

Simulation Structural Plasticity II



Project Manager
Fabian Czappa

Researchers
Julian Schmidt

Principal Investigator
Prof. Dr. Felix Wolf

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Clusters
Lichtenberg Cluster Darmstadt

Institute
Department of Computer Science

University
Technische Universität Darmstadt

Introduction

The human brain is an ever-developing complex organ – it does so even during adult life, e.g., as a reaction to learning, a disease, or other general stimulations. Understanding the driving forces to the development and reorganization is key in predicting outcomes (for example, brain surgery). Many current efforts in neuroscience disregard the changes in the connectome because of a plethora of reasons. Firstly, including dynamics of this kind complicates the models – some of which do not benefit from additional complexity. Secondly, models for changes in the connectome are computationally expensive in and of themselves. Lastly, because of the nature of the subject, finding in vivo evidence for changes is difficult, too, as it requires long-time studies of small details – in contrast to, e.g., long-term studies on memory capabilities of diseases, which have an effect that is easier to measure.

Methods

In our project, we use the Model of Structural Plasticity, which describes a way the brain can reorganize on the basis of activity on the neuron level: Each neuron has its specific target activity, and the attempt of all neurons to reach their target levels the overall brain. Additionally, we use an approximation of this model: In its original form, the Model of Structural Plasticity contains the calculation of pairwise interactions – which is infeasible for a large number of neurons. Thus, we use an approximation technique original from astrophysics to speed up the calculation while keeping the approximation error relatively small. We develop an in-house simulation code (“Relearn”, available on Github), which brings us to the forefront of simulations regarding structural plasticity. No other simulation

can handle structural plasticity on the scale of our simulation (“Nest” has a module for structural plasticity, yet they are constrained by the size of the simulation).

Results

During this project period, we focused on cleaning the code. This includes thorough testing and benchmarking to identify bottlenecks during execution and errors in the business logic. While we found no significant errors (we found one in the logging mechanism that reports general information but no processed output and one in the handling of data input, which would have arisen in other use cases than ours), we were able to identify two distinct bottlenecks and starting working on replacements. These results, while not in a publishable state yet, have improved the quality of our simulation and enabled us to perform different studies.

Discussion

In the future, we will study the scalability of the replacement modules for the bottlenecks and aim for a full-scale simulation (100 billion neurons). This will enable us and the community to investigate effects on the scale of the whole brain while also allowing fast simulations on a smaller scale. After all, there is undeniable evidence that structural plasticity occurs in the human brain during all stages of life, and bringing the community into position to utilize these effects in the simulation is a corner stone of our mission in HKHLR.

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