Viewing rate-based neurons as biophysical conductance outputting models

Martinius Knudsen[1], Sverre Hendseth[1], Gunnar Tufte[2], and Axel Sandvig[3]

[1] Department of Engineering Cybernetics, NTNU, Norway
[2] Department of Computer Science, NTNU, Norway
[3] Department of Neuromedicine and Movement Science, NTNU, Norway

Abstract. In the field of computational neuroscience, spiking neural network models are generally preferred over rate-based models due to their ability to model biological dynamics. Within AI, rate-based artificial neural networks have seen success in a wide variety of applications. In simplistic spiking models, information between neurons is transferred through discrete spikes, while rate-based neurons transfer information through continuous firing-rates. Here, we argue that while the spiking neuron model, when viewed in isolation, may be more biophysically accurate than rate-based models, the roles reverse when we also consider information transfer between neurons. In particular we consider the biological importance of continuous synaptic signals. We show how synaptic conductance relates to the common rate-based model, and how this relation elevates these models in terms of their biological soundness. We shall see how this is a logical relation by investigating mechanisms known to be present in biological synapses. We coin the term 'conductance-outputting neurons' to differentiate this alternative view from the standard firing-rate perspective. Finally, we discuss how this fresh view of rate-based models can open for further neuro-AI collaboration.

Keywords: Artificial neural network · Spiking neural network · Computational neuroscience · Conductance models

1 Introduction

Progress in neuroscience research has been impressive. We now understand the central nervous system with increasing anatomical detail. Additionally, we are rapidly elucidating how the brain functions down to cellular and molecular resolution. Based on brain anatomy and cellular results, research can now also focus on understanding how the neural connectome is functionally integrated on macro, meso- and microscale levels. Central to future progress within this research field is the development of powerful computational tools to analyse the big data sets generated by the neural connectome. Within this area, the subfield Computational Neuroscience exists where researchers attempt to model and simulate these brain processes at different levels of abstraction; in particular, cell and network level modelling where biological detail is a prerequisite

as well as a constraint. In terms of network modelling, empirically derived phenomenological models such as *spiking neural networks (SNN)*[15] are commonly employed. While these network models seem to replicate biological dynamics, SNNs have had limited success with regards to task-solving.[4] However, there's growing research investigating how SNNs *can* be applied to tasks such as feature learning and control[14, 13, 7] using Hebbian-like[2] spike-timing dependent plasticity (STDP)[17, 25] learning rules. Research in the area has however yet to show rivalling results to that of conventional feed forward *Artificial Neural Networks (ANN)* employed by the machine-learning community.

AI-ANN research has achieved impressive success in a wide variety of applications by use of abstracted neuron models. However, the name 'artificial neurons' implying a replica of biology, tends not to be well accepted by the neuroscience community. While AI frequently looks to biology for inspiration, researchers in the field have granted themselves freedom from biological detail. Even leading AI researchers are now stating that ANN models are more mathematical constructs than attempts of modelling the complexity of biological neurons[18]. These artificial neurons negate the characteristic spiking behaviour prevalent in biological neurons, and instead output rates of neural spikes. For this reason, these artificial neurons are commonly referred to as 'firing-rate' models. A common and long-standing debate is whether or not firing-rates sufficiently capture the information transfer between neurons[3, 8, 11, 24].

SNNs are typically praised as being more biologically accurate, and are sometimes even referred to as the 3rd generation of neural networks[15].[5] In this paper we investigate commonalities between the commonly employed neuron models within the two fields, as well as their respective distinctions. In particular, we look at the resulting output signals of each model and see how they compare with respect to the receiving downstream neuron. We find that a fresh look at the standard rate-based model suggests that these models are more similar to their spiking counterparts than they may first appear. We also argue that simplified point spiking neurons may be biologically inferior to firing-rate models when considering synaptic transfer in network wide simulations. To support this argument, we investigate information flow between biological neurons, and compare this to spiking and firing-rate neurons. By this investigation, we further find that firing-rate neurons can be viewed as *conductance-outputting models*, and see how this alternative view increases these models' biological support and intuition. We finally discuss why this alternative view is of importance.

2 Background

2.1 Information transfer between biological neurons

Information flow between biological neurons is a complex process involving a cascade of events. A summarized illustration of this flow from one neuron to

[4] partly due to this not being the main priority of these models

[5] with rate-based models being the 2nd generation and threshold perceptrons being the 1st.

