The effect of the recovery variable parameters on oscillating Izhikevich networks

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Abstract

Recent studies have corroborated that the brain organises dynamically into functional groups, and that oscillation is a central component in this process. How oscillation is mediated, however, remains unclear. One approach to illuminating its emergence is through computational modelling of biologically realistic, yet tractable, oscillatory neural networks. In a recent paper, Oliveira et al. (2019: Neurocomputing), analyse Izhikevich spiking neural networks, containing a mixture of excitatory and inhibitory neurons. They find that the resulting population oscillation rate rate may be predicted by a heuristic function, which may be utilised in constructing models. By extending the aforementioned work, we attain a computationally efficient implementation which captures the emergence of synchronised population bursting. We uncover and elaborate on why it occurs in this specific model, and demonstrate that the population burst rates rely heavily on the sub-threshold oscillations of the single neurons. Further, we find that there is a deterministic and approximately linear relationship between the the two, which also holds for mixture models. Our results clarify the effect of the specific parameters on oscillatory behaviour in the Izhikevich model, and explain why population bursting emerges in this model scheme. These findings may be used in order to more accurately characterise rhythmically bursting oscillatory network models bursting at specific rates. Lastly, our findings suggest a possible mechanism for synchronous neural oscillation in vivo.

Keywords: Izhikevich, Oscillations, Recovery, Sub-threshold

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1. Introduction

The theory of neuronal group selection (TNGS) posits neuronal groups as functional units within the brain. Since the proposal of TNGS, studies have corroborated the existence of such units. With a myriad of different brain areas and rhythms, there is correspondingly a possible myriad of modes of synchrony with its underlying mechanisms remaining to be uncovered. One type of a highly synchronous neural group is Central Pattern Generators (CPGs), which mediate rhythmic behaviour within the nervous system such as respiration and motor control. This work focuses on a subset of neuronal behaviours that occur within biology, by analysing a highly connected network of inhibitory and excitatory neurons, with a parallel to that of Central Pattern Generators (CPGs) in that the population bursts rhythmically. It does so by using the Izhikevich neuron model [1], consisting of two coupled differential equations. The model is chosen due to its trade-off between computational efficiency, and biologically meaningful behaviour - being able to exert a rich array of neuronal behaviours as observed in vivo, including regular spiking, intrinsic bursting, and low-threshold spiking, the two latter being the primary modes observed in this work. As stated by Izhikevich [1, 2]; when the value determining the sensitivity to sub-threshold oscillations increases towards a specific value, Izhikevich neurons reach their bifurcation point. Increasing the value thereafter entrains regular firing without any external stimulus, due to sub-threshold oscillation. Given the aforementioned, we wanted to investigate the relationship between neuronal sub-threshold oscillations and network behaviour, by extending the model of [3]. We hypothesised that the network behaviour would depend on the sub-threshold oscillation rate of the single neurons, and thus also that the single neuron firing rate may be used to predict the emergent network burst rate. Whether the mechanism for synchronous population bursting is biologically plausible is discussed. We also draw parallels to the neurophysiological aspects of the neuron models, discussing possible implications within the biological cellular machinery.

2. Methods

We implemented and replicated the results of [3], and further extended their work by extending (1) the parameter-space, (2) a comparative analysis of the single neuron behaviour and the network behaviour as a whole, and (3)

by introducing Gaussian noise to a subset of neurons, rather than depolarising a single, random neuron each time step.

Formally, the Izhikevich neuron model employed is defined by,

$$\frac{dv}{dt} = 0.04v^2 + 5v + 140 - u + I_{\text{syn}} \tag{1}$$

$$\frac{du}{dt} = a(bv - u) \tag{2}$$

and if $v \geq 30$ mV, then $v \leftarrow c$ and $u \leftarrow u + d$.

This work is primarily focused on the effect of the parameters a and b in regularly spiking networks, with two classes of neurons having $(c, d) = (-65, 8)$, and $(-65, 2)$, respectively.

The synaptic model is defined by a simplified differential equation,

$$\frac{dg}{dt} = -\frac{g}{\tau_g} \tag{3}$$

$$I_{\text{syn}} = \sum_{i=1}^{N} g_i w \tag{4}$$

where g is reset to 1 upon spiking, and otherwise decays exponentially, as defined in Eq. 3. I_{syn} is modelled per neuron as the sum over each synaptic conductance times a synaptic weight constant $w \in [10, 20]$ (Eq. 4). At each time-step of the model simulation, a random neuron is stimulated by $I_{\text{external}} = 100\,\mu\text{A}$.

