Scaling a slow-wave sleep cortical network model using NEOSIM ☆

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Abstract

We describe a case study transforming a simulation model coded in sequential C++ to run in parallel under Neosim, to enable much larger compartmental network models to be run. For some network models cut down scale is sufficient; however, there are cases where network behaviour cannot be reproduced on a smaller model (e.g. Neurocomputing 32–33 (2000) 1041). The example we present is a model of slow-wave sleep oscillations. In an earlier paper (Neurocomputing 38 (2001) 1657) we outlined the design of the Neosim framework for scaling models, focussing on networks of compartmental neuron models built using existing simulation tools Neuron and Genesis. Here, we explain how a Hodgkin–Huxley network model coded in C++ for a cortical network was adapted for Neosim, and describe the experiments planned. This case study should be of interest to others considering how best to scale up existing models and interface their own coded models with other simulators. © 2002 Elsevier Science B.V. All rights reserved.

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1. The model

Accurate simulation of a realistic size neural network based on Hodgkin–Huxley-type models of individual neurons would require enormous computational resources far exceeding the power of currently available single CPU computers. In some cases,

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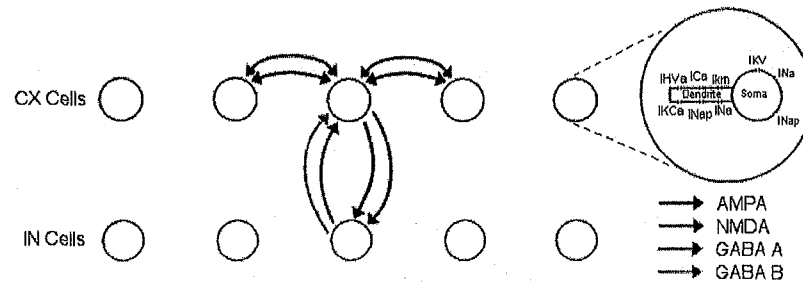
Fig. 1. The compartmental network model of isolated cortex.

this problem can be successfully resolved by scaling down the size of simulated network. One well-known example of this approach is a model of thalamocortical spindle oscillations—7–14 Hz activity usually observed during early sleep. These oscillations are generated as a result of interaction between thalamic relay and reticular cells and the simplest model which is able to simulate this activity can include only a few neurons [4].

However, there are problems which cannot be scaled down easily. One example is a model of slow-wave sleep (SWS) oscillations—rhythmic (<1 Hz) activity observed during natural sleep or under some types of anaesthesia [10]. During SWS the whole thalamocortical network is switched periodically between up (active) and down (silent) states and the mechanisms controlling these transitions were unknown until recently. Based on the studies of an isolated cortical slab, it has been hypothesized that random summation of the miniature excitatory postsynaptic potentials (minis) in a single cortical cell (or a few cells) during silent phases of SWS can initiate network activity at each cycle of SWS oscillations [11]. As these events occur independently in different cells, the probability of a whole network reactivation should grow as the size of the network increases thus leading to faster oscillations. Using analytical studies it was found that in a large enough cortical network (>100,000,000 neurons) the probability of burst initiation can increase sufficiently to provide oscillations in the frequency range of SWS activity [11].

A computational Hodgkin–Huxley-type model of the cortical network was developed in sequential C++ to test these predictions. This model included layers of cortical excitatory cells and inhibitory interneurons interconnected with GABAa, AMPA and NMDA synapses (Fig. 1). As network size was critical factor, an artificial increase of minis amplitude was a necessary approach to study SWS activity in a small network including a few hundred cells [1]. Systematic study of the influence of the network size on the properties of spontaneously generated SWS-like oscillations requires simulations of much larger networks—the goal which possibly can be reached using parallel computer simulations.