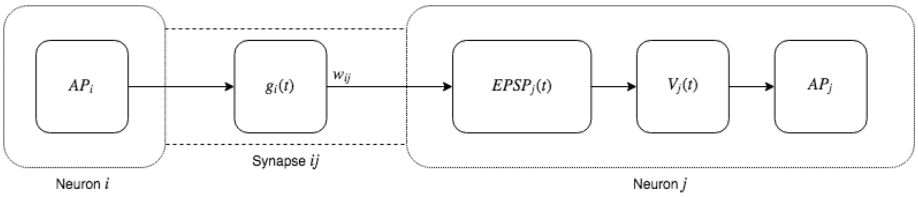

Fig. 1. Neural flow of information through an excitatory synapse between biological neurons. AP is the Dirac delta action potential, $g(t)$ is the synaptic conductance ranging from 0 to 1, w_{ij} is the synaptic efficiency, $EPSP(t)$ is the excitatory post-synaptic potential, $V(t)$ is the membrane potential at the soma.

the next is shown in Figure 1. Here, upstream neuronal action potentials (APs) cause the release of neurotransmitters into the synaptic cleft between two neurons. In excitatory synapses, this neurotransmitter is commonly glutamate[19], and can be viewed as a carrier of synaptic conductance[26, p.173]. The amount of glutamate released into the synaptic cleft follows the firing-rate of the presynaptic neuron[26, p.175], where high pre firing-rates yield greater releases of neurotransmitter up to some saturation point. The increase of glutamate in the synaptic cleft results in higher synaptic conductance as glutamate pairs with postsynaptic ionotropic receptors, such as AMPA[9], which open and become permeable to ionic currents. This allows for an influx of sodium into the postsynaptic dendrite, resulting in a depolarization known as an excitatory postsynaptic potential (EPSP). The more receptors available, the higher the synaptic weight/efficiency, and the stronger effect the presynaptic activation has on the postsynaptic neuron. The EPSP further propagates down the dendrite to the soma, depolarizing the somatic membrane potential $V(t)$. If the potential rises above a certain threshold, the neuron fires an action potential (AP). This AP in turn propagates down the axon causing the release of glutamate at a synapse between it and a downstream neuron, repeating the whole process there.

2.2 Information transfer in simple spiking neuron models

Many SNN simulations employ point neuron models and largely simplify the synapse in order to make these models computationally efficient[10]. These models often view discrete spike-events as the main source of information passing between neurons. Figure 2 illustrates the flow of information between spiking neurons. These discrete spikes influence postsynaptic membrane potential directly in proportion to the synaptic efficiency:

$$V_j = \sum_{i=0}^{N} \delta_i \left(V_i\right) w_{ij} \tag{1}$$

Here, N is the nr of presynaptic neurons i connected to a downstream neuron j, δ_i is the Dirac delta function which equals 1 when the membrane potential V_i

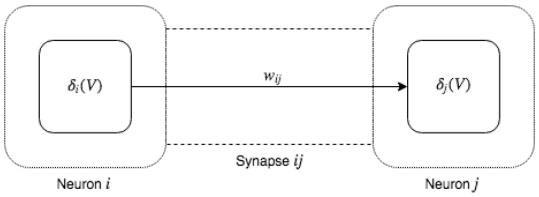

Fig. 2. Neural flow of information between simple spiking point neurons. δ is the Dirac delta function.

of the presynaptic neuron goes above some firing threshold and 0 otherwise. In these networks the weight is often updated through STDP[17, 25] which considers the timing of pre-post spiking events similarly to a Hebbian rule.

2.3 Information transfer between firing-rate neuron models

The standard ANN employs the firing-rate model[22]. These neurons do not model internal states such as spiking neurons do with their membrane potentials. They are thus typically static in nature,[6] whereas spiking neurons are highly dynamic. The function of the firing-rate neuron is modelled on the observation that biological neurons fire APs at rates proportional to the strength of their inputs[22]. By assuming that most of the information lies within these firing rates, firing-rate models simply convert upstream firing rates directly into new firing rates, foregoing the complex dynamics of membrane potentials and spikes.

