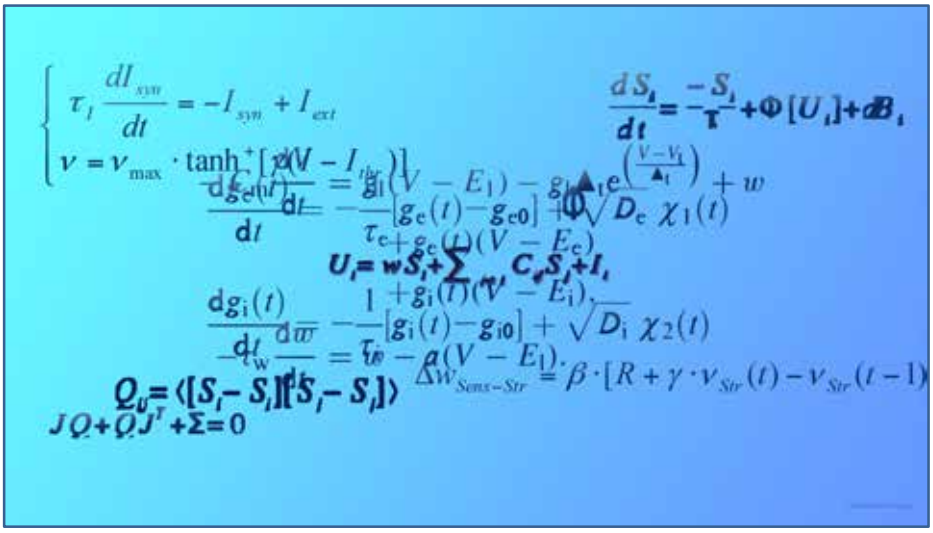

This is an assemblage of models published by partners from SP4 during the RUP.

Image by K. Fregnac

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Abstract:	SP4 Theoretical Neuroscience overview of work achieved during period of SGA1 M1-M12
Keywords:	SP4 Theoretical Neuroscience

Table of Contents

1. SP Leader's Overview

1.1 Key Personnel

SubProject Leader: Alain DESTEXHE (CNRS, P10)

SubProject Deputy Leader: Idan SEGEV (HUJI, P60), Viktor JIRSA (AMU, P78)

SubProject Manager: Katherine FREGNAC (CNRS, P10)

1.2 Progress

As detailed in the annex, the different models developed in SP4 progressed in the first year of SGA1. Perhaps the largest change is that there is now a much more focused integration of the different models developed in SP4, with the rest of the HBP. Many models directly contribute to the Platforms, and there is now a larger use of the experimental data collected in the HBP. More specifically, WP4.1 has strong connections with SP6, but also SP9 and the experimental SPs (SP1,2,3). WP4.2 and WP4.3 have strong connections with SP9, but also with SP6 and SP7. The models developed in WP4.4 have connections with SP5, SP6, SP9 and SP10, and WP4.5 naturally has a strong connection with SP5, as well as new connections with SP8 for modelling brain pathologies, a theme that we expect to take more and more importance in the future. A detailed comparison - and possibly a unification - of different mean-field models developed in SP4 would be necessary.

It must also be noted that nearly all models developed in SP4 also have a strong connection with SP5, and in particular in the Neural Activity Resource, where model data will be posted in the future.

There are also stronger connections within SP4, in particular the development of mean-field models is now better connected to the building of spiking networks in other tasks, and here again, we expect that mean-field models will take an increasing importance, and form the basis of simulations of normal and pathological brain activity.

In the present deliverable D4.7.1, we include a detailed report of all models developed in SP4 during the first year of SGA1. We first list the different properties and Components related to each model. In a second part (Annex), we provide the details for each model, with figures.

1.3 Deviations

The main deviation is that some of the tasks are slightly behind schedule because of the delayed funding of SGA1. However, the situation was worse at M6 (see 6-month report) and was partially compensated by an increased work in the second six-month period, so we expect to catch up with the planned activities in SGA1.

There was no consequences for the other SPs.

1.4 Impact of work done to date

The impact is mostly through publications, SP4 has one of the highest international impact at this level.

1.5 Priorities for the remainder of the phase

The priority remains to catch up with the schedule and be able to produce all the work planned for SGA1. There are areas that are already ahead of schedule, so we are confident that this will be done as planned. Another priority is to participate to the Platforms, and here, the high SP4 participation to the CDPs should naturally satisfy this priority. Note that the newly planned Neural Activity Database in SP5 will increase the impact of SP4 to Platforms, since virtually all SP4 models will contribute.

2. WP 4.1 Bridging Scales

2.1 Key Personnel

Work Package Leader: Alain DESTEXHE (CNRS, P10)

2.2 WP Leader's Overview

The work in WP4.1 has been very efficient and productive in the first year of SGA1, despite the problem of funding in the first few months. For this reason, we are a little bit behind schedule, but a lot of work has been produced in the last six months. A large number of publications were produced and the work is contributing to the Platforms.

Major achievements:

- Development of simplified models of neurons with dendrites (in progress), intended to be used in SP6
- Development of simplified models of dendritic integration and correlation processing by active dendrites (work in progress), aimed at being implemented in neuromorphic systems (SP9)
- Development of compact models for the input-output transfer function of dendritic neuron models for use in SP6
- Development of different types of mean-field models to describe population dynamics in cerebral cortex (work in progress), these models can be used in SP4 and SP6, as well as to interpret experimental data on imaging (SP1, SP2, SP3)
- Development of simplified models of brain signals (work in progress), aimed at being used in more complex models in SP4 and SP6

2.3 Priorities for the remainder of the phase

WP4.1 should continue the planned work and catch up with the planned activities in SGA1.

3. WP 4.2 Generic Models of Brain Circuits

3.1 Key Personnel

Work Package Leader: Markus DIESMANN (JUELICH, P20)

Other researchers: Marja-Leena LINNE (TUT, P103), Viktor JIRSA (AMU, P78), Sacha VAN ALBADA (Juelich, P20)

3.2 WP Leader's Overview

The exchange of ideas and techniques at the EITN has been fruitful throughout the funding period. I perceive an increase in openness and a standardisation of tools that will bolster future collaborations and the success of the Project.

On the down side, the administrative overhead consumed considerable resources, which detracted from the time available to do science.

The curation and dissemination of the cortical microcircuit model has already led to three publications from other groups (Schwalger et al. ArXiv 2016; Lee et al., ArXiv 2016; Cain et al., PLoS CB 2016). The multi-area model approaches the boundaries of what can currently be simulated with the available software and hardware, and thereby gives guidance to the simulation technology development.

Major achievements:

- Development of a first version of a multi-area model of macaque visual cortex with stable ground-state activity obtained with the help of a novel semi-analytical mean-field method (Schuecker et al., 2017). The model constitutes a supercomputing simulation test case for SP7.
- Ported a cortical microcircuit model from NEST to the SpiNNaker neuromorphic hardware system (SP9). This achievement represents the first full-density cortical microcircuit simulation on neuromorphic hardware.
- Development of a first version of a neuron-glia network model, (work in progress; the developed models and methods can be used both in SP4 and SP6).
- Simplification of HH-type neuron reduced to a set of 4 variables using mass/charge constraints and approximations of gating dynamics and demonstrations of its capacity to reproduce a K+-elevation-induced bursting (to be used in mean field models of SP4).

3.3 Priorities for the remainder of the phase

One of the priorities for the remainder of SGA1 is to further investigate local field potentials in the 4x4 mm^2 model of cortex based on the hybrid scheme for predicting LFP signals from spiking point-neuron networks developed in T4.1.4 (Einevoll). For the multi-area model of macaque visual cortex, a priority is to provide an internal release of the model specification. This will be a testbed for the mean-field theory that will continue to be developed in T4.1.3. A further priority is to submit a manuscript on the dynamics of the model. Further work concerns the completion of the study in which the full-scale cortical microcircuit model is made available for research on the neuromorphic hardware. For the neuro-glial model for bursting activity, the priority for the upcoming phase is to develop a mesoscopic population-level description able to provide further insight into the conditions underlying the emergence of epileptic activity and its spread to neighbouring areas. The main concern for the study of astrocyte-neuron interactions is to bring the model into closer agreement with experimental data and release the resulting validated model.

4. WP 4.3 Learning and Memory

4.1 Key Personnel

Work Package Leader: Wulfram GERSTNER (EPFL, P1)

Together with the groups of Walter SENN (Berne), Andre GRUENING (Surrey), and Misha TSODYKS (Weizmann)

4.2 WP Leader's Overview

Everything is working nicely.

Our impact is achieved via publications in good international journals.

Major achievements:

- obtainment of learning rules for neuron models with spatial structure. Important for link to brain simulation (SP6).
- obtainment of abstract learning principles for supervised learning in neurons with dendrites. Important for the link to neuromorphic computing (SP9).

- obtainment of new learning rule for learning under surprise as a modulator. Important for link to neuromorphic (SP9).
- established pipeline for transfer of plasticity algorithms to standard simulators, such as NEST, very important for SP6 and SP7

4.3 Priorities for the remainder of the phase

The groups of Walter SENN (Berne), Andre Gruening (Surrey), and Misha Tsodyks (Weizmann) and Wulfram Gerstner (EPFL) will put high priority on pushing the recent scientific results achieved during the first year of SGA1 into publications in great international journals. The interaction with other partners in SP4, with CDP5, and, most importantly, with other SubProjects will also remain a high priority. Finally, the smooth transition from SGA1 to SGA2 needs not only administrative but also scientific preparation so that continuity of the research work is also a high priority.

5. WP 4.4 Models of Cognitive Processes

5.1 Key Personnel

Work Package Leader: Gustavo DECO (UPF, P77)

5.2 WP Leader's Overview

From a scientific point of view, the progress performed by all the Work Package partners is very satisfactory. The research plan is advancing in the directions desired and at the forefront of their respective topics. This is already a very important achievement considering the very difficult conditions in which the work had to be performed in the last 12 months, e.g., the delayed SGA1 grant signature, the sudden introduction of novel organisation procedures requesting every task to re-define their goals in terms of Components, and the need to prepare the SGA2 proposal in the same period.

On the other hand, important efforts have been performed in the Work Package to build both internal (within SP4) and external (other SPs) collaborations. A particular problem we have identified is that, while all partners are aware of the need to make their models available for the Platforms, the mechanisms by which this will be carried out is not always clear, neither it is clear who will perform the integration of the models into the Platforms. This problem is aggravated by the fact that the work plan and work-flows for the Platforms in SGA1 were planned independently and thus, integrating our work into Platforms often involves an intromission to the working plan and objectives of the Platforms. During the planning of SGA2 proposal, this has been significantly improved.

Major achievements:

- Development of a novel model of whole-brain resting-state brain activity capturing nonlinear brain region dynamics based on the normal form of the Hopf bifurcation. To be embedded into the neuroinformatics Platform (SP5) for automatised data/modelling workflows.
- Investigation of a network model of regular-spiking and fast-spiking neurons, compatible with experimental data, as well as with neuromorphic hardware in SP9.
- Obtainment of a nonlinear model of the retina which can accurately predict the responses of ganglion cells to multiple moving objects in the same scene. The model is of relevance to provide the neurorobotics Platform (SP10) with biologically plausible neural networks able to process visual stimuli.
- Development of computational model of basal ganglia and the study of dopamine depletion-induced increase of AMPA efficacy in cortico-striatal synapses to medium spiny neurons (MSNs). The model will serve as the starting point for a detailed model of the basal ganglia planned in SP6.

- Extension of spatial navigation models to consider episodic memory and (general) non-spatial planning, for application on autonomous robotic devices developed in SP10.
- Development of a deep convolutional auto-encoder network able to automatically learn a mapping from natural images to topological saliency distributions. The model is the starting point for the development of CDP4.

5.3 Priorities for the remainder of the phase

A priority for the remaining 12 months of SGA1 is to make sure that the scientific goals are achieved and meet the requirements to be continued during SGA2. Another priority is the consolidation of the connections that have been built. We need to make sure that in the following 12 months all WP4.4 partners have identified and contacted adequate partners in the Platforms and that their future working plans for SGA2 are being developed in coordination with those partners.

6. WP 4.5 Linking Model Activity and Function to Experimental data

6.1 Key Personnel

Work Package Leader: Sonja GRÜN (JUELICH, P20), Viktor JIRSA (AMU, P78)

Task leader T4.5.1: Sonja GRÜN (JUELICH, P20)

Postdoc in T4.5.1: Michael VON PAPEN (JUELICH, P20) (from 6. 3. 2017). Contributions also from Dr Nicole VOGES (JUELICH, P20)

Task leader T4.5.2: Viktor JIRSA (AMU, P78)

Postdocs in T4.5.1 / T4.5.2: Dr. Spase PETKOSKI and Dr. Andreas SPIEGLER (from 1.4.2016). Contributions also from PhD student Francesca MELOZZI (AMU, P78).

6.2 WP Leader's Overview

What went particularly well?

- Resting state 100-electrode array electrophysiological recordings from non-human primate provided by external experimental partners.
- Performed statistical characterisation of resting state data: Single unit activities separated into excitatory and inhibitory neurons, estimation of firing statistics of these classes and characterization of network interactions.
- The integration of structural data (connectivity, region mapping) of different origins (DTI, Allen Brain Atlas) in whole mouse brain network models and the initial modelling of spontaneous resting state.
- Whole brain network models derived from human connectome data of epileptic patients using Epileptor, a neural mass model capturing the temporal evolution of a seizure including on- and offset, were validated against empirical data.

What didn't go according to plan?

- Validation of the reduced top-down models against high- dimensional neuronal network models, enabling parameter space explorations to guide high performance computations (SP7). For this we rely on the input from our partners (upstream Components) working on mean-field models and this will be looked after in the next phase.
- Hiring of postdoc for T4.5.1 was only possible in March 2017.