2.1. Experiments design

Similar to [3] we performed 30 trials per model parametrisation, and calculated the mean population burst rate for the converging models. To extend the parameter search space, we performed experiments using different values for the free variables a, b, w, n, τ_g. That is, we calculated the average population burst rates for converging models for 30 models for the set of parameters generated by calculating the cross product of the sets $a = [0.015, 0.022, ..., 0.050]$, $b = [0.15, 0.26, ..., 0.70]$, $s = [10, 20]$, $g = [5.0\ ms, 6.5\ ms]$, $n = [500, 1500]$.

The firing frequency was determined by computing the frequency with the highest energy in the average power spectrum density. If synchronous behaviour did not emerge, the frequencies of the single neurons, operating

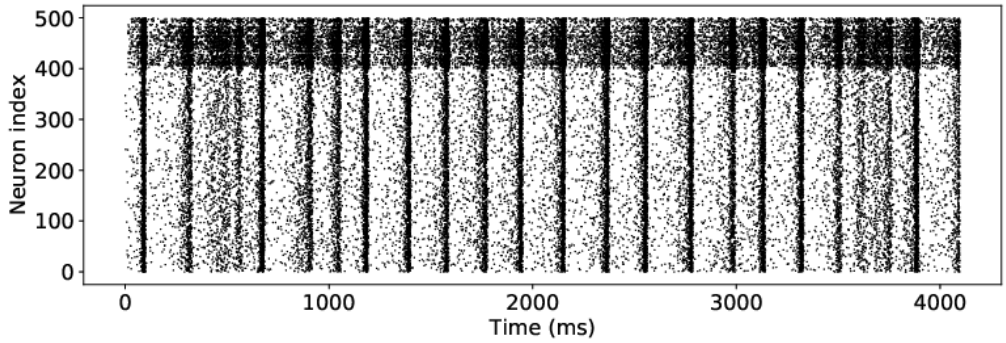

Figure 1: Raster plot for a network model consisting of $n = 500$ neurons, with parameter values $a = 0.005, b = 0.42$, a weight of $w = 11$, and $\tau_g = 5.0\,\text{ms}$.

on a timescale several times faster than synchronous population bursting, dominates the energy spectrum, and thus we are guaranteed that the model is bursting synchronously for (1) energies close to 1, and (2) energies for frequencies in the lower end of the spectrum. This was also confirmed by analysing the Fourier transform of the population average membrane potential signal.

3. Results

When neurons are close to their bifurcation point, they are more easily excited to an extent such that they fire bursts of action potentials. In the configuration of the model analysed in this paper the neurons are almost always brought into this regime. Interestingly, this synchronises the spiking of the network as a whole. Furthermore, the emergent frequency at which the network fires is in large determined by the single excitatory neuron behaviour. We attained comparative results when running experiments for the same parametrisations as in [3].

We observed that neurons fired bursts of action potential whenever spiking (see figure 1). The reason for this may be understood if considering the phase portrait of a neuron (figure B.7) for certain constant values and initial conditions.

Interestingly, when comparing the population burst rate to the single neuron firing rate, a clear linear relationship was found, as shown in figure 4. Further, we observe that there is an exponential relationship between the

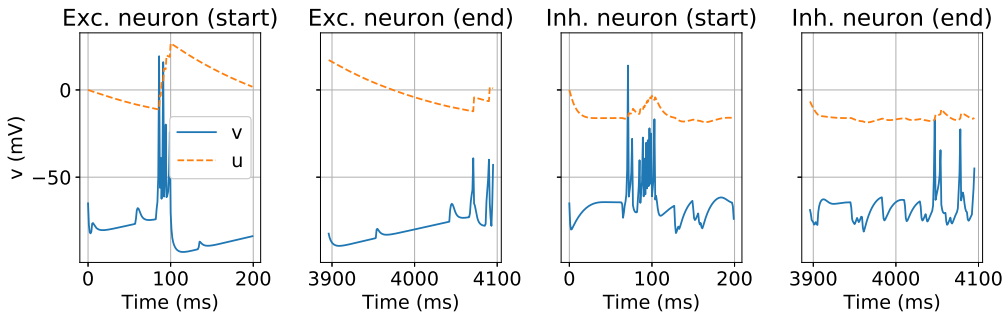

Figure 2: Voltage traces of two randomly chosen neurons for two time intervals. Inhibitory neurons fire longer bursts, due to their parametrisation of $(a, d) = (0.1, 2)$.

