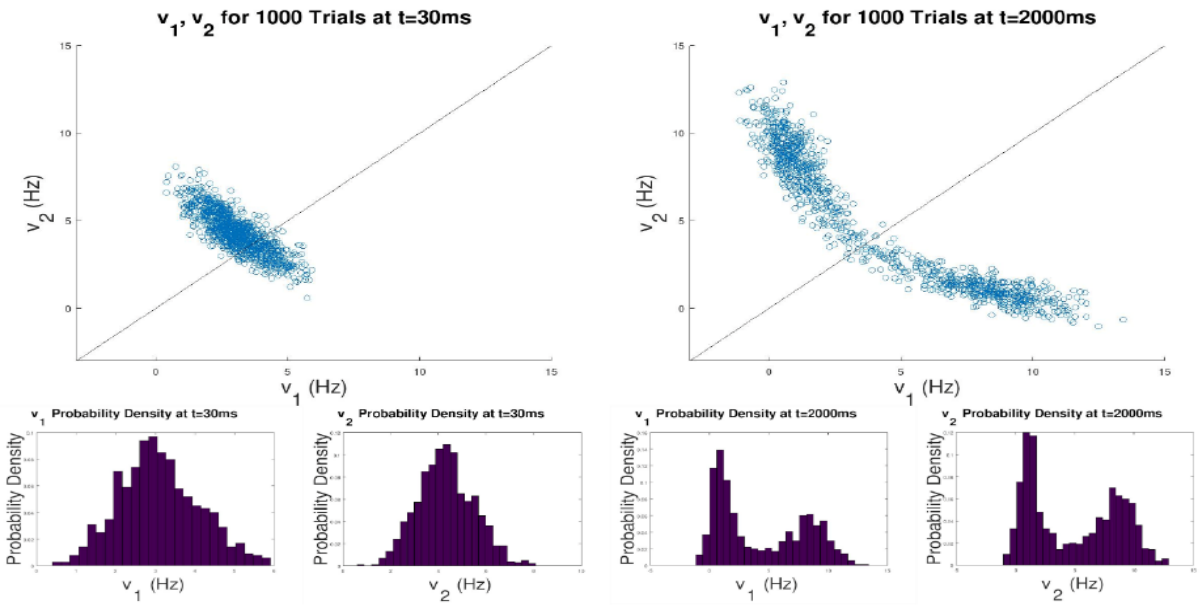

Figure 6. Simulated **v** values from the 1000 trials used to generate the β = 0.6 row of Table 1. At t =30ms, the **v** values are roughly normally distributed. At t = 2000ms, the **v** values are no longer normally distributed.

ied here is to transform small changes in the variance of the randomness that they consume into network-level electrochemical signals that can be consumed by other structures in the brain.

Assuming that the majority of stochastic brain structures respond in similar ways to small changes in the variance of the randomness they consume, a brain-independent consciousness able to exert a small influence over the variance of said randomness would likely be able to exercise significant control over the functioning of the brain. While the brain is full of small populations of strongly interconnected cells that perform tasks in relative isolation, these populations of interconnected cells are themselves interconnected, and the level of interconnectedness between them can be quantified (Tononi et al., 1994). The brain is highly recursive. A bias of equivalent magnitude to the roughly 10% bias predicted by our model when exerted on the output of a generic stochastic neural network over the duration of a single task does not seem, at face value, to offer very much control over the operation of the brain. However, due to the interconnectedness of stochastic brain structures, a bias of this size, when applied continuously over all stochastic brain structures, will compound very quickly. The computational result of a given stochastic neural network is dependent on both its input and the randomness that it consumes. If the randomness that a given structure (name it structure A) consumes is slightly biased, and its input originates from the output of another stochastic structure (name it structure B) subject to a bias of the same magnitude, then the net effect of the biasing influence on structure A will be greater than the net effect of the biasing influence on the randomness that structure A consumes. The conscious will of a brain-independent consciousness could be accumulated by stochastic neural networks over time if the results of slightly biased computations are continually being fed back into biasable structures as input.

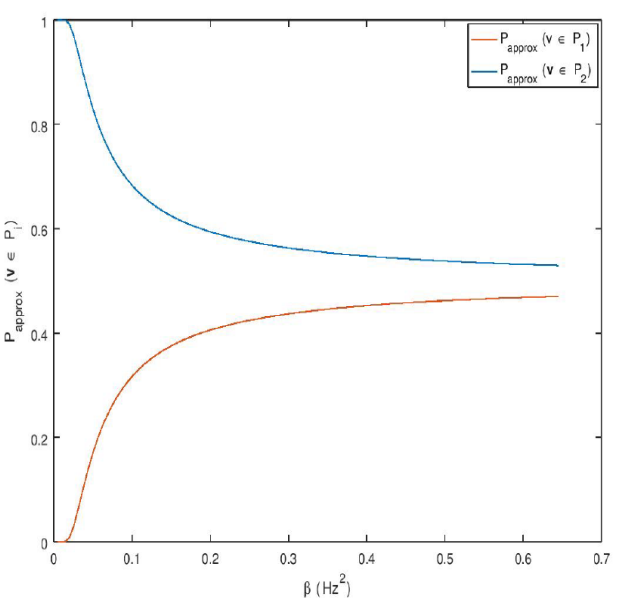

Figure 5. Equation (7) evaluated at a variety of variance values for 30ms starting at **v** = F_0 + (-½, ½).