- Impact of work done
- Electrophysiological resting state data and its characterization provide base line data for the comparison with balanced random network simulations.
- The integration of the Allen Brain Atlas for the mouse brain network models, and the results from the initial modelling of the resting state is an important step that will allow further validation of the empirical data (calcium imaging) for behavioural functions and pathologies. Here we have provided proof of concept for the predictive value of individual connectome (DTI data), as well as superiority of the tracer data compared to DTI.
- The validation of the human connectome for *modelling* temporal dynamics of the epileptic patients will have a big impact on further work into individualised predictive *modelling* for epileptic patients, and should be of great importance to clinicians.

Major achievements:

- preparation of a workflow to integrate model data into the Neural Activity Resource in SP5 (collective work in SP4)
- implementation of a data analysis workflow (available as collab.humanbrainproject.eu/#/collab/2493) as one branch of the integrative loop implementation for comparison and validation of the layered cortex model (from SP4 and CDP4) on experimental data (to be delivered by NAR of SP5)
- Construction and simulation of mean field network model derived from Allen Mouse Connectome and computational demonstration of biologically realistic resting state functional connectivity validated against empirical fMRI mouse data, to be used in SP1 and verify results from SP6 (CDP1)
- Reconstruction of epileptic patients' brain networks (N=15) and demonstration of simulations of biologically realistic seizure propagation patterns validated against empirical patient SEEG data (to be used in SP8)

6.3 Priorities for the remainder of the phase

T4.5.1 will focus on the Use Case of comparison of the NEST 4x4mm² microcircuit model with resting state data from premotor cortex of non-human primate in collaboration with T4.2.1. Therefore, distance dependent neuronal weights will be included into the 4x4mm² microcircuit model. The analysis workflow of the resting state data and its comparison to the modelled data will be integrated into the HBP collaboratory as a collab and made available to HBP members. The further analysis we will include to extract higher order correlations from both simulation and experiment and work towards the formulation of strategies for the model parametrization to obtain compatible correlation structures as obtained in the experimental data. This will include a parameter study of the model and successive validation with model.

Further validation of the whole brain mouse network models against empirical data recorded in SP1 using calcium imaging before and after stroke will be in the next step. This should help constraining the structure-function relationship and will form the basis for later extensions towards behavioural functions and pathologies (SP8). Further work is also expected in comparison of different modalities of structural data (tracer vs. DTI) and their predictive value for the functional connectivity.

Regarding human data, further exploration of the predictive value of the connectome is expected, where again the validation will be against the empirical data from epileptic patients. Here we will compare the effects that different lesioning strategies have on the propagation of the instabilities in the dynamics.

7. WP 4.6 EITN

Please refer to Deliverable D4.6.1 for SGA1 M1-M12 activity.

Major achievements:

- organisation of very successful workshops at the EITN, with a strong participation from the community outside HBP.

8. WP 4.7 Scientific coordination

8.1 Key Personnel

Work Package Leader: Alain DESTEXHE (CNRS, P10)

8.2 WP Leader's Overview

The coordination of SP4 has been working very well, with regular meetings (videoconferences and physical meetings), in which the WP leaders are all actively participating. All partners contribute to the modelling work in a very collegial way that works well for SP4.

Alain Destexhe participated to the DPIT team, who proposed a restructuration of SP5, as required by the E.C. officers. The main contribution of SP4 was to suggest and support the creation of the "Neural Activity Resource" (NAR) in SP5, which will naturally have strong links with SP4, as the models developed emphasise the modelling of the activity of the brain at multiple levels.

8.3 Priorities for the remainder of the phase

Priorities are to eEnsure SP4 has good communication between all partners in this SubProject as well as that the SP reaches its SGA1 goals as best as possible. Continue on disseminating the work being done, within the consortium as well as outside of the HBP.

8.4 Milestones

Table 1: Milestones for WP No. & Name

MS No.	Milestone Name	Lead Partner	Task(s) involved	Expected Month	Achieved Month	Comments
MS4.5.1	Overview of available brain activity data and model pairings, including the type of data, and identified first use cases for the comparison of activity data and model simulations.	20	T4.5.1	M12	M12	Milestone has been achieved (see Deliverable 4.7.1 for details): For Project 1, a first Use Case was identified, namely to compare activity data from the motor/premotor cortex of an awake non-human primate with the neural network model from Potjans and Diesmann (2014). The experimental data are available for an ongoing 'resting state' as well as during an active 'reach-to-grasp' task. Workflows towards the comparison between activity data and models have been designed and a preliminary version is available in the HBP collaboratory. For project 2, experimental data from epileptic patients was used to construct virtual epileptic patients (Jirsa et al., 2016). In a first Use Case Proix et al. (2017) showed the favourable application of this model to predict surgical outcome. Workflows towards the comparison between activity data and models have been designed or already made available in the HBP collaboratory https://collab.humanbrainproject.eu/#/collab/2493
MS4.5.2	Implementation of Allen Mouse Atlas as a large-scale brain model and simulation of resting state networks.	20	T4.5.2	M12	M12	Milestone has been achieved (see Deliverable 4.7.2 for details): The Allen Mouse Atlas has been used to create the structural connectivity (SC) as required for large-scale brainwork models (Melozzi et al., 2017). This procedure was then used to build the large-scale brain network models, which were in turn used to simulate resting state functional connectivity (FC). These results were used to analyse the predictive value of brain structure for brain function (Melozzi et al., in preparation; see also https://collab.humanbrainproject.eu/#/collab/1609/nav/14240).
MS4.7.1	SP4 Roadmap for SGA2	10	T4.7.1	M13	M13	Started in M8

9. T4.1.1 - Simplified dendritic neuron Models

9.1 Key Personnel

Task Leader: Idan SEGEV (HUJI, P60)

Other Researcher: Alain DESTEXHE (CNRS, P10)

9.2 SGA1 DoA Goals

1) To reduce model complexity, in both morphological (simpler cable structures) and physiological (minimal membrane ion channels) domains and compare these reduced neuron models (e.g. of L2/3 pyramidal cells) among various species.
2) Theoretical analysis of dendritic computations, in particular on the role of dendritic excitability (Na+, Ca2+ and NMDA spikes), and consideration of how nonlinear dendrites integrate synaptic inputs under *in vivo* conditions with intense background synaptic activity.

9.3 Component Progress

PLA Components:

951. complex to simplified models - owner: Guy EYAL, type: model (DoA Goal 1)

62. Simplified neuron models – owner: Idan SEGEV, type: model (DoA Goal 1)

60. Model of dendritic integration with excitable dendrites - owner: Alain DESTEXHE, type: model (DoA Goal 2)

1031. Mean-field models of interacting spiking neurons with dendritic compartment –owner: Romain VELTZ, type: model (DoA Goal 2) reported in T4.1.3

CDP Contributions:

- CDP2 - Mouse-Based Cellular Cortical and Subcortical Microcircuit Models

Component 62 Simplified neuron models is part of CDP2-UC-002 - Multi-scale validation)

9.3.1 complex to simplified models

Description of Component (from PLA): an algorithm that takes detailed 3D reconstructed neuron morphology + electrical properties and then generates a simplified conductance-based model of it.

CDP to which Component contributes (if relevant): N/A

Progress on Component: Having developed the reduction scheme we already tested it on L5 and L2/3 pyramids, as well as for cortical Martinotti and Basket cells interneurons. In all case the performance of the reduced model ad compared to the full model is excellent, and the computational time saved ranged between 50-120 folds.

Quality Control:

- Upstream - Fitting Generalised Integrate-and-FIre models
- Upstream - SP6-T6.2.1-SGA1-Models of human dendritic spines
- Upstream - SP2 - Morphological cortical connectivity profiles of neocortical pyramidal neurons
- Upstream - SP2 - Morphological data of human neocortical pyramidal neurons
- Upstream - SP6-T6.2.1-SGA1-Detailed passive models of human neurons

- Upstream - Morphological and physiological data from the same neurons in adult mouse
- Upstream - Morphological and physiological data from the same neurons in adult human
- Upstream - Template of morphing rules to translate pyramidal neuron function from rodent brain to microcircuits in the human brain
- Downstream - SP2 - Multilevel maps of quantitative cell distributions and morphologies
- Downstream - SP2 - Maps of different human neuronal circuits

9.3.2 Simplified neuron models

Description of Component (from PLA): Reduced neuronal models that preserve the cable properties of the full detailed ones.

This work continues in SGA-1 with the "Systematic methods for reducing single neuron model complexity" ("Neuron_Reduce"). This is an algorithm that takes detailed 3D reconstructed neuron morphology + electrical properties and then generates a simplified conductance-based model of it.

CDP to which Component contributes: CDP2 « Mouse-Based Cellular Cortical and Subcortical Microcircuit Models » - UC-002 - Multi-scale validation

Progress: Neuron_Reduce is a general- purpose software to reduce neuron model complexity. As such it will serve mean field models in SP4 as well as models for dendritic plasticity, as it preserves some level of morphological complexity of dendritic neurons as well as nonlinear dendritic properties.

We completed the first version of the reduction scheme (Neuron_Reduce). It provides an analytic scheme for taking a complex conductance-based neuron model as an input and automatically generate simplified, yet accurate reduced model. For L5 pyramidal cell the reduced model rums 100 times faster than the full model. We now examine this reduction scheme on variety of neuron types.

Links:

Eyal et al. Neuron_Reduce: An analytical method to efficiently reduce neuron model complexity (in preparation).

Quality Control:

- Upstream - SP2 - Morphological cortical connectivity profiles of neocortical pyramidal neurons
- Upstream - SP2 - Morphological data of human neocortical pyramidal neurons
- Upstream - Template of morphing rules to translate pyramidal neuron function from rodent brain to microcircuits in the human brain
- Upstream - Morphological and physiological data from the same neurons in adult human
- Upstream - 3D reconstructions of 200 cells in human neocortex (temporal, cingulate and frontal)
- Upstream - SP6-T6.2.1-SGA1-models of nonlinear human neurons
- Upstream - SP6-T6.2.1-SGA1-Modelling synaptic inputs to human dendritic spines
- Downstream - Single-compartmental models of cortical cells, including non-linear IF models and GLM

9.3.3 Model of dendritic integration with excitable dendrites

Description of Component (from PLA): This is a simplified model of excitable dendrites using the AdEx model, compatible with neuromorphic hardware.

This model, started in the Ramp-Up Phase, continues in SGA-1.

This models simulates signal propagation of sodium, calcium and NMDA spikes in an active linear dendrite which consists of nc dendritic compartments, each of AdEx type.

CDP to which Component contributes (if relevant): N/A

Progress on Component: A first model is now submitted for publication.

Quality Control:

- Upstream - T3.2.4 (4) Prediction of photostimulation effects through simulation and closed-loop feedback
- Upstream - Plasticity: STDP for a multi-compartment model with NMDA spikes (Algo STDPbackprop)
- Upstream - Plasticity: Dendritic predictive plasticity that reproduces STDP data (Algo STDPpredictive)
- Downstream - Plasticity: STDP algo that predicts stimuli ahead in time (Algo STDPprospective)

10. T4.1.2 - Input-output transfer functions of morphologically detailed neuronal models

10.1 Key Personnel

Task Leader: Michele GIUGLIANO (UA, P81 - leader)

Other Researcher: Idan SEGEV (HUJI, P60)

Other Researcher: Christophe VERBIST (UA, P81), Mario NEGRELLO (UA, P81)

10.2 DoA Goal(s)

We study the mapping into an output spike train of the inputs received by a neuron, under a periodic sinusoidal regime. This mapping is known as dynamical transfer function and quantifies the bandwidth of information processing associated to intrinsic neuronal excitable properties.

Changes to DoA Goal(s): No change has been foreseen for the moment.

10.3 Components Progress

A delay of 4M in the progress of T4.1.2 has being experienced. This resulted from (a) late recruitment of personnel at UA following the delayed SGA1 contract signature. In addition, (b) the part-time postdoctoral fellow recruited on the Project (Dr. M. Negrello) unexpectedly dropped-out on 31/7/2016. Immediate contingency actions were taken, recruiting a PhD researcher (C. Verbist) on 1/10/2016. Additional contingency measures included in-kind contributions (4MM undergrad researcher, 3MM IT manager, 1.5MM of M. GIUGLIANO's own time) to boost the start of the (unexperienced) PhD.

PLA Components:

1007. Single-compartmental models of cortical cells, including non-linear IF models and GLM – owner: Michele GIUGLIANO, type: model

1008. Multi compartmental reconstructed cortical cells: their input-output transfer properties - owner: Michele GIUGLIANO, type: model

CDP Contributions: CDP2 - Mouse-Based Cellular Cortical and Sub-Cortical Microcircuit Models

Component 1008 contributes to CDP2-UC-001-single cell modelling

10.3.1 Multi compartmental reconstructed cortical cells: their input-output transfer properties (from PLA)

Description of Component (from PLA): The dynamical input-output response properties of detailed models of neurons, across human and rodent cortical cell types are analysed and related to published experimental data and theoretical frameworks. Specific relationships of (linear) system theory and Fourier analysis of firing rates are obtained while injecting somatic current- and conductance-inputs and then generalised to realistic distributed synaptic activation. Consequences of dendritic impedance “loads”, AP initiation, and response non-linearity are contrasted against published experiments. These results constrain single-compartmental models using non-parametric filtering and ad hoc and spike-initiation mechanisms as in non-linear IF models and GLM. Such characterizations of detailed and simplified models will facilitate interpretation of SP6 large-scale simulations, and their examination in collaborations with SP2 and SP6 will benefit neuromorphic designs (SP9).

CDP to which Component contributes (if relevant): CDP2, Mouse-Based Cellular Cortical and Sub-Cortical Microcircuit Models, UseCase: UC-001-single cell modelling.