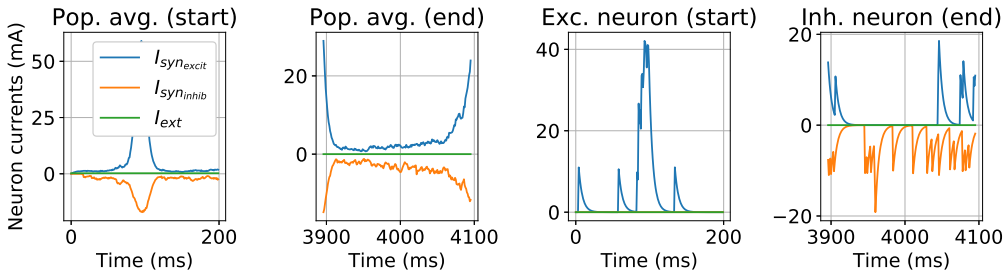

Figure 3: Currents at two time intervals for the population average, and two random neurons. Excitatory and inhibitory currents mirror one another on average due to synchronicity.

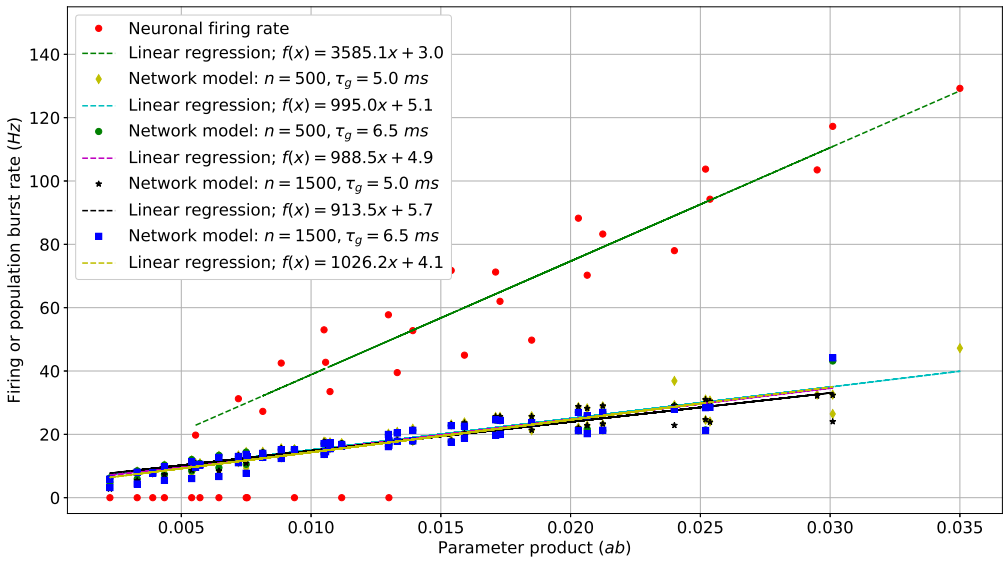

Figure 4: Neuronal sub-threshold oscillation rate and synchronised population burst rate reveal a linear relationship between ab and the burst rates for converged (synchronous) models. Data points for non-synchronous models are not included. Convergence to synchronous bursting is less likely for higher higher parameter-products and firing rates.

sensitivity variable, and its time scale, given constant firing rates.

When considering the single neuron sub-threshold oscillation firing rate, there is a deterministic relationship between the rate and the parameters a and b, as exemplified in in figure 5. This may be written formally as,

$$F(a, b) = a_c a + b_c b + c, \tag{5}$$

$$F(a, b) \approx c_1 ab + c_2 \tag{6}$$

$$b \approx f(a) = k_1 a^{k_2} \Leftrightarrow a \approx f(b) = k_3 b^{k_4}, \tag{7}$$

where (...).