Figure 3 illustrates this information exchange between firing-rate neurons. These neurons often employ some non-linear activation function on their inputs. Traditionally, this has been a squashing function which acts to bound the outputs between 0 (not firing) and 1 (firing at maximum frequency) using a sigmoid function f:[7]

$$\begin{aligned} a_j &= f\left(\sum_{i=0}^{N} a_i w_{ij} + b_j\right) \\ f(x) &= \frac{1}{1+e^{-x}} \end{aligned} \tag{2}$$

Here, a_i is the activation level (firing-rate) of the presynaptic neuron i, b_j is the neuron's natural bias (baseline activity even in the absence of input[28]), and w_{ij} is the weight (synaptic efficiency) between neuron i and j.

From a biological perspective, a firing-rate neuron seems to be an overly simplified model compared to spiking neurons and in particular compared to the complexity of real biological neurons. In addition, rate neurons seem to dismiss any possible information that may lie within the timing of spikes[16]. We shall

[6] apart from a few special versions e.g. continuous ANNs

[7] although several successful but less biologically motivated activation functions have come about in recent years[20]

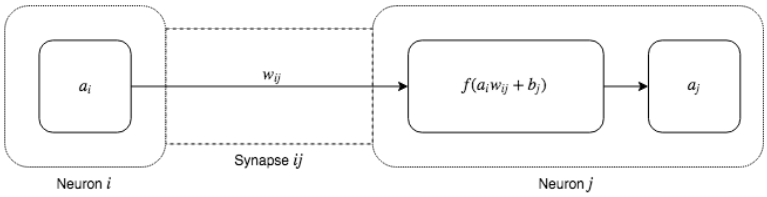

Fig. 3. Neural flow of information between firing-rate neurons. a_i is the activation of a presynaptic neuron, w_{ij} is the synaptic efficiency between them, and b_j is the bias; the neuron's natural firing frequency.

in the next section examine how important the conveying of spikes really is in the view of postsynaptic neurons.

3 Portraying rate-based neurons as conductance-outputting models

Reviewing the information flow between biological neurons described in the previous section, it does not seem that a post-synaptic neuron actually ever receives a discrete all-or-nothing spike from its upstream connections. Instead, the post-synaptic neuron receives a continuous conductance as a *result* of presynaptic spikes. The neuron is thus oblivious to any spiking events other than the resulting conductance changes in the synaptic cleft. In this case, the passing of discrete spike events often employed in spiking networks, appears no more biologically realistic than the firing-rates used by rate-based neurons when considering network wide simulations. In fact, we state that firing-rates viewed as conductance traces are a more biologically realistic view than either of the above. In the following sections, we present three different arguments to support this claim.

3.1 Argument 1: Mathematical equivalence

We here propose that rate-based neurons can alternatively be viewed as conductance-outputting models. To exemplify this, we investigate a mathematical model describing the total synaptic current going into a biological neuron j at any given dendritic location[26, p.174]. This current is the sum of synaptic currents from neurons i to j at the given location:

$$I_{syn}(t) = \sum_{i=0}^{N} g_i(t) w_{ij} (E - V_j(t)) \tag{3}$$

were $I_{syn}(t)$ is the total synaptic current, $g_i(t)$ is the conductance, E is the reversal potential of the input current, $V_j(t)$ is the post-synaptic membrane potential, and w_{ij} is the synaptic efficiency. If we allow ourselves a common simplification; that the incoming current is independent of the post-synaptic potential (PSP), we can simplify equation 3:

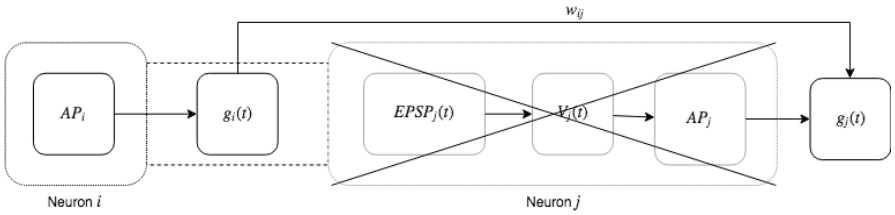

Fig. 4. Revised neural information flow demonstrating the direct mapping of conductances between neurons i and j.

$$I_{syn}(t) = \sum_{i=0}^{N} g_i(t) w_{ij} \tag{4}$$

We now have an equation similar to (2) representing the integration of inputs employed by common ANN models. Here, the output firing-rate of the neuron $a_i(t)$ has been replaced by a conductance $g_i(t)$, thus making $g_i(t)$ essentially a function of $a_i(t)$. This conductance model effectively represents an average of the firing-rate, as seen in Figure 5 which shows the relation between membrane potential and the output conductance. Similar to the bounded firing-rate due to the squashing function, $g(t)$ can be equally bounded between 0 and 1 with the values representing the concentration of glutamate within the synaptic cleft. Glutamate can thus be non-present (0) or at saturation (1).