Quality Control:

- Upstream - SP2 - Morphological cortical connectivity profiles of neocortical pyramidal neurons
- Upstream - SP2 - Morphological data of human neocortical pyramidal neurons
- Upstream - Fitting Generalised Integrate-and-FIre models
- Upstream - 3D reconstructions of cortical neurons from cortical tissues of human brain obtained from brain surgery in hospitals
- Upstream - 3D reconstructions of cortical neurons from brain slices
- Upstream - SP6-T6.2.4-SGA1-Models of rat hippocampal neurons
- Downstream - T3.1.4 Information Theoretic Network Model of Layer 5 Pyramidal Cells
- Downstream - Single-compartmental models of cortical cells, including non-linear IF models and GLM
- Downstream - SP6-T6.2.7-SGA1-Simplified brain models
- Downstream - Modelling and analysis of a cortical slice with spontaneous and perturbed slow-wave activity
- Downstream - Command-line tools
- Downstream - SP9 model: Emergence of Computational Capabilities through Learning
- Downstream - SP9 BrainScaleS standalone next generation single chip physical model system

10.3.2 Single-compartmental models of cortical cells, including non-linear IF models and GLM

Description of Component (from PLA): This Component is based on the results from "Multi compartmental reconstructed cortical cells: their input-output transfer properties" to constrain single-compartmental models using non-parametric filtering and ad hoc and spike-

initiation mechanisms as in non-linear IF models and GLM. Such characterizations of detailed and simplified models facilitate interpretation of SP6 large-scale simulations, and their examination in collaborations with SP2 and SP6 will benefit neuromorphic designs (SP9).

10.3.3 Fitting Generalised Integrate-and-FIre models

Description of Component (from PLA): Neuron models from data: algorithmic pipeline to extract parameters from measurements. The methodology has been verified, written up in a paper that appeared in PLOS Computational Biology 2015 and will be used in the future "routinely in the Allan Institute" for large-scale data collection.

It can also be used as a tool to extract simplified neuron models from detailed biophysical neuron models.

The paper is available as:

Automated high-throughput characterization of single neurons by means of simplified spiking neuron models C. Pozzorini, S. Mensi, O. Hagens, R. Naud, C. Koch and W. Gerstner PLOS Computational Biology 2015

the code for automatic parameter extraction available at:
https://github.com/pozzorin/GIFFittingToolbox/wiki/Automated-high-throughput-single-neuron-characterization

11. T4.1.3 - Mean-field and population models

11.1 Key Personnel

Task Leader: Olivier FAUGERAS (INRIA, P33)

Other Researcher: Etienne TANRÉ (INRIA, P33), Romain VELTZ (INRIA, P33)

Other Researcher: Marc DE KAMPS (ULEEDS, P104), Yi ming LAI (ULEEDS, P104)

Other Researcher: Alain DESTEXHE (CNRS, P10),

11.2 DoA Goal(s):

- To derive mean-field and population density descriptions from point neuron models.
- To study the influence of network connectivity and noise correlations on the models' computational properties.
- To model *in vivo* conditions (high-conductance states).
- To compare this approach with the one followed in SP6 and the one in Task 4.4.1, where simulations of "brain states" will be performed with integrate and fire neurons.
- To explore whether the SpiNNaker architecture may provide hardware acceleration to multi-dimensional population density techniques

Changes to DoA Goal(s): None

11.3 Component progress

PLA Components:

1030. Mean-field models of interacting populations of rate and spiking neurons – owner: Olivier FAUGERAS, type: model (DoA Goal 1, 2)

1031. Mean-field models of interacting spiking neurons with dendritic compartment - owner: Romain VELTZ, type: model (DoA Goal 1,3)

1034. Population density techniques for the simulation of populations and neural circuits - owner: Marc DE KAMPS, type: model (DoA Goal 1,5)

1554. Mean-field model of AdEx networks, spontaneous activity and responsiveness - owner: Alain DESTEXHE, type: model (DoA Goal 4)

1054. Population activity equations: Finite-N mean-field model for interacting populations (with adaptation)- owner: Wulfram GERSTNER, type: model (DoA Goal x)

CDP Contributions: CDP1 - Development of Whole Mouse Brain Model and Related Mouse Brain Atlas

Components 1030, 1034 & 1054 contributes to CDP1-P3: A virtual imaging lab app and CDP1-P4: A virtual behaviour lab app.

11.3.1 Mean-field models of interacting populations of rate and spiking neurons

Description of Component (from PLA): We develop mathematical models of the thermodynamic limit of networks of rate and spiking neurons.

Progress on Component: We developed a systematic cumulant expansion for neuronal networks on the level of individual units. The theory allows the calculation of first and second cumulants of the finite-size fluctuations present in networks of binary units in the strong coupling regime and beyond thermodynamic equilibrium. The work was released with the publication Dahmen et al. 2016, Phys Rev X. The theory goes beyond the often employed level of description on the level of homogeneous populations. In particular, it shows that finite-size effects exist that may break the homogeneity between cells. Therefore, it is complimentary to population-density approaches followed within this work package.

The spontaneous activity of the cortical microcircuit is often dominated by collective oscillations. We have finished the work on the identification of the anatomical origin of these oscillations in the gamma range. The work has been released as Bos et al. 2016, PloS CB. The method, based on a mean-field reduction of LIF model neurons, finds an analytical measure for the contribution of individual connections to cortical oscillations. It identifies a sub-circuit of layers 2/3 and 4 as the potential origin of gamma oscillations, as well as layer 5 as the origin of slow fluctuations. Moreover, it allows the targeted model construction with desired oscillatory and stability properties.

We developed an analytical approach to treat feed-forward networks of rate units with correlation-sensitive synaptic plasticity in a mean-field approximation. The work was released as Grytskyy et al. 2016. The formalism reveals a transition at a critical coupling strength at which soliton solutions on long time-scales emerge.

Task-related references

David Dahmen, Hannah Bos, and Moritz Helias (2016) Correlated Fluctuations in Strongly Coupled Binary Networks Beyond Equilibrium. Phys. Rev. X 6, 031024

Bos H, Diesmann M, Helias M (2016) Identifying Anatomical Origins of Coexisting Oscillations in the Cortical Microcircuit. PLoS Comput Biol 12(10): e1005132. doi:10.1371/journal.pcbi.1005132

Dmytro Grytskyy, Markus Diesmann, and Moritz Helias (2016) Reaction-diffusion-like formalism for plastic neural networks reveals dissipative solitons at criticality. Phys. Rev. E 93, 062303 https://doi.org/10.1103/PhysRevE.93.062303

11.3.2 Mean-field models of interacting spiking neurons with dendritic compartment

Description of Component (from PLA): It is an algorithm for an efficient computation of dendritic signals propagation.

CDP to which Component contributes (if relevant): N/A

11.3.3 Population density techniques for the simulation of populations and neural circuits

Description of Component (from PLA): It is a set of mathematical techniques that reduce the simulation group of spiking neurons to that of a single population. We apply the resulting simulator to a model of language production.

Quality Control:

- Upstream - T3.3.3 Decoded spike patterns of neural ensembles in cortex and hippocampus during multimodal scene representation
- Upstream - Mean-field model of AdEx networks, spontaneous activity and responsiveness
- Upstream - population activity equations: Finite-N mean-field model for interacting populations (with adaptation)
- Upstream - Single-compartmental models of cortical cells, including non-linear IF models and GLM: We discussed the possibility to use population density techniques to model a two-compartmental neuron where one of the compartments receives a current injection with noise superimposed. Compartmental simulations suggest effects of the spike rise which translates in a shape change of the f-I curve. We are studying whether this can be captured by PDTs. Also Michele made a suggestion for a synaptic plasticity rule that could be captured by our techniques and we're exploring that atm.
- Downstream - Simplified EEG models: We have delivered our PDTs and simulator to Mikkel Lepperod, one of Gaute's students. They are using this to model populations of stellate cells in entorhinal cortex. We are currently investigating resonance spectra of individual populations and circuits. Direct LFP modelling will come later.

11.3.4 Mean-field model of AdEx networks, spontaneous activity and responsiveness

Description of Component (from PLA): Design of mean-field models of populations of excitatory and inhibitory neurons, including adaptation, in order to capture the population dynamics during spontaneous activity, as well as the dynamics of evoked responses. The model should predict the correct spontaneous activity states for given parameters of connectivity and synaptic weights. It should also predict the correct time course of the network response to external input.

Progress on Component: A mean-field model has been derived, and is now submitted for publication. We are now refining this model so that it captures the network responses to external input.

Quality Control:

- Downstream - Population density techniques for the simulation of populations and neural circuits

11.3.5 Population activity equations: Finite-N mean-field model for interacting populations (with adaptation)

Description of Component (from PLA): The finite-N mean-field model uses groups (populations) of generalised integrate-and-fire models with parameters extracted from data that are connected to each other in a wiring diagram consistent with cortical microcircuits. From a theoretical perspective it makes the transition from a description by single neurons (spikes, voltage) to the level of population activity (level of mean-field models, population models, Wilson-Cowan models, field equations). In contrast to the Wilson-Cowan model, the equations for population activity are:

i. derived from underlying generalised integrate-and-fire models;

ii. take into account neuronal adaptation and

iii. are valid not just for N to infinity but also for fairly small groups.

The functionalities are:

A. theoretical analysis

B. speed-up for large-scale brain simulations

C. Can be used with microcircuit structures such as those of the Potjans-Diesmann model

D. could be used as a basis for cortical wave simulations or simplified EEG models

CDP to which Component contributes: N/A

Progress: EPFL-LCN (Gerstner lab) made excellent progress on this Component. An arXive version has been published. A final paper has been submitted and is currently in the second round of review. See ANNEX to Deliverable for details.

Links: https://arxiv.org/abs/1611.00294

Archive publication: T. Schwalger, M. Deger and W. Gerstner (2016) Towards a theory of cortical columns: From spiking neurons to interacting neural populations of finite sizearXiv:1611.00294

Quality Control:

- Upstream - 944: Full density model of cortical microcircuit , Van Albada (Diesmann lab). The upstream Component is published, we have received it and it was used in our Component, and we gave positive feedback.,
- Upstream - 61: Fitting Generalized Integrate-and-FIre models., Gerstner (EPFL), The upstream Component is published, we have received the Component, and it was used in our Component, and we gave positive feedback.
- Downstream - Simplified EEG models (Einevoll) [added value] we have not officially delivered, but we have discussed with Gaute Einevoll at a preliminary state.
- Downstream - Mean-field models of interacting populations of rate and spiking neurons [essential] (Faugeras). Olivier Faugeras told us that he is very interested and he has received the preprint version of our model
- Downstream - Population density techniques for the simulation of populations and neural circuits [important] (De Kamps) we have not officially delivered, but Mark de Kamps is aware of our model and told us that he is interested.

12. T4.1.4 - Models of brain signals

12.1 Key Personnel

Task Leader: Alain DESTEXHE (CNRS, P10)

Other Researcher: Bartosz TELENCZUK (CNRS, P10)

Other Researcher: Gaute EINEVOLL (NMBU, P44), Torbjørn V NESS (NMBU, P44)

12.2 DoA Goals:

1. to obtain methods to generate LFP signals from both detailed models (as developed in SP6), and simplified models (see Task 4.1.1).

2. to extend this to the calculation of more global signals, such as the surface EEG (electrocorticogram or ECoG), voltage-sensitive dye (VSD) signals, as well as the local magnetic field (LMF) generated by neuronal populations.

Changes to DoA Goals: None

12.3 Components Progress

PLA Components:

1234. Model of calcium imaging signals – owner: Alain DESTEXHE, type: model(Goal 2 & CDP1): Biophysically based model of calcium imaging signals (this Component is part of CDP1)

63. Simplified model of local field potentials – owner: Alain DESTEXHE, type: model (DoA Goal 1): Simple model of LFPs based on unit-LFP relations from extracellular recordings

902. Simplified EEG models – owner: Gaute EINEVOLL, type: model (DoA Goal 2): We will provide a simplified and memory efficient formalism for calculating EEG signals from neural simulations in NEURON and NEST. This will help relating large scale brain simulations to experimentally measurable quantities like the EEG, and also greatly simplify analysis, by simplifying the link between the EEG signal and the underlying neural sources.

896.Improved LFP model with quasi-active conductances – owner: Gaute EINEVOLL, type: model (DoA Goal 1)

CDP Contribution: CDP1 - Development of Whole Mouse Brain Model and Related Mouse Brain Atlas

Component 1234, 902 & 896 contribute to CDP1-P3: A virtual imaging lab app.

CDP2 - Mouse-Based Cellular Cortical and Sub-Cortical Microcircuit Models

Component 63. Simplified model of local field potentials (model, Destexhe) contributes to CDP2-UC-002 - Multi-scale validation

12.3.1 Model of calcium imaging signals (from PLA)

Description of Component (from PLA): Biophysically based model of calcium imaging signals (this Component is part of CDP1)

CDP to which Component contributes: CDP1 Development of Whole Mouse Brain Model and Related Mouse Brain Atlas, CDP1-P3: A virtual imaging lab app

Progress on Component: This model starts on year 2, on April 1st 2017.

Links: N/A

Quality Control:

- Upstream - SP2 - Multilevel maps of quantitative cell distributions and morphologies
- Upstream - SP2 - Maps of different human neuronal circuits
- Downstream - Structural and functional connectivity at different scales
- Downstream - Allen Mouse Atlas (AMA) based brain network

12.3.2 Simplified model of local field potentials (from PLA)

Description of Component (from PLA): Simple model of LFPs based on unit-LFP relations from extracellular recordings.\r\n

CDP to which Component contributes: CDP2-UC-002 - Multi-scale validation, generic models and algorithms

Progress on Component: We have calculated the "kernel" to relate single spikes to LFPs, and we are now using this information to generate a simplified model of the LFP.