In order to understand why this is, we may look at the coupled differential equations. A phase plot illustrates why the trajectories give rise to regular spiking of given frequency, where several spikes need to occur before the bifurcation point is reached and a refractory period is induced, as

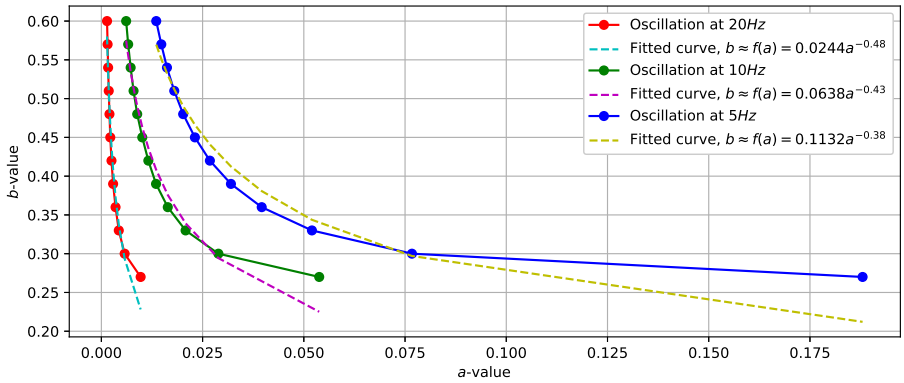

Figure 5: Considering a constant single neuron oscillation rate for different values of a and b, with $d = 8$.

shown in figure 2. Note however that this only holds for certain intervals of parameters. In the network case, however, the neuronal signals is strong enough to separate the trajectories such that spiking occurs. In fact, due to the topology and weights configuration, bursting occurs. We may describe the system's behaviour analytically as,

$$F(a, b) \approx 1000ab + f_{\min}, \tag{8}$$

where $f_{\min}$ is the minimum possible population burst rate before the activity becomes desynchronous, typically $f_{\min} \approx 4$ Hz.

In sum, a and b determine the neuronal sub-threshold oscillation rate, upon which the network burst rate depends. Thus, the population burst rate may be predicted by the single neuron firing rate. As for the inhibitory neurons; there appears to be only a minor effect on the spike frequency from their negative currents.

4. Discussion

When considering the phase portrait in figure B.7, it may be seen that given a non-changing stimulus, intersecting nullclines would lead a neuron to firing regularly. However, as may be seen in the raster plot, the resulting behaviour at the network level is effectively the induction of a refractory period, where the recovery variable 'cools down', or decreases succinctly for altering the phase portrait such that the variables' trajectories yet again do

not intersect, resulting in bursts at the next time of spiking. This is due to that the signal I is still greater than the subtracted value for u in the system. Thus, incrementing of u by $u \leftarrow (u+d)$ does not induce a refractory period for the neuron until this condition changes. This leads to a series of consecutive spikes and increments of u by d, or a burst, eventually inducing a refractory period. It is worth mentioning that weights $w \geq 1$ incorporate a constant for the difference between the reverse and membrane potential (typically denoted $(E_{syn} - V_{post})$), and as such may be regarded as biologically plausible.

Note also that as the inhibitory neurons have a slightly higher time scale a for the recovery variable u, they may impose a slightly higher negative current on the population, inducing a slightly longer build-up of the membrane potential v.

Excitatory neurons drive synchronisation - bio. plausibility? Bursting occurs due to the current signals, which is a result of the topology and synaptic configuration.

Inhibitory neurons are close to their bifurcation point; a slight stimulus leads to spiking. Little effect on network. Inhibitory neurons generally burst more due to: (1) a shorter refractory period (a higher a-value), and (2) a lower after-spike reset for the recovery variable (lower d)

For lower weights w, I_{syn} is lower, and the network desynchronises. This also holds for having fewer synapses per neuron, n_{syn}. It would be interesting to look at the behaviour when weights approach a 'critical value'. It might be hypothesised that bursting occurs due to the strength of the synaptic current I_{syn}. Given the above, formally,

$$I_{\text{syn}} \propto n_{\text{syn}} w, \tag{9}$$

we may test the effect on the average neuronal current $\hat{I}$, where,

$$\hat{I} \sim \frac{df(a,b,I)}{dt} \hat{n}_{\text{syn}} w \tag{10}$$

We hypothesise that this would lead to fewer bursts, and yet synchronous firing due to bursting, until a point where bursting ceases, and the activity becomes less synchronous. This hypothesis was confirmed in initial experiments.

small bifurcation analysis; phase portrait showing saddle-node bifurcation, along with example u and v plots?

ion channel activation not modelled directly. however, recovery variable u captures refractoriness, and thus is the parallel to the ion channel activation/inactivation. Izhikevich equations; *one* time scale. although abstracted, it still models the membrane permeability changes implicitly, in that the time scale and induced refractoriness is due to the temporal dynamics of the sodium-potassium ion channels.