In our biological model, the integrated input to neuron j further causes a rise in the EPSP and the somatic potential $V_i(t)$. $V_i(t)$ impacts whether the neuron will fire an AP. These APs in turn cause the release of glutamate into the synaptic cleft, and therefor increase the conductance at the synapse. If we were to simply bypass these intermediate steps through EPSP, $V_i(t)$ and AP, we can instead define a direct mapping function h from input current to output conductance where we obtain $g_j(t)$ (the output of neuron j) directly:

$$g_j(t) = h(I_{syn}(t)) \tag{5}$$

Figure 4 illustrates this point. $g_j(t)$ is hence obtained as a function of the total current $I_{syn}(t)$ going into neuron j, the same way $a_j(t)$ is a function of $I_{syn}(t)$ (where $I_{syn}(t) = \sum a_i w_{ij}$ in the rate-based case). As in the rate based case which often includes a bias b; $f(I_{syn}(t) + b_f)$, it is possible to include this natural and biologically relevant activation level to our conductance outputting model as well: $h(I_{syn}(t) + b_h)$.

In summary, the conductance outputting neuron model circumvents the internal voltage dynamics of the neuron as well as the firing of APs. Instead a direct mapping from input current to output conductance is performed $I_{syn}(t) \rightarrow g(t)$, and this can be viewed as similar to the mapping of the input current to the firing-rate of ANN neurons $I_{syn}(t) \rightarrow a(t)$.

Table 1. Simulation parameters

Parameter	Value	Unit	Description
R	10	KΩ	Membrane resistance
C	1	μF	Membrane capasitance
τ_g	10	ms	Time constant for conductance
$\delta(AP)$	20	mV	AP threshold
τ_{ref}	2	ms	Refractory period

3.2 Argument 2: artificial firing-rate neurons approximate biological conductance models in simulations

To demonstrate the above point; that the synaptic conductance is equivalent to the firing rate of the neuron, we run a simulation of a biological neuron model in which we apply an external input current. This input current, which can be viewed either as input from upstream neurons or an external electrode, modulates the resulting behaviour of our simulated neuron. Here, strong input currents cause the membrane potential to depolarize faster and hence output higher AP firing rates and thus influence the synaptic conductance to post-synaptic neurons.

The neuron model we use for this experiment is the leaky integrate-and-fire (LIF) model[5]:

$$C\frac{dV(t)}{dt} = \frac{RI_{syn}(t) - V(t)}{R} \tag{6}$$

Here, $V(t)$, R and C are the membranes voltage potential, resistance and capacitance respectively. I_{syn} is the input current. The membrane potential $V(t)$ is reset when surpassing the firing threshold $\delta(AP)$. The neuron goes into a short refractory period in which $V(t)$ is held at zero.

The conductance model we use is an exponential decay function borrowed from [26], which acts as a low-pass filter:

$$\tau_g\frac{dg(t)}{dt} = \delta(AP) - g(t) \tag{7}$$

Our simulation parameters are shown in table 1, with parameters also from [26].

Figure 5 shows the result of our simulation using a single LIF neuron and the conductance equations: (6) and (7) respectively. We have scaled $g(t)$ such that the its average lies between 0 and 1, as to output 1 during maximum firing frequency. This maximum frequency can be calculated using the neurons refractory period along with the duration of an AP, here set to 2ms and 1ms respectively[26]. For these values, the maximum firing frequency is calculated to be:[8]

[8] we have simplified here by disregarding depletion of neurotransmitters: i.e. we assume that neurotransmitters re-uptake is able to keep up with the pace of release.