Quality Control:

- Upstream - Simulation of brain lesion and cortical bistability on complexity
- Upstream - Photostimulation
- Upstream - Electrical perturbations on slices during sleep-like pattern before and after drug application
- Upstream - Intracortical SPES recording combined with hd-EEG and intracortical recording
- Upstream - Slow waves and complexity relationships explored by perturbations: definition of models, T3.2.2
- Downstream - T3.2.1 (3) Multipurpose simplified neuronal network model of different cortical areas matching SWA/wake transitions
- Downstream - Modelling and analysis of slow-wave activity across a cortical area with laminar organization
- Downstream - Collective behaviour of mean-field and neural population models: A comparative study
- Downstream - Improved LFP model with quasi-active conductances
- Downstream - SP6-T6.3.6-SGA1-Tool for LFP recording in NEST simulations

12.3.3 Improved LFP model with quasi-active conductances

Description of Component (from PLA): Using quasi-active conductances, we improve the estimation of the effect of active conductances on the Local Field Potential

CDP to which Component contributes: CDP1 Development of Whole Mouse Brain Model and Related Mouse Brain Atlas, CDP1-P3: A virtual imaging lab app

Progress & links:

We recently published a paper (onlinelibrary.wiley.com/doi/10.1113/JP272022/full) about the effect of quasi-active conductances on the LFP for single cells, and we are preparing a manuscript where this has been extended to populations of cells. The framework we have developed is in principle ready to be used for investigating the impact of active conductances on the LFP in large-scale brain simulations, e.g., from SP6, or in our SGA-2 Component "*Hybrid Schemes for combining point-neuron network simulations in NEST with biophysically detailed NEURON simulations (Einevoll, T4.1.4)*". We have also finished an investigation of the impedance spectrum of cortical tissue with a focus on the propagation of LFPs (eneuro.org/content/4/1/ENEURO.0291-16.2016.full).

Quality Control:

- Upstream - nmc-portal, (T5.6.1, RUP) We have extensively used the MNC-portal (bbp.epfl.ch/nmc-portal), and it has been a great resource, although we could have wished for more information on the connectivity.
- Upstream - NEURON. We have used NEURON to simulate the neural activity underlying all our LFP calculations.
- Upstream - NEST Support for Modellers, (T7.5.5, SGA1) When needed, we have received support for using NEST.
- Upstream - NEST - The Neural Simulation Tool. We have used NEST for producing plausible network activity that is used as input for cell populations.

- Upstream - Simplified model of local field potentials (T4.1.4, SGA1). We have so far not used input from this Component
- Downstream - Tool for LFP recording in NEST simulations (T6.3.6, SGA1). We have established contact, and made some initial plans for our collaboration.
- Downstream - Simplified EEG models (T4.1.4, SGA1). The quasi-active conductance scheme is ready to be used to probe the impact of active conductances on EEG recordings.
- Downstream - Multi-area recordings from visual and somatosensory cortices, perirhinal and entorhinal cortex and hippocampal CA1 (T3.3.3, SGA1). We have so far not contributed to this Component
- Downstream - Decoded spike patterns of neural ensembles in cortex and hippocampus during multimodal scene representation (T3.3.3, SGA1). We have so far not contributed to this Component

12.3.4 Simplified EEG models

Description of Component (from PLA): We will provide a simplified and memory efficient formalism for calculating EEG signals from neural simulations in NEURON and NEST. This will help relating large scale brain simulations to experimentally measurable quantities like the EEG, and also greatly simplify analysis, by simplifying the link between the EEG signal and the underlying neural sources.

CDP to which Component contributes: CDP1 Development of Whole Mouse Brain Model and Related Mouse Brain Atlas, CDP1-P3: A virtual imaging lab app

Progress & links:

We have developed and tested a framework for simplified EEG calculation from complex neuron models (master thesis, hdl.handle.net/11250/292868), and we plan to finish a manuscript on it during the fall 2017. Currently, we are preparing a short note about analytic four-sphere EEG models that will be useful when exploring the simplified EEG models.

Quality Control:

- Upstream - nmc-portal, (T5.6.1, RUP) We have extensively used the MNC-portal (bbp.epfl.ch/nmc-portal), and it has been a great resource, although we could have wished for more information on the connectivity.
- Upstream -NEURON. We have used NEURON to simulate the neural activity underlying all our LFP calculations.
- Upstream - NEST Support for Modellers, (T7.5.5, SGA1) When needed, we have received support for using NEST.
- Upstream - NEST - The Neural Simulation Tool. We have used NEST for producing plausible network activity that is used as input for cell populations.
- Upstream - population activity equations: Finite-N mean-field model for interacting populations (with adaptation) (T4.1.3, SGA1). We have so far not used input from this Component.
- Upstream - Intracortical SPES recording combined with hd-EEG and intracortical recording (T3.2.2, SGA1). We have so far not used data from this Component, but it might be important at a later stage
- Upstream - Population density techniques for the simulation of populations and neural circuits (T4.1.3, SGA1). We have so far not used input from this Component, but we have made initial plans for the collaboration

- Upstream - Improved LFP model with quasi-active conductances (T4.1.4, SGA1). We will use the quasi-active conductances to probe the impact of active conductances on the EEG signal.
- Upstream - Detailed models of human cortical neurons (T6.1.3, RUP). We have so far not used data from this Component, but detailed models of human cortical neurons will be important later.
- Downstream - Tool for LFP recording in NEST simulations (T6.3.6, SGA1). We have established contact, and will collaborate on LFP recordings from NEST.
- Downstream - Point-neuron model of the whole mouse brain (T6.2.6, SGA1). Large-scale point-neuron models will be very important for our work, and this Component will be important at a later stage.

13. T4.2.1 - Simplified network models of different cortical areas

13.1 Key Personnel

Task Leader: Markus DIESMANN (JUELICH, P20, lead)

Other Researcher: Viktor JIRSA (AMU, P78, lead), Davide LILLO (AMU, P78)

Other Researcher: Sacha van ALBADA (JUELICH, P20), Rembrandt BAKKER (JUELICH, P20), Maximilian SCHMIDT (JUELICH, P20), Moritz HELIAS (JUELICH, P20), Jannis SCHUECKER (JUELICH, P20)

13.2 DoA Goal(s):

1. The Task will construct multi-layered multi-area models of the cortex relating the local microscopic connectivity to the macroscopic connectivity of the brain. On the local level, this leads to models with a higher degree of self-consistency than previously possible, because the origins of synapses from remote sources are included, and the lower parts of the power spectrum of neuronal activity missing in purely local models can be investigated. On the global level, the bottom-up and top-down flow of activity between cortical areas in these hierarchical models are investigated and compared to results from neuronal mass models.

Changes to DoA Goal(s): none.

13.3 Components Progress

PLA Components:

Owner of Components 730 and 944 has been changed (initially entered as owned by Markus Diesmann, the owner is now Sacha van Albada) ; a mistake on the owner and task contribution of Component 777 has been corrected (previously Sonja Gruen à Markus Diesmann)

730. Multi-area model of cortical network at neuronal resolution – owner: Sacha VAN ALBADA, type: model (DoA Goal 1).

777. 4x4 mm2 motor cortex model – owner: Markus DIESMANN, type: model (DoA Goal 1)

944. Full density model of cortical microcircuit– owner: Sacha VAN ALBADA, type: model (DoA Goal 1)

1573. Neuro-glial model for bursting activity - owner: Viktor JIRSA, type: model (DoA Goal 1)

CDP Contributions: *Component 730 and 777 link to* CDP4 - Visuo-Motor Integration*, Use Case «comparative analysis of experimental and simulated data»* with more concrete links to be made in SGA2. The 4x4 mm^2 model provides the microscopic template substrate for mapping the population-level model constructed in CDP4, and the multi-area model provides anatomical constraints as well as a template implementation and techniques for the multi-scale modelling of inter-area interactions.

13.3.1 Multi-area model of cortical network at neuronal resolution

Description of Component (from PLA): Construct multi-layered multi-area models of the cortex relating the local microscopic connectivity to the macroscopic connectivity of the brain. On the local level, this leads to models with a higher degree of self-consistency than previously possible, because the origins of synapses from remote sources are included, and the lower parts of the power spectrum of neuronal activity missing in purely local models can be investigated. On the global level, the bottom-up and top-down flow of activity between cortical areas in these hierarchical models are investigated.

CDP to which Component contributes (if relevant): CDP4 - Visuo-Motor Integration, *comparative analysis of experimental and simulated data*

Progress: In SGA1, JUELICH published a paper (Schuecker et al., 2017) on the stabilization of the dynamics using high-level constrains with the aid of the mean-field tool set from T4.1.3 (Faugeras). We performed graph theoretical analyses of the corresponding connectivity matrix, and submitted a paper on the joint microscopic and macroscopic anatomy of the model. Further analysis has been performed on the origin of area-specific time scales of the spiking dynamics. A release of the model is planned for the end of SGA1.

Links:

- Schmidt M, Bakker R, Hilgetag C-C, Diesmann M, van Albada SJ. Multi-scale account of the network structure of macaque visual cortex (submitted for publication, 2017).
- Schuecker J, Schmidt M, van Albada SJ, Diesmann M, Helias M. Fundamental activity constraints lead to specific interpretations of the connectome (2017) PLoS CB 13(2):e1005179, doi:10.1371/journal.pcbi.1005179.

Quality Control:

Upstream Components

- Full density model of cortical microcircuit: finished Component, now an integral part of the present Component
- Macaque connectivity database (CoCoMac): intermediate release, used extensively by the present Component
- Python: intermediate release, used extensively by the present Component
- Elephant: intermediate release, not yet used by the present Component
- SP6-T6.3.6-SGA1-Tool for LFP recording in NEST simulations: not yet released
- SP6-T6.3.6-SGA1-Tools for configuring stimulation and recording in NEST simulations: not yet used by the present Component
- HPC systems at JSC: used extensively by the present Component
- Rule- and data-based connectivity generation in NEST: the present Component uses NEST connectivity functions for which the latest NEST release contains updates and bug fixes
- NEST Support for Modellers: ongoing, extensively used by the present Component
- NEST - The Neural Simulation Tool: ongoing, extensively used by the present Component

Downstream Components

- Mouse cortical regions for object recognition learning: not yet used by the downstream Component
- VisNEST: finished Component, already used for visualizations with VisNEST
- Rule- and data-based connectivity generation in NEST: we have not yet provided requirements for novel connectivity functions to the downstream Component
- NEST Requirements Management: we have generated requirements regarding novel recording devices, successfully addressed by the downstream Component

13.3.2 4x4 mm2 motor cortex model

Description of Component (from PLA): Microcircuit model extended to 4x4 mm^2 with motor connectivity.

CDP to which Component contributes (if relevant): CDP4 - Visuo-Motor Integration, *comparative analysis of experimental and simulated data*

Progress: The work towards this Component started in the RUP and the work will continue in SGA2. A preliminary version of the model has been developed and presented at CNS*2016, and first iterations comparing simulated and experimentally recorded activity have been performed together with T4.5.1 (Gruen). We expect to provide a release in SGA2.

Hagen E., Senk J., van Albada S.J., Diesmann M.: Local field potentials in a 4 × 4 mm² multi-layered network model. BMC Neuroscience 2016, 17(Suppl 1): P167

Quality control

Upstream Components:

- Full density model of cortical microcircuit: finished Component, now an integral part of the present Component
- NEST Requirements Management: This upstream Component successfully provides an ongoing service, which has for instance led to improved performance of threaded connection generation.
- NEST Support for Modellers: ongoing, extensively used by the present Component
- NEST - The Neural Simulation Tool: ongoing, extensively used by the present Component
- HPC systems at JSC: used extensively by the present Component

Downstream Components:

- Elephant: intermediate release, not yet used by the present Component
- Workflow for comparison of electrophysiological and simulated data: intermediate release, a prototype version of the model has been used to create a first version of the workflow
- VIOLA: intermediate release, already used for visualizations of simulation results

13.3.3 Full density model of cortical microcircuit

Description of Component (from PLA): the model has been published as

Potjans TC and Diesmann M (2014) The Cell-Type Specific Cortical Microcircuit: Relating Structure and Activity in a Full-Scale Spiking Network Model. Cerebral Cortex 24(3):785-806. DOI:10.1093/cercor/bhs358

The model is maintained and curated by task T4.2.1.

CDP to which Component contributes (if relevant): N/A

Progress: In the current grant period, a PyNEST model description has been developed and made available in the latest NEST release (Kunkel et al., 2017) as well as on Open Source

Brain. Furthermore, the model has been ported to SpiNNaker using a PyNN model description. This description has entered into an example workflow using the Collaboratory in combination with HPC resources and neuromorphic hardware, led by T4.5.1 (Gruen). The plan for SGA2 is to further increase the agreement of the model with fundamental properties of local cortical circuits like simultaneous balance and excitability. As an auxiliary study for these SGA2 plans, JUELICH re-implemented an earlier model of local cortical circuits from the literature in NEST (Maksimov et al., 2016).

Links:

- http://opensourcebrain.org/projects/potjansdiesmann2014
- Kunkel S et al. (2017). NEST 2.12.0. Zenodo. 10.5281/zenodo.259534.
- Van Albada SJ, Rowley AG, Hopkins M, Schmidt M, Senk J, Stokes AB, Galluppi F, Lester DR, Diesmann M and Furber SB (2016). Full-scale simulation of a cortical microcircuit on SpiNNaker. *Front. Neuroinform. Conference Abstract: Neuroinformatics 2016.* doi: 10.3389/conf.fninf.2016.20.00029.
- Johanna Senk, Alper Yegenoglu, Olivier Amblet, Yury Brukau, Andrew Davison, David Roland Lester, Anna Lührs, Pietro Quaglio, Vahid Rostami, Andrew Rowley, Bernd Schuller, Alan Barry Stokes, Sacha Jennifer van Albada, Daniel Zielasko, Markus Diesmann, Benjamin Weyers, Michael Denker, Sonja Grün (2017) A Collaborative Simulation-Analysis Workflow for Computational Neuroscience Using HPC. In: Di Napoli E., Hermanns MA., Iliev H., Lintermann A., Peyser A. (eds) High-Performance Scientific Computing. JHPCS 2016. Lecture Notes in Computer Science, vol 10164. Springer, Cham, doi:10.1007/978-3-319-53862-4_21
- **[Re] Cellular and Network Mechanisms of Slow Oscillatory Activity (<1 Hz) and Wave Propagations in a Cortical Network Model** Andrei Maksimov, Sacha J. van Albada (JUELICH), and Markus Diesmann (JUELICH) published in ReScience, 2016 October 17, Open Access

Upstream Components:

- NEST Requirements Management: This upstream Component successfully provides an ongoing service, which has for instance led to improved performance of threaded connection generation.
- NEST code with abstracted neuron model representations: This upstream Component delivered what it promised.