2. Neosim

Neosim is a simulation framework for building large-scale and detailed models of networks of spiking neurons. It is based on parallel discrete event simulation techniques,
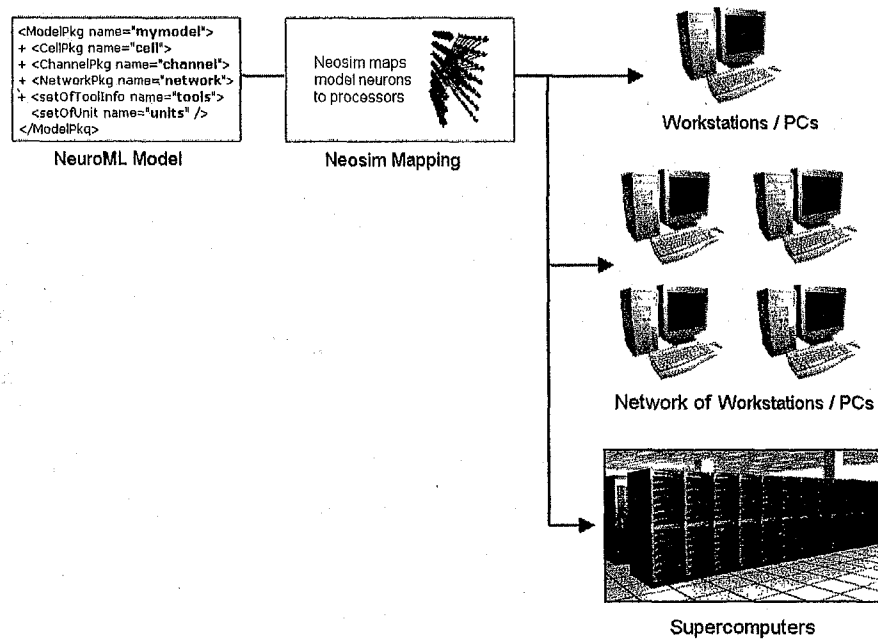
```
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+ <NetworkPkg name="network">
+ <setOfToolInfo name="tools">
   <setOfUnit name="units" />
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```

NeuroML Model

Neosim maps
model neurons
to processors

Neosim Mapping

Workstations / PCs

Network of Workstations / PCs

Supercomputers

Fig. 2. Neosim maps the NeuroML network model onto PCs, shared memory multiprocessors and super-computers.

which allow a simulation to be spread efficiently across a parallel machine, without the modeller having to be concerned with writing parallel code. An earlier paper [6] described its design in more detail, including its use for building network models of compartmental neurons constructed with the NEURON [8] and GENESIS [2] simulation tools. Neuron models constructed using the Catacomb simulator [3] can now also be included.

A Neosim model is composed from "entities", such as individual neuron models, which communicate using events, typically action potentials. Networks are specified at a high level in terms of populations of entities, and projections between these populations, where commonly used styles (e.g. 2D layers of cells, nearest neighbour connections) are provided as standard, and facilities are provided for defining model-specific populations and projections. Once a model has been recast in terms of Neosim entities and networks, it can be scaled automatically to run on a parallel or distributed machine, allowing much larger models to be run than will fit on a single CPU (Fig. 2).

3. Converting the model for Neosim

Fig. 3 In the sequential model of the cortical network, neurons were modelled using a C++ class which calculates the membrane potential and channel and synapse states
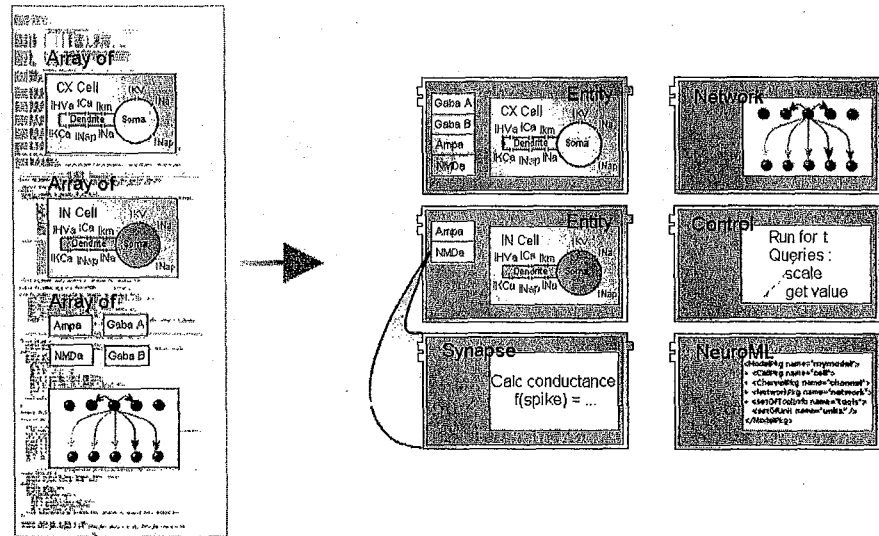
Fig. 3. The original C++ model was split into "Modules" for Neosim.

at each timestep. To convert this into Neosim entities, a wrapper class was written which directs incoming and outgoing spikes to/from this class.