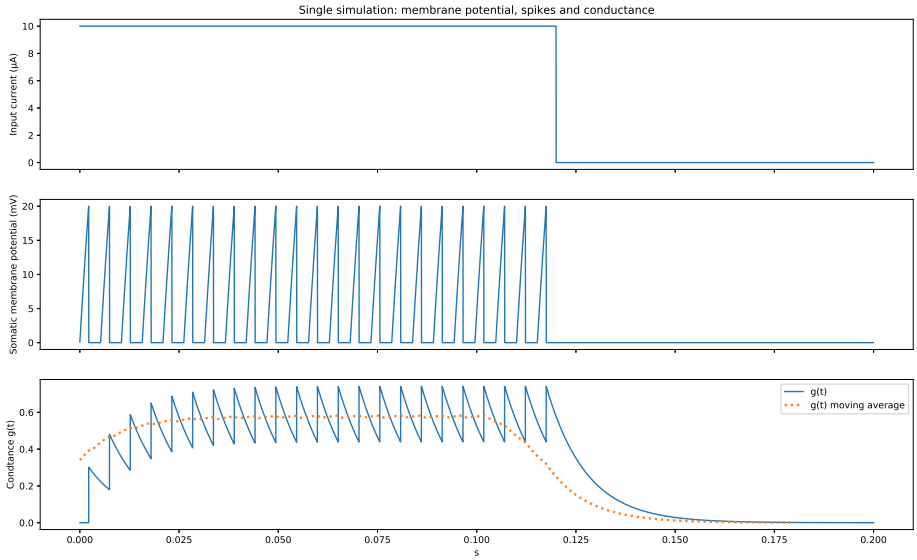

Fig. 5. *Top:* The input current of 10.6μA, cut off at 125 ms. *Middle:* The somatic membrane potential. The spikes are APs, here firing at 191 Hz. *Bottom:* The output conductance, showing a smoothed value of about 0.6, which is equal to: $\frac{\text{firing rate}}{\text{maximum rate}} = \frac{191}{333} \approx 0.6$.

$$\text{maxiumum rate} = \frac{1000\text{ms}}{2\text{ms} + 1\text{ms}} \approx 333\text{Hz} \tag{8}$$

We set a constant input current to the neuron of 10.6μA for 125ms before cut-off. We register that this yields a firing frequency of 191 Hz. As calculated in equation (8) the maximum firing frequency, given our parameters in table 1, is 333Hz. We find that $191/333.33 \approx 0.6$, which is the same value as our smoothed conductance converges to. These correspondences are true for all input current values, which we have verified below.

As can be observed in Figure 5, the conductance effectively represents the firing-rate average of the neuron, but more as a moving average due to the time constant τ_g rather than an instantaneous firing-rate. This is due to the conductance acting as a low-pass filter, which has advantages as the conductance provides a less erratic signal to the post-synaptic neuron. We can say that the function h in equation (5), yields an instantaneous average firing-rate similar to f in equation (2), while g is an low-pass filtered firing-rate. As such $g(t)$ approaches $a(t)$ for $\tau_g \to 0$.

We further plot the firing-rate against the stationary conductance for multiple input currents: 2-50μA. The resulting graph is shown in Figure 6. As expected, there is a linear dependence between the firing-rate $a(t)$ and the conductance $g(t)$. Furthermore, the scaled firing-rate is essentially equal to $g(t)$.

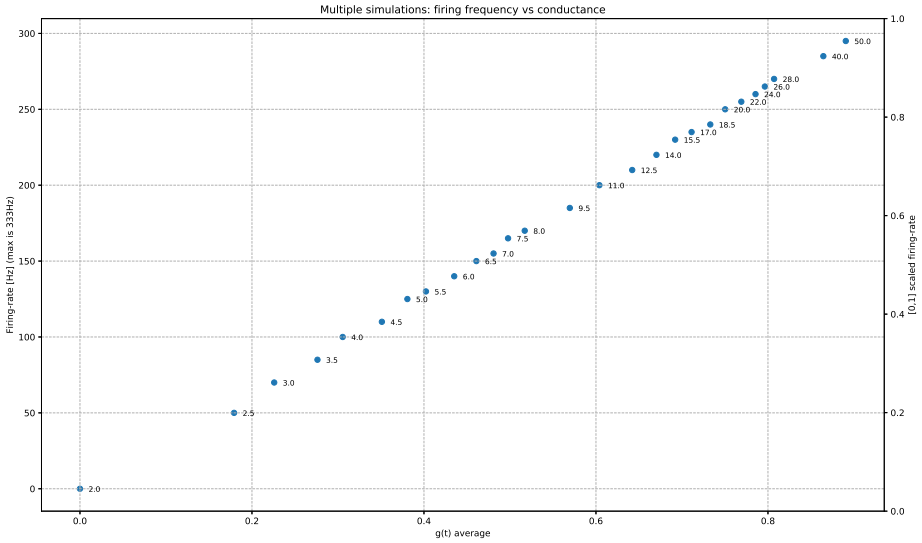

Fig. 6. The firing-rates calculated by $\frac{spikes}{200ms}$ vs the average $g(t)$ signal. 28 simulations where run each for 200ms real-time (20 000 timesteps at a simulation resolution of 0.01ms). Each simulation was run with a different input current with currents ranging from 2μA to 50μA (higher inputs yield higher firing-rates). The y-axis to the right displays the scaled firing-rate, showing that $g(t)$ is accurately encoding the firing-rate.