Downstream Components:

- Neuro-glial model for bursting activity: finished Component, used as example network by the downstream Component
- population activity equations: Finite-N mean-field model for interacting populations (with adaptation): finished Component, used as biological example network by the downstream Component
- Elephant: finished Component, delivers simulated activity data for testing statistical analysis functions implemented in the downstream Component
- Multi-area model of cortical network at neuronal resolution: finished Component, now an integral part of the downstream Component
- 4x4 mm2 motor cortex model: finished Component, now an integral part of the downstream Component

13.3.4 Neuro-glial model for bursting activity - owner: Vitkor Jirsa

Description of Component (from PLA): The impairment of neuron-glia cross talk may be responsible, among other factors, for the onset of seizures in brain cortex. We aim to build

a network model for neuro-glial tissue with a bottom-up approach: starting from a physiologically detailed and biophysically validated point-neuron model, embedded in its metabolic environment enclosing glial activity and interaction, we study its bursting properties and identify the minimal set of fast/slow variables that are involved in the bursting dynamics. This should lead to identification of a local slow permittivity variable modulating ictal-interictal alternation. Further mean-field derivation from this point-model may provide a mesoscopic biomarker of seizure, with spatial propagation depending on the connectivity of the cortical structure at study. Continuing up along the hierarchy, we may be able to build a simplified model for whole-area seizure, keeping into account local synaptic cabling and cross-area signalling. A resulting slow, large-scale variable of seizure modulation should be experimentally identifiable and measurable, to assess conclusions about seizure paths in the cortex as well as new therapeutic strategies for pharmacoresistant epileptic patients.

CDP to which Component contributes: Not contributing yet, but in the next phase expected to contribute to Development of Whole Mouse Brain Model and Mouse Brain Atlas (CDP1), we are responsible for the P3 Virtual imaging lab app

Progress: We started by considering a HH-type neuron with persistent K+ current, transient Na+ current, leak Cl- current and ATP pump current. The conductance-based equations for this model are coupled to the equations describing the change of ionic concentrations in the intracellular and extracellular space, as well as to the equation for passive diffusion from the K+ bath. The total set of 11 variables is then reduced to a set of 4 variables thanks to mass/charge constraints and approximations of gating dynamics. The reduced system is then analysed in slow/fast fashion. A 2D bifurcation diagram of the fast subsystem, comprised of membrane potential and K+ activation variable, is obtained for each fixed couple of values of the two slow variables (intra- and extracellular shift of K concentration from resting values). The passive diffusion from external K+-bath induces a slow-wave bursting behaviour of the neuron for a certain range of bath concentrations. This can be visualised as a closed trajectory in the slow-variables' plane, which crosses a SNIC line in both onset and offset bifurcations of the fast subsystem. Such SNIC/SNIC burster appears to be robust to slight changes of the metabolic parameters in the model, but bursters of other classes are observed as transients before stabilization to the aforementioned class. Our model is able to combine realism and computational handiness to reproduce a K+-elevation-induced bursting.

For the next phase we are trying to translate this local, point-neuron model into a mesoscale population mean-field model that will be able to describe dynamics inside a sketched copy of cortical column. Different approaches are available from master equation theory. The population dynamics should be a function of the biophysical parameters we have plugged inside the single-cell model. The resulting mean-field formalism may give us hints about what are the conditions (within our model) that lead to hyper-synchronous activity of the overall population and eventually to a spreading of the seizure to neighbouring areas. We should then compare our results with experimental literature in order to check validity and consistency of our workflow. We are confident about the possibility of bridging physiological knowledge about cortical focal seizures with a corresponding mathematical characterization.

Hired PhD student Davide Lillo.

Quality Control:

Upstream Components

- Model of biologically-realistic network states [added value]; It is not yet needed, we expect it in the next phase.
- population activity equations: Finite-N mean-field model for interacting populations (with adaptation) [important]; It is not yet needed, we expect it in the next phase.

- Full density model of cortical microcircuit [important]; It is not yet needed, we expect it in the next phase.

Downstream Components:

- SOFTWARE > Data Factory > Data Storage [essential]; Not yet implemented (we believe that this platform does not exist and hasn't been needed yet, but it should become relevant in the next phase)
- Effective connectivity changes inferred from optogenetic brain interrogation and calcium imaging [added value]; The Component is not yet active, and its connection are under discussion.
- Allen Mouse Atlas (AMA) based brain network; It is not yet implemented. We expect it to happen in the next phase.

14. T4.2.2 - Network models including neuron-glia interactions

14.1 Key Personnel

Task Leader: Marja-Leena LINNE (TUT, P103)

Other Researcher: Andre GRÜNING (SURREY, P102)

14.2 DoA Goal(s):

The objectives of our Task are:

1. To develop a framework to model neuron-astroglia interactions in plasticity and learning, with data from SP1;
2. To formulate novel plasticity algorithms that include mathematically abstracted astroglial functions;
3. To construct models of cortical networks based on (1) and (2).

14.3 Changes to DoA Goal(s):

No change in DoA Goals are expected. Since we did not have funding from April 2016 (and, consequently, the postdoc working in the Project was unemployed during this period of time), we have done the work using small institutional funding. This institutional funding was originally allocated to other applications than the ones proposed by us in the HBP. Due to delay in EU funding and complication using the institutional funding the work has so far addressed mainly Goals (1) and (2) of the DoA.

14.4 Component progress

PLA Components:

70. Astrocyte neuron interaction SYNAPSE model (ANI model) – owner: Marja-Leena LINNE, type: model (DoA Goal 1)

973. Astrocyte-Neuron interaction NETWORK model (ANN model) – owner: Marja-Leena LINNE, type: model (DoA Goal 3)

CDP Contributions: None for the moment, we do not receive funding through CDPs. But T4.2.2 is involved in following the progress made in CDP5 through Andre Gruning.

14.4.1 Astrocyte neuron interaction SYNAPSE model (ANI model)

Description of Component (from PLA): The astrocyte-neuron interaction (ANI) model is a detailed biophysical excitability model that involves the astrocyte, and the presynaptic and postsynaptic terminals of the so-called tripartite synapse. The model describes key cell membrane excitability as well as intracellular calcium mechanism that are shown to take part in the information transfer in the cortical synapse.

The SYNAPSE model for plasticity, involving astrocyte modulation (named as ANI model) will continue in SGA-1.

CDP to which Component contributes: CDP5, indirectly through Andre Grüning.

Progress: We have started the work towards the Component during RUP and the work continues in SGA2. All model elements to form the Component are now finalised and implemented. We are currently performing extensive testing of the model, validation of the model against experimental data, and writing up the publication. We expect to release the Component after the acceptance of the publication and no later than the end of SGA1.

Links: We have already published several publications in relation to this Component. These publications present work on initial selection of Component elements (ie. model elements). Publications and materials include:

Manninen T., Havela R., Linne M.-L. (2017) Computational models of astrocytes and astrocyte-neuron interactions: Categorization, analysis, and future perspectives. Computational Glioscience (peer-reviewed book chapter), 2017 (in press).

Manninen T., Havela R., Linne M.-L. (2017) Reproducibility and comparability of computational models for astrocyte calcium excitability. Frontiers in Neuroinformatics 11:11. https://doi.org/10.3389/fninf.2017.00011

Quality Control:

- Upstream - Neuro-Glia Vasculature (model) by T6.4.3 (RUP) Molecular models of neuro-vascular-glial coupling. T4.2.2 has been in contact with the developers and visited Geneva location to discuss about the Component. The upstream model can be used to simulate data for the use of the ANI model (Component 70).
- Downstream - Astrocyte-Neuron interaction NETWORK model (ANN model) by T4.2.2 (SGA1) Network models including neuron-glia interactions + Task responsible. The downstream Component development is in the very early phase, due to delay in funding.

14.4.2 Astrocyte-Neuron interaction NETWORK model (ANN model)

Description of Component (from PLA): ANN model involves detailed models of neurons, astrocytes, and synapses. To simulate the model, we use mathematical model order reduction techniques which we have developed recently. In the future, there may be a need to implement model order reduction techniques as software (in the Simulation Platform).

CDP to which Component contributes: CDP5, indirectly through Andre Grüning.

Progress: The development of this Component is in its early phase. We have developed and tested the dimensionality reduction (model order reduction) methodology used to simulate this model. We are in the process of specifying the network level model elements and selecting experimental data to be used for model validation. The release of the Component will happen at earliest at the end of SGA1, also due to delay in funding in the beginning of SGA1.

Links: One publication is accepted (description of the algorithm to perform the dimensionality reduction).

Lehtimäki M., Paunonen L., Pohjolainen S., Linne M.-L. (2017) Order reduction for a signaling pathway model of neuronal synaptic plasticity. IFAC2017 Conference (accepted).

Quality Control:

- Upstream - Astrocyte neuron interaction SYNAPSE model (ANI model) by T4.2.2. The upstream Component will be finalised during 2017 after which it can be used in the present Component.
- Upstream - Plasticity: Two-compartment neuron by T4.3.1, T4.3.2, and T4.3.3. Discussion is ongoing how to utilise the upstream Component in the future version of the ANN Component.
- Downstream - Plasticity models by T. 4.3.1 Synaptic plasticity and learning. The connection has been initialised very recently.

15. T4.3.1 - Plasticity algorithms

15.1 Key Personnel

Task Leader: Wulfram GERSTNER (EPFL, P1)

Other Researcher: Walter SENN (UBERN, P71)

15.2 DoA Goals:

1. To formulate synaptic plasticity algorithms from experimental data, in a way compatible with the models developed in the Platforms.
2. To develop models of learning and reward, compatible with neuromorphic systems, and finally develop models of behavioural learning and long-term memory in the brain.
3. The derivation of learning rules from plausible synapse models, the identification of rules for unsupervised learning and learning under the control of neuromodulators (encoding reward, surprise and novelty).
4. Functional consequences and the long-term memory capabilities of the brain.

Changes to the DoA Goals: No changes to the research plan are foreseeable at this stage. No deviations are visible at this point into the SGA1, and no corrective actions need to be taken.

15.3 Components Progress

PLA Components linked to this task:

66. Plasticity: STDP for structural plasticity (STDPstructural) – owner: Wulfram Gerstner, type: model (DoA 1-4), reported under task 4.3.2

969. Plasticity: Two-compartment neuron – owner: André Grüning, type: model (DoA x), reported under task 4.3.3

1033. Plasticity: STDP for a multi-compartment model with NMDA spikes (Algo STDPbackprop) – owner: Walter Senn, type: model (DoA Goals 1-4). Reported under task 4.3.2

1066. Plasticity models: SP 4 (theory) T. 4.3.1 synaptic plasticity and learning (DoA Goals 1-4). Owner: W. Gerstner

1182. Plasticity: voltage-based STDP (Clopath model) – owner: Wulfram Gerstner, type: model (DoA 1)

1032. Plasticity: Dendritic predictive plasticity that reproduces STDP data (Algo STDPpredictive) – owner: Walter Senn, type: model (DoA 1-4)

1203. Plasticity: INST/FILT Rule – owner: André Grüning, type: model (DoA goal 1-4) reported under T4.3.3

CDP contributions: Component 1033, 969 and 1203 are relevant for CDP5.

15.3.1 Plasticity models: SP 4 (theory) T. 4.3.1 synaptic plasticity and learning

Description of Component (from PLA): This Component refers to the TASK plasticity in SubProject 4 (theory). It resembles several upstream Components. Please look at each of these Components to find out more.

CDP to which Component contributes (if relevant): CDP No., CDP Name & Use Case

Progress: For several models we have received the upstream Component (see below) and checked the compatibility with NEST. Currently none of the models has been officially released in NEST but a pipeline has been established, via contacts with Markus Diesmann and Hans-Ekkehard Plesser who represent the NEST development team in HBP.

Links: Not applicable.

Quality Control:

- Upstream -1182 Plasticity: voltage-based STDP (Clopath model), (Gerstner). We have received the model. The upstream Component is of excellent quality. We tested NEST compatibility, but within the current version of NEST the model is not implementable.
- Upstream - 66 Plasticity: STDP for structural plasticity (STDPstructural) (Gerstner). We have received the model. The upstream Component is of excellent quality. We tested NEST compatibility, but it would further adjustments of the NEST software to make it easily implementable in NEST.
- Upstream - 65 Plasticity: STDP with heterosynaptic plasticity and homeostasis for memory formation (Gerstner). We have received the model. The upstream Component is of excellent quality. We tested NEST compatibility, but within the current version of NEST the model is not implementable.
- Downstream - 980 Model for high-level contributions to low-level vision (Ullman). We have not officially released or delivered any model, but discussions are in progress in view of SGA2.

  Analogous statements hold for other downstream Components: we expect that the downstream path will become more important in SGA2.

15.3.2 Plasticity: voltage-based STDP (Clopath model)

Description of Component (from PLA): Synaptic plasticity is broader then standard spike-timing dependent plasticity (STDP), because synapses can change without postsynaptic spike if presynaptic activity is combined with postsynaptic voltage. This Component is based on the CLOPATH model (Nature Neuroscience, 2010). The aim is to adapt it so that it fits published dendritic plasticity experiments of Letzkus et al.

The standard reference for this model is pre-HBP:
Connectivity reflects coding: a model of voltage-based STDP with homeostasis
C Clopath, L Büsing, E Vasilaki, W Gerstner
Nature neuroscience 13 (3), 344-352

CDP to which Component contributes (if relevant): CDP No., CDP Name & Use Case

Progress: We made a modification of the Clopath model that enables us to better describe data of plasticity in dendrites. See Annex to Deliverable for more detail.

Links: Not applicable.