Rapid sodium-influx *can* trigger a burst of action potentials(?). Thus, a potential mechanism for synchronous rhythm generation can be mediated as demonstrated in the model of this paper.

reverse potential and membrane potential difference $(V_m - V_{\text{ion}})$ not considered in the synaptic modelling.

5. Conclusion

It was observed that for most models in the parameter-intervals employed, a predictable population burst rate will emerge. The reason for this was uncovered, and shown to be due to the bursting dynamics of the excitatory neurons, almost instantaneously leading to spiking throughout the entire network. Action potentials then reverberate until the phase planes for the coupled two differential equations have intersecting nullclines. While this enables shifting neuronal behaviour into regular spiking, it effectively leads to a 'global' refractory period, due to the network's connectedness.

Within the bursting regime of Izhikevich neurons, coupling enabling signal reverberation may ultimately lead to synchronised refractory periods and population bursting. The dynamics constituting the mechanism are described within this paper, and an intuition, as well as example functions for predicting the resulting network behaviour, is provided. It is unknown whether a similar mechanism for producing rhythmic behaviour and synchronicity is at play in vivo. Using the Izhikevich neuron model which entrains biologically meaningful behaviour, this might suggest that this mechanism is one which could be at play within biology. Nevertheless, it is worth being aware of that this is a mechanism which might emerge when simulating neurons using differential equations.

References

[1] E. M. Izhikevich, Simple model of spiking neurons, IEEE Transactions on Neural Networks 14 (2003) 1569–1572.

(a, b)-intervals by model	$(a, b) =$ $((0.0100, 0.205),$ $(0.070, 0.210))$	$(a, b) =$ $((0.0050, 0.0075),$ $(0.3150, 0.455))$	$(a, b) =$ $((0.0225, 0.0400),$ $(0.3500, 0.655))$	$a = 0.05,$ $b = [0.655, 0.700]$
Neuron	0	7.3-10.8	38-80	121-130
$n = 500, \tau_g = 5.0\ ms$	4.3-5.2		19.9-21.7	41.5-45.9
$n = 500, \tau_g = 6.5\ ms$	4.1-6.1		19.6-21.2	N/A
$n = 1500, \tau_g = 5.0\ ms$	4.0-5.1		20.2-21.6	42.1-47.4
$n = 1500, \tau_g = 6.5\ ms$	4.1-6.1		19.9-21.7	N/A

Table B.1: A table summarising the firing rates (Hz) (i.e. subthreshold oscillation rate for the neuron, and synchronised population bursting average for the networks) for a and b parameter intervals that are close in parameter-space.

[2] E. Izhikevich, Which Model to Use for Cortical Spiking Neurons?, IEEE Transactions on Neural Networks 15 (2004) 1063–1070.

[3] L. D. Oliveira, R. M. Gomes, B. A. Santos, H. E. Borges, Effects of the parameters on the oscillation frequency of Izhikevich spiking neural networks, Neurocomputing (2019).

[4] D. Goodman, Brian: a simulator for spiking neural networks in Python, Frontiers in Neuroinformatics 2 (2008) 1–10.

Appendix A. Implementation

We used the Brian 2 spiking neural network simulator [4] for implementing and running the experiments for this paper. The code is openly available, and may be found at https://github.com/williampeer/Izhikevich_network_modelling.