3.3 Argument 3: Synaptic plasticity models using discrete spikes don't work well

When introducing plasticity into SNN simulations, it has been common practice to employ some sort of phenomenological STDP model[25]. This model takes into account the precise timing of pre- and post-synaptic action potentials in order to update the synaptic weight between two neurons. The model is motivated by experimental findings[25] as well as its computational efficiency.

Further experimental evidence however, goes against these simple STDP models, as neuroscience has discovered more and more diverse STDP phenomena[23, 4] as shown in Figure 7. This indicates that the process of synaptic tuning is governed by other factors than mere spike-times. More biophysically inspired plasticity models rely on *continuous* conductances, calcium currents and internal cell voltages rather than discrete spikes[23, 6]. These models have demonstrated the ability to account for multiple STDP phenomena demonstrating the importance of continuous signals in the synapse. The dependency on continuous synaptic signals further argues against simplistic spike-based views.

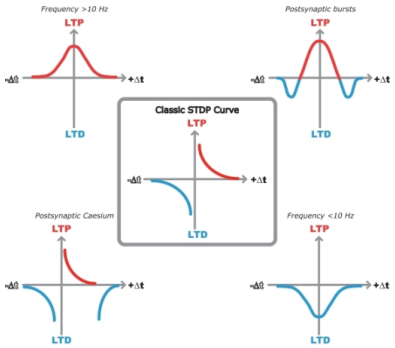

Fig. 7. The many STDP phenomena that have been experimentally observed can only be explained using continuous synaptic states. Here, we see that long-term potentiation shows varying dependence on spike-timing. The x-axis is the pre-post spiking interval, while the y-axis is the amount of positive/negative potentiation that occurs due to this timing. Courtesy of [4]

4 Discussion

4.1 Finding the right abstraction level

Biological neurons transmitting spikes to one another as a means of information transfer is a simplistic view of a highly complex transaction. Within chemical synapses in the brain, transmission is in the form of continuous and bounded concentrations of neurotransmitters. Hence, utilizing spiking neuron models does not necessarily make the overall network dynamics similar to biology if the information transfer between neurons is insufficiently modelled. We argue therefore that simplistic SNN networks utilizing discrete spikes at synapses are biologically inferior compared to rate-based, or as shown in this paper 'conductance-outputting', neural network models. Only when applying conductance-based synapses in spiking networks should we expect similarity to biological dynamics.

Neurons portrayed as point conductance-outputting models is naturally a large simplification too, compared to the vast internal dynamics and structures of biological neurons. However, in our quest towards understanding neural networks and replicating some of their amazing abilities, reducing complexity and computational cost of our models is essential. We must ask which details really matter. For example, one could argue that if (a) we had a complete architectural map of the brain down to the molecular level, and (b) we had the computational resources to simulate all molecular interactions, we could essentially simulate a complete working brain. Most researchers do not think (or at least hope) that this level of detail is necessary to understand the principles governing computation within neural networks. It may be that the complex emergent behaviour can be replicated by simple local interactions without modelling the complex dynamics within each and every neuron and synapse. We know from work on

Cellular Automata that complex behaviour in networks can come about from such simple local rules[27].

So how many levels of dynamics and interactions are we required to model in order to achieve computability on a par with biological networks? Is there an upper limit to complexity? These are fundamental questions we need to answer. For this we need to better understand complex dynamics in simpler networks and how these dynamics translate to more sophisticated models. Conductance-outputting neurons seem a good candidate in this context. In fact, several experiments have indeed demonstrated that multiple dynamical phenomena observed in biological networks can be simulated by networks using continuous firing-rate models[24].