Quality Control:

- Downstream 1066 Plasticity models: SP 4 (theory) T. 4.3.1 synaptic plasticity and learning. The original version of the Clopath model was delivered downstream.

15.3.3 Plasticity: Dendritic predictive plasticity that reproduces STDP data (Algo STDPpredictive)

Description of Component (from PLA): The algorithm is derived as a gradient-based plasticity rule in a supervised learning scenario, but the same rules also work in a reinforcement learning scenario (when modulated by reward) and in an unsupervised learning scenario (depending on the somato-somatic connectivity pattern that acts as a self-supervision signal). The algorithm is an extension of a previous model and shows that dendritic predictive plasticity can reproduce most of the experimental results on spike-timing dependent synaptic plasticity (SDTP) on the dendritic tree. A neuron is still reduced to 2 compartments, a somatic and dendritic compartment, but the action potential and all synaptic inputs is modelled by conductances. The dendritic predictive plasticity model is applied to a population of neurons that controls the joints of a line man who learn to stand up and walk in a combination of unsupervised, supervised and reinforcement learning.

CDP to which Component contributes (if relevant): CDP No., CDP Name & Use Case

Progress: The theoretically derived error-correction rule was successfully applied to synaptic plasticity data. It does reproduce many of the STDP data for synapses depending on the location on the dendritic tree, as this was envisaged in the Project proposal. The doctoral student working on that Project now finalises the application to the virtual line man that learns to stand up and walk based on unsupervised, supervised and reinforcement learning. For details see Annex

Links: Cosyne abstract 2017, http://cosyne.org/cosyne17/Cosyne2017_program_book.pdf.

Quality Control:

- Upstream - 1182 Plasticity: voltage-based STDP (Clopath model), (Gerstner). We have received the model, used it to identify the original experimental data and compare the fits between the Clopath model and ours
- Downstream Components: See the various downstream links in the PLA.

16. T4.3.2 - Learning in networks of neurons

16.1 Key Personnel

Task Leader: Walter SENN (UBERN, P71)

Other Researcher: Wulfram GERSTNER (EPFL, P1)

Other Researcher: Misha TSODYKS (WEIZMANN, P84),

16.2 DoA Goal(s)

1. To formulate synaptic plasticity algorithms from experimental data, in a way compatible with the models developed in the Platforms.
2. To develop models of learning and reward, compatible with neuromorphic systems, and finally develop models of behavioural learning and long-term memory in the brain.

3. The derivation of learning rules from plausible synapse models, the identification of rules for unsupervised learning and learning under the control of neuromodulators (encoding reward, surprise and novelty).
4. Functional consequences and the long-term memory capabilities of the brain.

Changes to the DoA Goal(s): There are no changes and no deviations.

16.3 Components Progress

PLA Components:

1066. Plasticity models: SP 4 (theory) T. 4.3.1 synaptic plasticity and learning – owner: wolfram GERSTNER, type model (DoA Goal 1) reported under Task 4.3.1

969. Plasticity: Two-compartment neuron – owner: André Grüning, type: model (DoA Goal 1-4) reported under Task 4.3.3

1033. Plasticity: STDP for a multi-compartment model with NMDA spikes (Algo STDPbackprop) – owner: Walter SENN, type model (DoA Goal x)

1032. Plasticity: Dendritic predictive plasticity that reproduces STDP data (Algo STDPpredictive) - owner Walter Senn, type model (DoA Goals 1-4). This Component is relevant for CDP5.

66. Plasticity: STDP for structural plasticity (STDPstructural) – owner: Gerstner, type: model (DoA Goal 1-4). This Component is relevant for NEST and CDP5.

971. Neural network model of working memory – owner: Misha TSODYKS, type: model (DoA Goals 2-4). Relevant for the link to cognitive sciences as well as WP4.4.

CDP Contributions: CDP5 - Functional Plasticity for Learning in Large-Scale Systems

Components 1032 & 66 contributes to CDP5

16.3.1 Plasticity: STDP for a multi-compartment model with NMDA spikes (Algo STDPbackprop)

Description of Component (from PLA): Error-backpropagation is a successful algorithm for supervised learning in neural networks. Whether and how this technical algorithm is implemented in cortical structures, however, remains elusive. Also STDPbackprop is a version of the original error-backpropagation algorithm for spiking neurons equipped with nonlinear dendritic processing. An error expressed as mismatch between somatic firing and membrane potential may be backpropagated to the active dendritic branches where it modulates synaptic plasticity. While the original algorithm only considered firing rates, the biological implementation enables learning for both a firing rate and a spike-timing code. Moreover, when modulated by a reward signal, the synaptic plasticity rule maximises the expected reward in a reinforcement learning framework

Progress: Paper has been published at PLoS Computational Biology.

Links: Schiess M, Urbanczik R and Senn W: **Somato-dendritic Synaptic Plasticity and Error-backpropagation in Active Dendrites.** PLoS Comput Biol. 2016 Feb 3;12(2):e1004638. doi: 10.1371/journal.pcbi.1004638. eCollection 2016.

Quality Control:

- For up- and downstream Components see PLA.

16.3.2 Plasticity: Dendritic predictive plasticity that reproduces STDP data (Algo STDPpredictive)

Description of Component (from PLA): AlgoProspective is a synaptic plasticity rule that learns predictions on a single neuron level on a timescale of seconds. The learning rule allows a spiking two-compartment neuron to match its current firing rate to its own expected future discounted firing rate. For instance, if an originally neutral event is repeatedly followed by an event that elevates the firing rate of a neuron, the originally neutral event will eventually also elevate the neuron's firing rate. The plasticity rule is a form of spike timing dependent plasticity in which a presynaptic spike followed by a postsynaptic spike leads to potentiation. Even if the plasticity window has a width of 20 milliseconds, associations on the time scale of seconds can be learned. We illustrate prospective coding with three examples: learning to predict a time varying input, learning to predict the next stimulus in a delayed paired-associate task and learning with a recurrent network to reproduce a temporally compressed version of a sequence. In the special case that the signal to be predicted encodes reward, the neuron learns to predict the discounted future reward and learning is closely related to the temporal difference learning algorithm TD(λ).

Progress: Paper has been published in PLoS Computational Biology

Links: Brea J, Gaál AT, Urbanczik R, Senn W: **Prospective Coding by Spiking Neurons.** PLoS Comput Biol. 2016 Jun 24;12(6):e1005003. doi: 10.1371/journal.pcbi.1005003. eCollection 2016

Quality Control:

- For up- and downstream Components see PLA.

16.3.3 Plasticity: STDP for structural plasticity (STDPstructural)

Description of Component (from PLA): Learning & synaptic plasticity: Multi-contact synapses for stable networks: a spike-timing dependent model of dendritic spine plasticity and turnover

We have implemented a new model for the slow processes of formation, maturation, shrinkage and removal of excitatory synaptic contacts, based on spike timing dependent plasticity in combination with creation of new synapses. Main results are that the distribution of number of contacts has the typical bimodal shape: either there is no connection between neurons or the two neurons make 5+/2 connections. The simulation runs in big networks and has been used to study lesion experiments.

Progress: Ultimately, the aim is to make the simulation code available in NEST. The submission to NEST (in the form of a github release) has been done 1.12.2016.

The paper is now under review for journal publication.

Links:

Preprint available at: http://arxiv.org/abs/1609.05730 (Deger et al. 2016)

Quality Control:

- Upstream Component name (from PLA) + Task responsible + status (i.e. you have received nothing / intermediate release / finished Component) + your assessment of quality of upstream Component
- Downstream Component name (from PLA) + Task responsible + status (i.e. you have provided nothing / intermediate release / finished Component) + feedback from downstream Component's Task on quality of your Component

16.3.4 Neural network model of working memory

Description of Component (from PLA): The model aims to understand the origins of working memory capacity.

Progress on Component, including: planned releases achieved / which partner did what

Links: Include links to relevant material available publicly online.

Quality Control:

- Upstream Component name (from PLA) + Task responsible + status (i.e. you have received nothing / intermediate release / finished Component) + your assessment of quality of upstream Component
- Downstream Component name (from PLA) + Task responsible + status (i.e. you have provided nothing / intermediate release / finished Component) + feedback from downstream Component's Task on quality of your Component

17. T4.3.3 - Functional plasticity for multi-compartment neurons

17.1 Key Personnel

Task Leader: Andre GRÜNING (SURREY, P102)

Other Researcher: Walter SENN (UBERN, P71)

17.2 DoA Goal(s)

1. To formulate synaptic plasticity algorithms from experimental data, in a way compatible with the models developed in the Platforms.
2. To develop models of learning and reward, compatible with neuromorphic systems, and finally develop models of behavioural learning and long-term memory in the brain.
3. The derivation of learning rules from plausible synapse models, the identification of rules for unsupervised learning and learning under the control of neuromodulators (encoding reward, surprise and novelty).
4. Functional consequences and the long-term memory capabilities of the brain.

Changes to the DoA Goal(s): The hypercolumn simulations turn out to be too slow, even after reducing the complexity of the individual neurons. therefore, instead of an embedded simulation in the hypercolumn, we envisage running only a single multi-compartment neuron. Due to reduction in the SGA1 budget, SURREY (P102) concentrates on the neuromorphic implementations and learning showcases as required for CDP5.

17.3 Components Progress

PLA Components:

969. Plasticity: Two-compartment neuron – owner: André Grüning, type: model (DoA Goals 1-4).

1348. Plasticity: STDP for imitation learning (Algo STDPimitation) – owner: Andre Grüning, type: model (DoA Goal x)

1033. Plasticity: STDP for a multi-compartment model with NMDA spikes (Algo STDPbackprop) – owner: Walter SENN, type: model (DoA Goal x)

1032. Plasticity: Dendritic predictive plasticity that reproduces STDP data (Algo STDPpredictive) – owner: Walter SENN, type: model (DoA Goal x)

CDP Contributions: CDP5 - Plasticity, Learning and Development: modelling the Dynamic brain

Component 969, 1348 and 1033 contribute to CDP5-P3: Guiding Platform design on functional plasticity

Component 969, 1348 and 1033 contribute to CDP5-P4: Concept showcases in big-systems.

17.3.1 Plasticity: INST/FILT Rule

Description of Component (from PLA): The INST and FILE rules are efficient supervised learning rules for spiking neural networks: one relies on an instantaneous error signal to modify synaptic weights in a network (INST rule), and the other on a filtered error signal for smoother synaptic weight modifications (FILT rule). They have been tested with respect to their temporal encoding precision, and the maximum number of input patterns they can learn to memorise using the precise timings of individual spikes as an indication of their storage capacity. In comparison with existing work, we determine the performance of the FILT rule to be consistent with that of the highly efficient E-learning Chronotron rule, but with the distinct advantage that our FILT rule is also implementable as an online method for increased biological realism.

CDP to which Component contributes (if relevant):

Progress: Partner SURREY made excellent progress on this Component, and a first paper on the rules has been published. Refer to Annex of Deliverable for details

Links: http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0161335

Quality Control:

- Upstream - Plasticity models: SP 4 (theory) T. 4.3.1 synaptic plasticity and learning [important]: Discussion with W. Gerstner and W. Senn underway to incoporate more biological detail.

Downstream:

- Downstream - WP9.2 BrainScaleS standalone next generation single chip physical model system [important]: Work is under way to implement the INST/FILT rule on the PPU (new in SGA1). A NEST implementation under “PPU-like” conditions has been tested.
- Downstream - CDP5-P3: Guiding Platform design on functional plasticity [important]: Work is underway to implement this rule on the way together with WP9.3 (Spinnaker) to implement on Spinnaker, however Spinnaker is not yet ready to accept this Component. We aim to remedy this with a jointly supervised SP4/SP9 postdoc at the EITN to start from April 2017.
- Downstream - CDP5-P4: Concept showcases in big-systems [essential]: pending on previously mentioned CDP5-P3.
- Downstream - WP9.3 SP9 SpiNNaker software stack [important]: inclusion of INST/FILT into software stack by joint EITN postdoc from April 2017.

17.3.2 Plasticity: Two-compartment neuron

Description of Component (from PLA):

CDP to which Component contributes: CDP5- Plasticity, Learning and Development: modelling the Dynamic brain , CDP5-P3: Guiding Platform design on functional plasticity & CDP5-P4: Concept showcases in big-systems.

In light of the above note on change of objective, this Component focuses on the following:

This Component specifically seeks to analyse and extend existing two compartment neuron models on the neuromorphic Platforms. It utilises an abstract predictive learning scheme

based on the detailed dendritic morphology of layer 5 pyramidal neurons that, ultimately, leads to cortical functions such as stimulus classification, pattern completion and motor control. Predictive learning has recently been formulated in the context of a dendritic prediction of somatic firing. Goal-orientated learning, on to other hand, is an upcoming topic for the biologically detailed hypercolumn model, as well as for the Neuromorphic Computing Platform. SURREY (P102) concentrates on the neuromorphic implementations and learning showcases as required for CDP5.

Progress: A joint SP4/SP9 EITN postdoc from April 2017 has been advertised and selected to take forward the implementation of this model on the Spinnaker Platform. Implementation details for the Physical Model Platform are being discussed and depend on the newly introduced PPU – which however it first tested on the easier to adapt INST/FILT rule.

You can refer for Annex of Deliverable for details

Links: N/A.