```
class CX_Neosim : public NeosimEntity {
        CX cx;    // Existing C++ model of a neuron
        void handleSpike( double t, Input i ) {
            // Despatch spikes to synapse object
        }
        void advance( double t ) {
            // Advance internal state to t
        }
};
```

The major stylistic change at the cell level was how synapses are modelled. The original code dealt with synapses by using a direct pointer connection between source and target neurons. With Neosim this had to be split into two: a threshold object at the source neuron which generates spike events when the soma voltage exceeds the threshold; and the synapse object had to convert an event into a continuous conductance change at the target.

To specify the network connectivity of the cortical model with Neosim, we used an early version of NeuroML for simulation models [7]. This is a declarative model description language which is simulator independent; the aim is that different simulators will be able to exchange models written in this common language. The extract below specifies a line of 500 model cortical cells (CXCell) with the localized projection from

inhibitory neurons back to these cells.

```
<model>
    <population name="CXgrid" entityname="CXCell" type="Grid3D"
                xdim="500" ydim="1" zdim="1" />
    <projection name="INtoCX" srcPop="INgrid" dstPop="CXgrid"
                radius="2" autoconnect="false" boundmethod="mirror">
        <connection srcPort="out" dstPort="in"
                delay="2" xrange="2" yrange="1" zrange="1" />
    </projection>
</model>
```

4. Conclusion

We have transformed an existing simulation model coded in C++ to run in parallel under the Neosim kernel. On shared memory multiprocessors there is a linear speedup for this model. On the Terascale supercomputer at Pittsburgh Supercomputer Center there was near linear speedup up to 8 quad nodes (32 Alpha processors). The final Terascale computer will allow scaling to 3000 processors. This will allow testing of hypotheses on the behaviour of the cortical network for much larger numbers of neurons than is possible on a single CPU. Unlike earlier parallel modelling systems (PGENESIS [5,9]), the NEOSIM model specification (in NeuroML) is unchanged regardless of whether running on 1 or 1000 processors. Now that the model has been split into Neosim entities, it becomes straightforward to combine it with models written using other simulation tools with a Neosim interface, for example linking to a network model of thalamus built using the Neuron or Catacomb simulation environments.

References

[1] M. Bazhenov, I. Timofeev, M. Steriade, T.J. Sejnowski, Model of slow-wave sleep and its transition to activated states in thalamocortical network, 2000, SFN abstract.

[2] J.M. Bower, D. Beeman (Eds.), The Book of GENESIS: Exploring Realistic Neural Models with the General Neural Simulation System, 2nd Edition, Springer, Berlin, New York, 1998.

[3] R. Cannon, Catacomb, http://www.compneuro.org/catacomb.

[4] A. Destexhe, T. Bal, D.A. McCormick, T.J. Sejnowski, Ionic mechanisms underlying synchronized oscillations and propagating waves in a model of ferret thalamic slices, J. Neurophysiol. 76 (3) (1996) 2049–2070.

[5] N. Goddard, G. Hood, Parallel genesis for large scale modeling, in: J. Bower (Ed.), Computational Neuroscience: Trends in Research, Plenum Publishing, New York, 1997, pp. 911–917.

[6] N. Goddard, G. Hood, F. Howell, M. Hines, E. De Schutter, NEOSIM: portable large-scale plug and play modelling, Neurocomputing 38 (2001) 1657–1661.

[7] N. Goddard, et al., Towards NeuroML: model description methods for collaborative modeling in neuroscience, Philos. Trans. Roy. Soc. Ser. B 356 (1412) (2001).

[8] M.L. Hines, N.T. Carnevale, The NEURON simulation environment, Neural Comput. 9 (1997) 1179–1209.

[9] F. Howell, J. Dyhrfjeld-Johnsen, R. Maex, E. De Schutter, N. Goddard, A large scale model of the cerebellar cortex using PGENESIS, Neurocomputing 32–33 (2000) 1041–1046.

[10] M. Steriade, A. Nunez, F. Amzica, A novel slow (<1 Hz) oscillation of neocortical neurons in vivo: depolarizing and hyperpolarizing components J. Neurosci. 13 (8) (1993) 3252–3265.

[11] I. Timofeev, F. Grenier, M. Bazhenov, T.J. Sejnowski, M. Steriade, Origin of slow cortical oscillations in deafferented cortical slabs, Cereb. Cortex. 10 (12) (2000) 1185–1199.