4.2 Firing-rates vs conductance: why the perspective matters

In most sensory systems, the firing rate generally increases non-linearly with increasing stimulus intensity[12]. This discovery has led to the widely discussed assumption that neurons convey information through firing rates. Such a view has been highly influential in the resulting ANN models which employ firing-rate neurons. However, it is not obvious how downstream neurons would be able to observe upstream firing-rates at any instance of time. In order for such an analogy to work, there has to be some mechanism that dynamically encodes this firing-rate. Synaptic conductance resolves this problem by effectively representing the firing-rate as g.

The alternative view is important not only with respect to biological accuracy, but also because although firing-rate models are inspired by findings in neuroscience, today AI and neuroscience speak very different languages. This is largely due to the use of seemingly incompatible models. The 'artificial neuron' is often frowned upon by neuroscientists due to it's ambitious name and discrepancy with biological principles. We believe that the gap between the two fields can be bridged through an alternative view and terminology; viewing rate-based models as conductance-outputting, which revive these models in a more biologically plausible manner. An analogy such as this makes it easier to compare the benefits and limitations of models on both sides and creates a common platform for discussion. A different view can additionally stimulate new ideas and opportunities for more cross-disciplinary research. Such research is in fact becoming increasingly more common these days as many research institutions are connecting neuroscientists and AI researchers[1, 21]. Despite the impressive results of ANN research, as well as the fascinating findings in neuroscience, we are still far from understanding the computational abilities of biological neural networks. As researchers from both fields work on similar problems, a common language is both beneficial and highly necessary for collaboration and fruitful scientific progress.

5 Future work

This paper introduces the concept of conductance-based models: a model that is simpler than SNNs, yet more biologically intuitive than firing-rate neurons. Future work will involve the comparison of larger scale network dynamics of: (a) spiking networks with discrete synapses, (b) spiking networks with conductance modelled synapses, and (c) the continuous conductance-outputting model put forth here.

6 Conclusion

The common and widely applied firing-rate neuron model employed in ANN research has been examined and presented from a fresh point of view as a conductance-outputting model. The alternative view allows for better biological appreciation of these models, and also argues that they may be more biologically accurate than simple SNNs when employed in network wide simulations. This is especially prevalent when the synapse and plasticity models of SNNs base themselves on discrete events, which they often do. The takeaways from this is that one should not naively assume spiking neuron models to be biologically superior to firing-rate neurons. Information transfer between neurons is a large part of the equation and getting this part wrong can not be compensated by employing biologically sophisticated and detailed neural models. Furthermore finding good modelling abstraction levels is essential in order to better understand the computational abilities in neural networks. This is also important for creating a common language between researchers in neuroscience and AI.

References

1. Aaser, P., Knudsen, M., Ramstad, O.H., van de Wijdeven, R., Nichele, S., Sandvig, I., Tufte, G., Stefan Bauer, U., Halaas, Ø., Hendseth, S., Sandvig, A., Valderhaug, V.: Towards making a cyborg: A closed-loop reservoir-neuro system. In: Proceedings of the 14th European Conference on Artificial Life ECAL 2017. pp. 430–437. MIT Press, Cambridge, MA (2017). https://doi.org/10.7551/ecal_a_072
2. Attneave, F., B., M., Hebb, D.O.: The organization of behavior; a neuropsychological theory. The American Journal of Psychology **63**(4), 633 (2006). https://doi.org/10.2307/1418888
3. Brette, R.: Philosophy of the spike: Rate-based vs. spike-based theories of the brain. Frontiers in systems neuroscience **9**, 151 (2015). https://doi.org/10.3389/fnsys.2015.00151
4. Buchanan, K.A., Mellor, J.: The activity requirements for spike timing-dependent plasticity in the hippocampus. Frontiers in Synaptic Neuroscience **2**, 11 (2010). https://doi.org/10.3389/fnsyn.2010.00011
5. Burkitt, A.N.: A review of the integrate-and-fire neuron model: I. homogeneous synaptic input. Biological Cybernetics **95**(1), 1–19 (2006). https://doi.org/10.1007/s00422-006-0068-6