Quality Control:

- Upstream - Task 4.3.2 and 4.3.1 and their respective Components to provide biologically plausible learning rules.
- Downstream - 1348: STDP for imitation learning (Algo STDPimitation) [important] reported below.
- Downstream - CDP5-P3: Guiding Platform design on functional plasticity [important] Joint SP4/SP9 EITN postdoc to take forward on Spinnaker WP9.3 (Furber, Lester). Exploratory work on NM-PM/PPU (Schemmel, Hartel, Meier)
- Downstream - CDP5-P4: Concept showcases in big-systems [essential] pending on CPD5-P3.
- Downstream - T3.1.4 Information Theoretic Network Model of Layer 5 Pyramidal Cells [added value] Understanding of behaviour in larger scale simulations and effect of compartments on mean field behaviour (exchanges with M de Kamps)
- Downstream - T4.2.2 Astrocyte-Neuron interaction NETWORK model (ANN model) [essential]. Intenstive discussion with ML Linne re widening of model compartments to be able to cater for modulation by glia and analysis of network-level mechanisms constraining the *in vivo* implementation of learning rules and implementing integration, encoding and recall of multisensory memories [important]

17.3.3 Plasticity: STDP for imitation learning (Algo STDPimitation)

Description of Component (from PLA): In this Component we develop a model of song bird learning based on experimental data provided by external collaborator R Hahnloser at ETH and Uni Zurich. We apply the two-compartment neuron to a behavioural imitation learning task.

CDP to which Component contributes: CDP5- Plasticity, Learning and Development: modelling the Dynamic brain, CDP5-P3: Guiding Platform design on functional plasticity & CDP5-P4: Concept showcases in big-systems , as first show cases of the Platforms capabilities.

Progress: We implemented a stand-alone simulation with the view of implementing on the neuromorphic hardware as a showcase pending further progress with Components 1302 and 969. The progress in this control period up to date included progress on the theoretical understanding of songbird song learning and a recast of the underlying model.

18. T4.4.1 - Models of spontaneous brain activity

18.1 Key Personnel

Task Leader: Gustavo DECO (UPF, P77)

Other Researcher: Alain DESTEXHE (CNRS, P10) (Leader researcher from CNRS partner)

Other Researcher: Gorka ZAMORA-LÓPEZ (UPF, P77)

Other Researcher: Nikos KOUVARIS (UPF, P77)

18.2 DoA Goal(s)

To create models of the spontaneous activity of the brain and compare them to empirical recordings from humans. The models will be compatible with SP9 hardware, so that they can be simulated on the Neuromorphic Computing Platform. On a larger scale level of neural populations, we will use whole- brain models (spiking, mean-field, etc.) to obtain a large-scale model that includes multiple areas of the human cerebral cortex. This model will be directly constrained by anatomical measurements (DTI), and its spatiotemporal patterns of spontaneous activity will be compared to resting-state human brain recordings (fMRI). The models of spontaneous activity developed in T4.4.1 are mostly from human intracranial recordings. Within the framework of CDP1, these models will be extended to simulate the spontaneous activity and brain states in mouse, as recorded in SP1 using calcium imaging and single-cell mapping. These data will be used to constrain the models and obtain network models that fully reproduce the main features determined experimentally. The models produced will be fully compatible with neuormorphic hardware. This work will be performed with the help of one postdoc shared between SP4 and SP1, and will be carried out during the second year of SGA1.

Changes to DoA Goal(s): No change has been foreseen for the moment. As can be seen in the description of the Components, the goals of the Task have only been refined and broken into different compartments. The goal is not to create "just a model" of spontaneous brain activity but create models that reproduce empirical observations and we understand why. The models are complex and composed of several parts. Our aim is to understand how each part, e.g., the quality of DTI data necessary to constraint the models, affect the outcome of the model and support their realism.

18.3 Components Progress

PLA Components:

1069. Influence of topological heterogeneities on network activity – owner: Gorka Zamora-Lopez, type report (DoA Goal 1).

1070. Collective behaviour of mean-field and neural population models: A comparative study – owner: Gorka Zamora-Lopez, type report (DoA Goal 1).

1068. Investigation and correction of link weights in human structural connectomes via effective connectivity. – owner: Gorka Zamora-Lopez, type report (DoA Goal 1).

999. Macroscopic model of spontaneous human brain activity - owner: Gustavo DECO, type: model (DoA Goal 1).

1067. Python software to simulate spontaneous brain activity - owner: Gustavo DECO, type software (DoA Goal 1).

1206. Prototype software to estimate effective connectivity owner: Gustavo DECO, type software (DoA Goal 1).

1205. Effective connectivity changes inferred from optogenetic brain interrogation and calcium imaging – owner: Gustavo DECO, type report (DoA Goal 1).

1235. Local-network model of spontaneous activity in cortex – owner: Alain DESTEXHE, type model (DoA Goal 1).

CDP Contributions:

- CDP1 - Development of Whole Mouse Brain Model and Mouse Brain Atlas

  Component 1205 contribute to CDP1-P1: Reference set-up of the experiment

  Component 1070 contributes to CDP1-P3: A virtual imaging lab app

- CDP3 - Multi-level Human Brain Atlas

  Component 1068 contributes to CDP3-P8 Modelling and model validation using human quantitative data

- CDP4 - Visuo-Motor Integration (Component 1070 contributes to CDP4)

18.3.1 Investigation and correction of link weights in human structural connectomes via effective connectivity

The progress report on this Component currently remains confidential, as the data are currently proprietary and not yet in the public domain.

18.3.2 Influence of topological heterogeneities on network activity

The progress report on this Component currently remains confidential, as the data are currently proprietary and not yet in the public domain.

18.3.3 Collective behaviour of mean-field and neural population models: A comparative study

The progress report on this Component currently remains confidential, as the data are currently proprietary and not yet in the public domain.

18.3.4 Macroscopic model of spontaneous human brain activity

We construct a model to simulate whole brain activity at rest, at the scale of interconnected brain regions. The model is constrained using an empirically determined structural connectivity matrix (the network) via imaging and tractography. Local activity of the brain regions will be represented by models to simulate the activity of one brain region or neural population. There are many such population models available but each of these choices may lead to a different outcome of the network model. That is, the brain activity simulated by the model depends on how the model is built. Comparison of the model outcome to empirically observed resting-state fMRI will be crucial to determine the correctness of the choices taken to build the model.

CDP to which Component contributes (if relevant):

Progress: Over the recent years the efforts to describe the resting-state brain activity have focused on the representation of resting-state over its time-averaged functional connectivity. The "resting-state networks" widely discussed in the literature are an example of these efforts. However, spontaneous brain activity is also characterised by notable spatio-temporal fluctuations. In order to reproduce these fluctuations, we have introduced a new category of mesoscopic models to simulate the local brain region dynamics, the normal form of a Hopf bifurcation. With this novel approach, we reveal that the human brain during resting state operates at maximum metastability, i.e. in a state of maximum network switching.

We have applied this novel approach to model task-dependent brain activity, finding that structural rich-club brain regions exhibit oscillations during task but not during rest. Rich-

club hubs can harmonise a set of asynchronous brain regions, supporting functional coupling among them.

Links:

– Introduction of a novel category of mesoscopic models for dynamics resting-state activity: http://biorxiv.org/content/early/2016/12/21/065284

- Application of the novel framework to task-related brain activity: http://www.sciencedirect.com/science/article/pii/S1053811916306000

18.3.5 Python software to simulate spontaneous brain activity.

This is the code we develop to run simulations of the model " A model of spontaneous (human) brain activity"

CDP to which Component contributes (if relevant): None.

Progress: The Component is in **intermediate stage**. We have written the code to simulate macroscopic models of spontaneous human brain activity and we have tested different performance optimization strategies available in the Python programming "universe". These include both the vectorization of the multidimensional problem using the array structure provided by the NumPy library and the use of Numba, an optimization compiler which translates Python code into LLVM "just in time". Our preliminary observations show that code running with the Numba implementation is in most situations significantly faster than the code using NumPy-based vectorization, although its portability to high-performing computing remains to be tested. The vectorised code is, in this sense, more flexible.

Links: None

18.3.6 Prototype software to estimate effective connectivity

A method, developed during the Ramp-Up Phase, that uses structural and functional connectivities to estimate effective connectivity (most probable structural weights giving rise to observed functional connectivity), is planned to be used in a software during SGA-1.

CDP to which Component contributes (if relevant): CDP-3, Multilevel brain atlas. CDP3-P8 Modelling and model validation using human quantitative data.

Progress: The status of the Component is **advanced**. Following the development of a novel method to estimate effective connectivity during the Rump-Up Phase, we have devoted the past year to apply the method in practical examples and to create a public version of the software. The software to estimate effective connectivity was publicly released in November 2016 by making the code available in GitHub. Current version has been written in Python 2.7 and requires additional libraries NumPy, SciPy and Matplotlib (otional). In the current version, the model and requires (i) a structural connectome and (ii) BOLD time-series from functional MRI as input from the user. The software release includes example datasets which we used for two publications, see links below.

During second year of SGA1 (latest during SGA2) we have planned to integrate the software into the HBP Collaboratory with help from SP5 as a collaboration within CDP3. Future plans are to allow the method to work with EEG data and with wide-field calcium imaging.

Links:

– Public release of the software to estimate effective connectivity in GitHub: https://github.com/MatthieuGilson/EC_estimation

– Original peer-reviewed publication of the method, with explanation of the datasets: dx.doi.org/10.1371/journal.pcbi.1004762

– Manuscript showing practical application of the method during movie viewing: http://biorxiv.org/content/early/2017/02/20/110015

18.3.7 Local-network model of spontaneous activity in cortex

Description of Component (from PLA): Biophysically plausible models of spontaneous activity states in excitatory-inhibitory networks, including asynchronous states, and sleep slow-waves (previous Component name in RUP: Model of spontaneous network activity in cortex).

SGA2: continuation of a SGA1 model of spontaneous activity and slow-waves by AdEx networks.

Progress: We have a network model with RS and FS cells, displaying asynchronous states with the correct conductance level as measured *in vivo*. We are now investigating the properties of these network states, and how they can produce slow oscillations with Up and Down states.

19. T4.4.2 - Models of low-level vision

19.1 Key Personnel

Task Leader: Olivier MARRE (UPMC, P105)

Other Researcher: Shimon ULLMAN (WEIZMANN, P84)

Other Researcher: Ulisse FERRARI (UPMC, P105)

19.2 DoA Goal(s)

1. To develop models of the retina responding to complex stimuli that can be used as an input for models of the visual cortex.

Changes to DoA Goal(s): None.

Component Progress

PLA Components:

The name of the Component has been slightly modified to better fit the objectives (preivous name was *Network model of the retina responding to complex stimuli*).

981. Model of the retina responding to complex stimuli – owner: Olivier Marre, type: model (DoA Goal 1)

CDP contributions: None.

Model of the retina responding to complex stimuli

Description of Component: This model aims to do quantitative predictions of how cells /populations of cells are responding to complex stimuli.

CDP to which Component contributes: Not relevant

Progress: There was no planned release at that stage.

Links: Include links to relevant material available publicly online.

Quality Control:

- No Upstream Component
- Downstream Laminart with segmentation and retina into the NRP , Michael Herzog: intermediate release of information: we gave them access to our most recent reports.
- Downstream NRP - Sensor model library, Stefan Ulbrich (Dillman Rüdiger): intermediate release of information: we gave them access to our most recent reports.

19.2.1 Model for high-level contributions to low-level vision of the retina responding to complex stimuli

Description of Component (from PLA): This in an algorithm for using information from higher-level visual areas in the processing of information in low level areas such as primary visual cortex. It will also lay the ground for a later development of a network model.

CDP to which Component contributes: Not relevant

Progress: There was no planned release at that stage.

Links: Include links to relevant material available publicly online.

Quality Control:

- Upstream - added value - Plasticity models: SP 4 (theory) T. 4.3.1 synaptic plasticity and learning
- Upstream- added value - Laminart with segmentation and retina into the NRP
- Downstream - added value - Laminart with segmentation and retina into the NRP

20. T4.4.3 - Models of motor control

20.1 Key Personnel

Task Leader: Jeanette HELLGREN KOTALESKI (KTH, P39)

Other Researcher: Jovana BELIC (KTH, P39); Mikael Lindahl (KTH, P39)

20.2 DoA Goals

1. This Task will develop models of the basal ganglia system using mainly data from the literature, but also data produced in SP1-3 via NIP can be used. The model will be challenged in neurorobotics experiments (SP10) and also be used to interpret PD data together with SP8. The action selection capabilities of the model system will be compared with previously developed basal ganglia models used for studying such functions. In addition, strategies will be developed for classifying cellular and network changes correlated with disease as compensatory vs symptom causing factors.

Changes to DoA Goal(s): No significant changes in plans are foreseen.

20.3 Components Progress

PLA Components:

1025. Motor control model – Owner: Jeanette Hellgren Kotaleski, type model (DoA x).

The model built in this task will mainly build on literature data, but in addition data from SP1 (T1.2.3) will be used when released. In SP6, a data-driven microcircuit level model of the basal ganglia is foreseen (T6.2.5), thus insights derived from that work will feed into the current model as well. Dopamine is important both for controlling membrane excitability and synaptic effects in the basal ganglia, and also for reward dependent learning. Several SP6 Components relating to dopamine signalling are directly useful for the current task (e.g. the Components 'Dopamine receptor induced signalling in striatum ' as well as 'SP6-T6.1.2-SGA1-Subcellular Model of Timing Dependent Reward/Dopamine Plasticity '. As the model is simulated using Nest it also uses Components from work done in SP6, especially the Component called 'Nest-the Neural Simulation tool' (T6.3.5).

CDP Contributions: None for the moment. As basal ganglia are important in motor control, the model built in T4.4.3 will be possible to use in CDP4 'Visuo-motor integration' in later

SGAs. In addition, plasticity rules used in CDP5 can be plugged in and tested in the model we are building currently.

20.3.1 Motor control model

Description of Component: One important aspect of motor control is to select which actions to perform in a certain situation and recruit the appropriate motor program. The basal ganglia are crucial for this and their function is disturbed in Parkinson's Disease (PD), where neurons exhibit inappropriate synchronisation and oscillations. This Task will develop models of the basal ganglia system using data from SPs 1-3 and SP5, as well as the literature. The model will be challenged in neurorobotics experiments (SP10) and also used to interpret PD data together with SP8. The action selection capabilities of the model system will be compared with previously developed basal ganglia models used for studying such functions. In addition, strategies will be developed for classifying cellular and network changes correlated with disease as compensatory vs symptom causing factors.