While there are many conceivable ways for a stochastic system to be made sensitive to the effects of small changes in the variance of the randomness that it

consumes, the binary decision-making structures that we studied here accomplish this in a very specific way. The state of the system is initialized near a separatrix on the phase plane (the $v_1 = v_2$ line in the case of our model), and because of this, changes in the variance of the underlying randomness being consumed by the system effect how far the state of the system is expected to wander from its starting point over any small, fixed time interval (small enough that the probability of falling into a stable fixed point is negligible), which in turn effects how likely the system is to cross the separatrix. The system is prone to undergoing noise-induced state transitions. This type of system is common in nature. Many natural systems, from lasers to populations of viruses, are prone to undergo the same sort of noise-induced transitions (Forgoston, 2018). In the human brain, unlike in many similar systems, the noise involved in noise-induced transitions is presumably produced by a noise source that is sensitive to micro-PK. It is reasonable to suspect that any stochastic system sufficiently like the stochastic systems in the brain that derives its randomness from a noise source sensitive to micro-PK should be able to be influenced by a brain-independent consciousness in the same way that we hypothesize the brain is.

A complete theory of brain-independent consciousness must describe the dynamics of three distinct but related processes:

1. *Consciousness-to-brain communication.* How does a brain-independent consciousness drive brain activity?
2. *Brain-to-consciousness communication.* How are electrochemical signals in the brain experienced by a brain-independent consciousness?
3. *Binding.* Why is it that brain-independent consciousnesses are seemingly bound to their bodies? Why can't my will raise your arm?

The results presented here only attempt to present a partial explanation of item (1) of this list.

Our model assumes that a brain-independent consciousness can exert influence on the seemingly random firing times of pyramidal neurons, but it does not address how this influence is achieved in the first place. This problem, however, has been previously examined by other authors. It has been theorized that at the most basic level, consciousness-to-brain communication is achieved via the Quantum Zeno Effect, abbreviated QZE (Stapp, 2015). In a nutshell, the QZE is the observation that rapid, repeated observations of some measurable aspect of a quantum system will slow the time-evolution of the system in a way that the measured aspect will tend to (probabilistically, of course) become "frozen" in the state it was first measured in (Misra & Sudarshan, 1977). It may be the case that rapid, repeated probing of quantum processes in the brain by consciousness biases the states of these processes by holding them in fixed states for long periods of time (Stapp, 2015). If there are quantum processes occurring inside of pyramidal cells, then "freezing" their states for a period of time may affect the overall behavior of the cells, affecting the time at which they fire. When performed over large groups of cells simultaneously, the theorized "quantum state freezing" could conceivably produce changes in the randomness consumed by entire stochastic brain structures, perhaps causing the sorts of variance changes studied here.

If biased noise is being consumed by stochastic brain structures, observing the effects of the biased noise should be straightforward using conventional techniques. It is likely possible to directly falsify predictions made by Equation (7). *In vivo* observations of stochastic decision-making neural networks played a large role in deriving the biologically realistic models of these networks that we studied here (Deco et al., 2007; Wang, 2002). In the opinion of the author, constructing falsifiable dynamical models describing the interaction between consciousness and network-level neuronal activity should be a primary goal of Parapsychology going forward.

Implications and Applications

If it is indeed the case, as we have hypothesized, that stochastic structures in the human brain integrate consciousness-to-brain communications into network-level computations being carried out in the brain, then the results presented here have immediate implications for medicine. If a specific set of stochastic attractor networks in a patient's brain becomes damaged, it should, in theory, be possible to replace each network with a microcontroller that, when biologically necessary, numerically solves the appropriate set of stochastic Wilson-Cowan equations using randomness derived from an entropy source that is suspected to be sensitive to micro-PK (such as a quantum random number generator). Such a device, if constructed[2] with an appropriate, micro-PK sensitive source of randomness, may at least partially restore the consciousness-to-brain communication previously occurring in the replaced brain tissue. Perhaps this might allow some people immobilized due to brain damage to regain some degree of mobility.

NOTES

[1] There are no constraints on Equation (1) that stop v_i from randomly assuming negative values. Neither population

of neurons can fire at less than 0 Hz, but no attempt to reconcile this will be made here. This only becomes noticeable when some F_i is close to zero on one axis, as is the case here where $F_2^{(v_1)} \approx 0.8$.

[2] Equation (7) can be used to compute the effect of a small shift Δv in the initial value of v (which would correspond to a short-lived mean shift in the values of the $\xi_i(t)$ random variables) as $\Delta P(\mathbf{v} \in P_i, t=t_f) = P(\mathbf{v} \in P_i, t=t_f$, initial v of $v_i+\Delta v) - P(\mathbf{v} \in P_i, t=t_f$, initial v of $v_i)$ but small shifts in initial v values do not have a large effect on $P(\mathbf{v} \in P_i, t=t_f)$ in the same way that small shifts in β do, so a detailed discussion of this is not undertaken here

[3] Such a device could clearly be constructed in the general case, as making a reasonably powerful computer simulate the requisite Wilson-Cowan equations in a biologically feasible amount of time is simply a matter of increasing the simulation step size until the computer can perform the task. However, it is not immediately clear that such a device could currently be constructed to only occupy the volume of the tissue that it is replacing. Devices such as this, if found to work as intended, might have to be worn outside of the body.

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