6. Clopath, C., Gerstner, W.: Voltage and spike timing interact in stdp - a unified model. Frontiers in Synaptic Neuroscience **2**(JUL), 25 (2010). https://doi.org/10.3389/fnsyn.2010.00025
7. Diehl, P.U., Cook, M.: Unsupervised learning of digit recognition using spike-timing-dependent plasticity. Frontiers in Computational Neuroscience **9**, 99 (2015). https://doi.org/10.3389/fncom.2015.00099
8. Gerstner, W., Kreiter, A.K., Markram, H., Herz, A.V.: Neural codes: firing rates and beyond. Proceedings of the National Academy of Sciences of the United States of America **94**(24), 12740–1 (1997). https://doi.org/10.1073/PNAS.94.24.12740
9. Honoré, T., Lauridsen, J., Krogsgaard-Larsen, P.: The binding of [3h]ampa, a structural analogue of glutamic acid, to rat brain membranes. Journal of Neurochemistry (1982). https://doi.org/10.1111/j.1471-4159.1982.tb10868.x
10. Izhikevich, E.M.: Which model to use for cortical spiking neurons? IEEE Transactions on Neural Networks **15**(5), 1063–1070 (2004). https://doi.org/10.1109/TNN.2004.832719
11. de Kamps, M., van der Velde, F.: From artificial neural networks to spiking neuron populations and back again. Neural Networks **14**(6-7), 941–953 (2001). https://doi.org/10.1016/S0893-6080(01)00068-5
12. Kandel, E.R., Schwartz, J.H., Jessell, T.M.: Principles of Neural Science, vol. 4. McGraw-Hill Education (2013). https://doi.org/10.1036/0838577016
13. Kheradpisheh, S.R., Ganjtabesh, M., Masquelier, T.: Bio-inspired unsupervised learning of visual features leads to robust invariant object recognition. Neurocomputing **205**, 382–392 (2016). https://doi.org/10.1016/j.neucom.2016.04.029
14. Kheradpisheh, S.R., Ghodrati, M., Ganjtabesh, M., Masquelier, T.: Deep networks can resemble human feed-forward vision in invariant object recognition. Scientific Reports **6** (2016). https://doi.org/10.1038/srep32672
15. Maass, W.: Networks of spiking neurons: The third generation of neural network models. Neural Networks **10**(9), 1659–1671 (1997). https://doi.org/10.1016/S0893-6080(97)00011-7
16. Mainen, Z.F., Seinowski, T.J.: Reliability of spike timing in neocortical neurons. Science (1995). https://doi.org/10.1126/science.7770778
17. Markram, H., Gerstner, W., Sjøstrøm, P.J.: Spike-timing-dependent plasticity : a comprehensive overview. Frontiers research topics **4**(July), 2010–2012 (2012). https://doi.org/10.3389/fnsyn.2012.00002
18. Medium: Google brain's co-inventor tells why he's building chinese neural networks: Andrew ng on the state of deep learning at baidu. Medium (2015)
19. Meldrum, B.S.: Glutamate as a neurotransmitter in the brain: review of physiology and pathology. The Journal of nutrition (2000). https://doi.org/10736372
20. Nair, V., Hinton, G.E.: Rectified linear units improve restricted boltzmann machines. Proceedings of the 27th International Conference on Machine Learning (2010). https://doi.org/10.1.1.165.6419
21. Numenta: https://numenta.com/
22. Rumelhart, D.E., Widrow, B., Lehr, M.A.: The basic ideas in neural networks. Communications of the ACM (1994). https://doi.org/10.1145/175247.175256
23. Shouval, H.Z., Wang, S.S.H., Wittenberg, G.M.: Spike timing dependent plasticity: a consequence of more fundamental learning rules. Frontiers in computational neuroscience **4**(July), 1–13 (2010). https://doi.org/10.3389/fncom.2010.00019
24. Sompolinsky, H.: Computational neuroscience: beyond the local circuit. Current Opinion in Neurobiology **25**, xiii–xviii (2014). https://doi.org/10.1016/J.CONB.2014.02.002

25. Song, S., Miller, K.D., Abbott, L.F.: Competitive hebbian learning through spike-timing-dependent synaptic plasticity. Nature neuroscience **3**(9), 919–926 (2000). https://doi.org/10.1038/78829
26. Sterratt, D., Graham, B., Gillies, A., Willshaw, D.: Principles of Computational Modelling in Neuroscience. Cambridge University Press (2011). https://doi.org/10.1109/MPUL.2012.2196841
27. Wolfram, S.: Cellular automata as models of complexity. Nature **311**(5985), 419–424 (1984). https://doi.org/10.1038/311419a0
28. Wurtz, R.H.: Visual receptive fields of striate cortex neurons in awake monkeys. Journal of neurophysiology (1969). https://doi.org/10.1152/jn.1969.32.5.727