Appendix B. Results

Replication of tables,

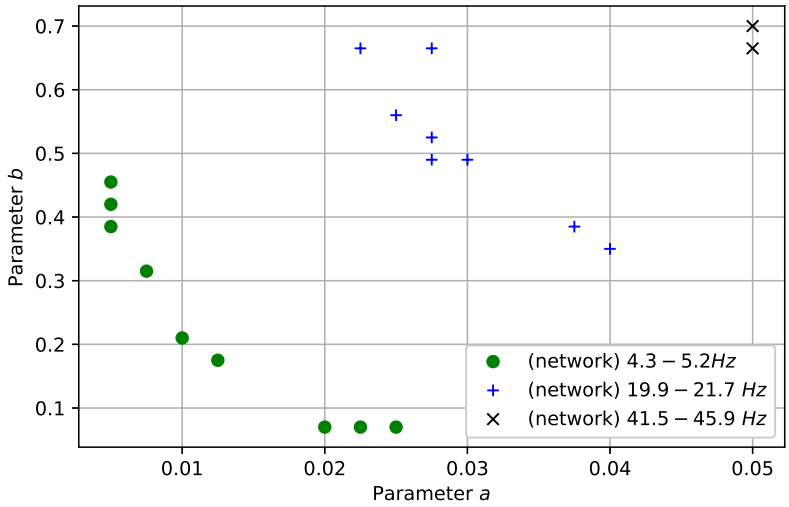

Figure B.6: Clusters of firing rates for different values of a and b, with $n = 500$, and $\tau_g = 5\ ms$. Note how the clusters are centred around constant values for the product of a and b.

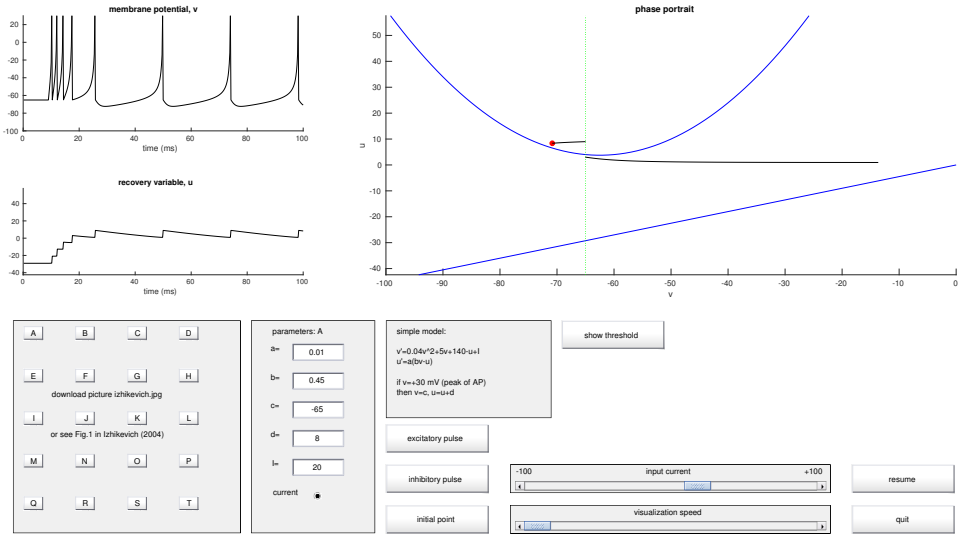

Figure B.7: Note that the nullclines do not intersect before the recovery variable u has been incremented several times by the after-spike reset variable d, which results in that the nullclines intersect.

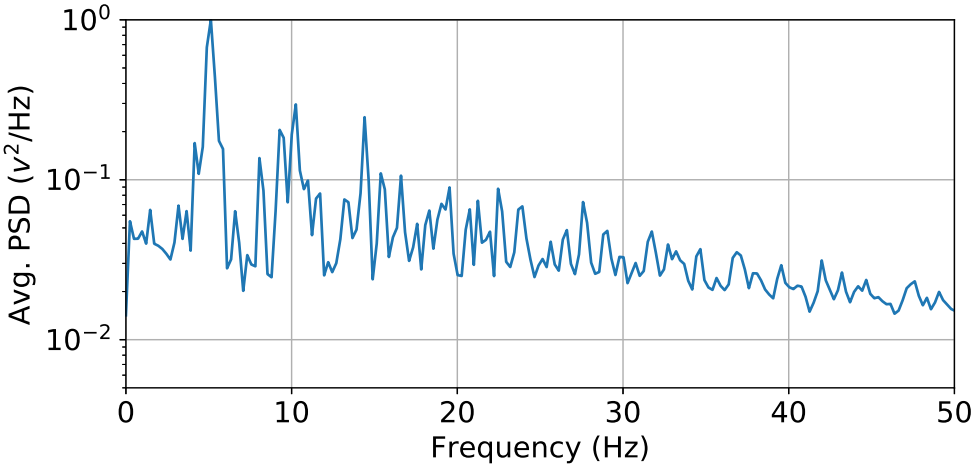

Figure B.8: The power spectrum density reveals $\approx 4.15\,\mathrm{Hz}$ as the most prominent burst rate.