Links: A first draft point neuron model of the basal ganglia system is published:

Lindahl M, Hellgren Kotaleski J. Untangling Basal Ganglia Network Dynamics and Function: Role of Dopamine Depletion and Inhibition Investigated in a Spiking Network Model, eNeuro. 2017 Jan 12;3(6). pii: ENEURO.0156-16.2016. doi: 10.1523/ENEURO.0156-16.2016. PMID: 28101525;

Model can be found at github https://github.com/mickelindahl/bgmodel

The basal ganglia model consists of 80000 point neurons. The model was used to test the role of parameter changes associated with dopamine depletion. Multiple changes of parameters for synaptic efficacy and neural excitability that could improve action selection ability and at the same time reduce systems level oscillations following dopamine depletion were identified. This study increases our understanding of the relation between network dynamics and network function in health and disease.

Quality Control:

- Component released 'Motor control model'; received released Components called 'Dopamine receptor induced signaling in striatum' from RUP and Component' called SP6-T6.1.2-SGA1-Subcellular Model of Timing Dependent Reward/Dopamine Plasticity (from the SGA1 phase)

List of PLA dependencies:

- Upstream SP6-T6.2.5-SGA1-Models of basal ganglia nuclei
- Upstream SP6-T6.1.2-SGA1-Subcellular Model of Timing Dependent Reward/Dopamine Plasticity (
- Upstream Cellular properties of neurons within striatum
- Upstream Connectivity and morphology of neurons within striatum
- Upstream SP2 - Selected multimodal regional maps with cognitive features
- Downstream SP3-Shrewbot++ robot Platform
- Downstream Elephant
- Downstream NEST - The Neural Simulation Tool
- Downstream NEST code with abstracted neuron model representations

21. T4.4.4 - Models of spatial navigation

21.1 Key Personnel

Task Leader: Neil BURGESS (UCL, P82)

21.2 DoA Goals

1. To implement a neural-level model of spatial navigation, explore its use for episodic memory and planning, and help to implement on robots and neuromorphic computing hardware.

Changes to the DoA Goals: No changes to these goals are planned.

21.3 Components Progress

PLA Components:

This task comprises a single PLA model Component:

984. Hippocampal and striatal model of spatial navigation, with extension to planning and episodic memory - owner: Neil Burgess, type: model (DoA Goal 1). To be used to investigate the different learning rules and neural representations involved in spatial navigation, and how they combine to guide action. The output is behaviour in classic navigation tasks used in rodents, neural firing characteristics, effects of lesions and behaviour are compared with data from the literature. Future work will investigate how this model can be extended to episodic memory and planning.

We have begun conversation with PLA Components from SP3: Rodent physiology: pattern completion in episodic memory; Shrewbot++ robot Platform. There will be mutual added value in these exchanges.

This is not a Component for a Co-Design Project.

CDP contributions: None.

21.3.1 Hippocampal and striatal model of spatial navigation, with extension to planning and episodic memory

Description of Component (from PLA): A network model of firing-rate coded neurons in hippocampus and striatum which performs spatial navigation. This model is used to investigate the different learning rules (temporal difference in striatum, incidental in hippocampus) and representations (e.g. sensory/action in striatum, and place cells etc in hippocampus) involved in spatial navigation, and how they combine to guide action. The output is behaviour in classic navigation tasks used in rodents, neural firing characteristics, effects of lesions and behaviour are compared with data from the literature. Future work will investigate how this model, constrained by plentiful data on navigation, can be extended to episodic memory and planning.

CDP to which Component contributes: N/A

Progress: Progress on this Component has been delayed by recruitment issues arising from the late delivery of money to UCL for SGA1 (end of September 2016). Progress since recruitment of Dr Andrej Bicanski (starting 1st Jan 2017) has begun in the finalising a rate-coded model of the hippocampal and striatal contributions to spatial navigation following work in the Ramp-Up Phase. He has also begun considering the model's extension to episodic memory and (general) non-spatial planning. In addition, initial steps have been made in implementation on the neuromorphic Platform SpiNNaker. Additional researcher will be recruited to provide the missing 9 person months of work needed to ensure delivery of this task's Milestone at M24.

Quality Control:

- Upstream Component: (Francesca Cacucci, SP3 Episense T3.3.2, SGA1) Rodent physiology: pattern completion in episodic memory. We have held discussions concerning the comparison of model and data before M24. Both Projects suffered from delayed recruitment due to arrival of money only in September 2016.
- Downstream Component: (Tony Prescott, SP3 Episense T3.3.4, SGA1) Computational modelling of multisensory episodic memory. We have held discussions and shared code from a previous model (Byrne, Becker, Burgess, 2007) to aid their development of a model of episodic memory.
- Downstream Component: (Martin Pearson, SP3 Episense T3.3.5, SGA1) Robotic systems: hardware implementation of multisensory episodic memory. We have held discussions and shared code from a previous model (Byrne, Becker, Burgess, 2007) regarding the implementation of models of spatial and episodic memory on SpiNNaker neuromorphic Platform and on their Shrewbot++ robotic systems

22. T4.4.5 - Development of a large-scale, mean field model on sensorimotor integration

22.1 Key Personnel

Task Leader: Gustavo DECO (UPF, P77)

Other Researcher: Nikos KOUVARIS (UPF, P77)

CDP4 Researcher: Rainer GOEBEL (Leading researcher from UM, P117)

CDP4 Researcher: Mario SENDEN (UM, P117)

22.2 DoA goal(s)

1. This Task contributes to CDP4, supporting the development of a mean-field model of sensorimotor integration. The developed simplified mean field model provides insights in the computational mechanisms underlying sensorimotor operations preparing the integration in the NEST Platform. The model will be developed in collaboration with Task 4.4.2, and the required conceptual models of object perception and attention-for-action will be provided by SP3 (cross-cutting Project ContextDeepNet) and SP2 (WP2.4).

Changes to DoA goals: No change has been foreseen for the moment.

22.3 Components Progress

PLA Components:

Elaboration of Components undergoing during M1-M6, should be finallised during M6-M12

Task T4.4.5 owns or participates in the following Components:

1070. Collective behaviour of mean-field and neural population models: A comparative study – owner: Gorka Zamora-Lopez, type: report (DoA Goal 1).

1207. Large-scale model of visuo-motor integration – owner: Gustavo DECO, type: model (DoA Goal 1).

CDP Contributions:

- **CDP4- Visuo-Motor Integration**

Task 4.4.5 is fully embedded as a support task for CDP4.

- **CDP1- Development of Whole Mouse Brain Model and Related Mouse Brain Atlas**

Component 1070 contributes to CDP1-P3: A virtual imaging lab app.

22.3.1 Collective behaviour of mean-field and neural population models: A comparative study

Description of Component: There exist many population models to simulate the activity of one brain region. The goal of this Component is to understand how those models behave, collectively, when simulating a network of interacting brain areas.

CDP to which Component contributes (if relevant): CDP4 Visuo-motor integration

Progress: Status of the Component is in **initial stage**. During the first 12 months we have gathered some evidence of how different local models to simulate the local dynamics of the nodes in a network alter the global network dynamics. However, those observations have been performed in generic oscillatory models and in models of spiking neurons, instead of mean-field and population models. The results of this Component are critical to build the large-scale network models of the resting-state and it will be given priority in the period M12-M24 after the work from the Components in advanced state is finalised.

Links: None.

Quality Control:

- Upstream: Mean-field models of interacting populations of rate and spiking neurons (Olivier Faugeras, INRIA). We will establish collaboration with the INRIA partner during M12-M24 once we start actively developing the Component.

- Downstream: Macroscopic model of spontaneous human brain activity (Gustavo DECO).

Development of a large-scale, mean field model on sensorimotor integration

The "Large-scale model of visuo-motor integration" is developped as part of the CDP4 at the UPF (Spain) in Gustavo Deco's lab. Rainer Göbel (CDP leader) and Mario Senden are collaborating to this Project.

CDP to which Component contributes (if relevant): CDP4, Visuo-motor integration

Progress: The status of the Component is in **indermediate stage**. A rate neuron model for the saccade generator in the reticular formation has been implemented (UM-P117). Based on network interactions among reticular formation neurons, the implemented model reproduces typical neuronal activation profiles observed during saccade generation, realistic saccade trajectories and cell tuning properties. A number of rate neuron has already been implemented in the NEST Platform. This model forms the back-end of a larger architecture of visuo-motor integration, transforming eye-displacement vectors into motor commands for the eye muscles. At the next stage, a mean-field description will also be developed (UPF, P77) and implemented for the NEST.

Furthermore, it has been developed (UM-P117) a deep convolutional autoencoder network able to learn a mapping from natural images to topological saliency distributions. The performance of this network is good and can predict salience distribution given previously unseen natural images. This model forms the front-end of a larger architecture of visuo-motor integration, providing salience distributions as input to a target selection process.

Links: None.

23. T4.5.1 - Comparing models with mouse and human brains

23.1 Key Personnel

Task Leader: Sonja GRÜN (JUELICH, P20)

Other Researcher: Viktor JIRSA (AMU, P78)

23.2 DoA Goal(s):

1. This Task will develop strategies, principles, and algorithms allowing comparative assessment of experimental data and different model approaches at different scales and description levels. It will analyse, compare and contribute to improve model modules of visuo-motor integration, which require the interaction of different cortical brain areas and subcortical structures.
2. The Task will compare local network processes and dynamic interactions in simplified models building on point neurons and population models, with biophysically detailed micro- and meso-circuit models. Massively parallel single unit and LFP data resulting from large-scale spiking network simulations and experimental data will be analysed to validate and iteratively improve the models.
3. Whole brain network models derived from human and mouse connectome data using neural mass models will also be validated against empirical data.

Changes to DoA Goal(s): None.

23.3 Components Progress

PLA Components:

The OdML Component has been transferred to SP5.

812. Workflow for comparison of electrophysiological and simulated data - Owner: Sonja Grün, type: service (DoA Goal 2).

418. Massively Parallel Electrophysiology data - owner Sonja Grün, type: data (DoA Goal 2).

1574. Structural and functional connectivity at different scales – owner: Viktor Jirsa, type: model (DoA Goal 3)

1008: Multi compartmental reconstructed cortical cells: their input-output transfer properties – owner Giugliano, type Model reported in T4.1.2

CDP Contributions: CDP4

Component 418 contributes to the Use Case comparative analysis of experimental and simulated data.

23.3.1 Workflow for comparison of electrophysiological and simulated data

Description of Component: Example workflow for comparison of experimental and simulational spiking neuron data by correlation analysis.

CDP to which Component contributes: CDP4, Visuo-Motor Integration

Progress: The Component is in an early stage. We have outlined general requirements for a reproducible workflow for the comparison of experimental and modelled data in Denker and Grün (2016). We work closely with T9.1.5 and our work is based on the guideline comparison 'NEST SpiNNaker Elephant Demo' (https://project-

lifecycle.herokuapp.com/Component/886/) available in the Collaboratory. The comparison of experimental and modelled data is currently developed outside the collaboratory and will be integrated in a later stage of SGA1.

The comparison of experimental and modelled data is carried out for a system in a 'resting' state. On the experimental side we use Utah array recordings from the motor/premotor cortex of an awake non-human primate not performing any task. Spiking activity and local field potentials were measured for 15 min and the data was separated into periods of rest and movement. We separated the single units into excitatory and inhibitory neurons based on their respective waveforms and calculated the single neurons' firing statistics (firing rate, coefficient of variation) and network interaction measures (pairwise correlation coefficients). This was done for each rest and movement periods and the corresponding results were compared, showing that inhibitory neurons are more strongly affected by the motor state. The associated workflow and analyses, 'Analysis of single unit activity during rest and movement', were made available in the Collaboratory (collab.humanbrainproject.eu/#/collab/2493).

Links:'NEST SpiNNaker Elephant Demo' (https://collab.humanbrainproject.eu/#/collab/507), 'Analysis of single unit activity during rest and movement' (https://collab.humanbrainproject.eu/#/collab/2493)

Quality Control:

Upstream Components:

- 'Massively Parallel Electrophysiology data' (owner: Sonja Gruen, T4.5.1) [essential]: data are available, not yet integrated into the NAR (T5.7.2), since the NAR is not in place yet
- SGA1 - Federated data storage with flexible permission management and remote access [essential]: was newly defined as 'Neural Activity Resource' (T5.7.2) after the DPIT process, and is not yet in place
- odML [important]: is moved to SP5; not yet needed
- UNICORE [important]: part of SP7; is in place and was used https://collab.humanbrainproject.eu/#/collab/507/nav/6326; not yet needed here
- PyCOMPSs Repository [added value]: is partly in place; not yet integrated
- Collaboratory Task Service [important]: is in place; currently not used
- Collaboratory Provenance Service [added value]: to my knowledge not in place, will be important for daily work
- Collaboratory Storage Service [essential]: is in place, but on the long run should be replaced by HPC storage; not yet needed
- Collaboratory Jupyter Notebook [essential]: in place, in use
- HPC systems at JSC [essential]: in place, explored together with different other tasks
- NEST - The Neural Simulation Tool [essential]: NEST: has been used to generate input to but not yet as integral part of the workflow
- 4x4 mm2 motor cortex model [essential]: first version available, in use
- Elephant [essential]: available, in use

Downstream Components:

- SP4 -SGA2 - Integrative Loop for Comparison of Experimental and Simulated Data using the Validation Framework [important] – is an SGA2 Component, not yet available

- SP6-T6.4.4-SGA1-Validation result service [important]: will be used

23.3.2 Massively parallel electrophysiological data

Description of Component: Extracellular electrophysiological recordings (100 electrode Utah array) from non-human primate from motor and premotor cortex during resting state. Further data sets will be integrated from recordings with the same technique and area but during performance of a reach-2-grasp task (Riehle A, Wirtssohn S, Grün S and Brochier T (2013) Mapping the spatio-temporal structure of motor cortical LFP and spiking activities during reach-to-grasp movements Front. Neural Circuits 7:48. DOI: 10.3389/fncir.2013.00048), plus full metadata annotation (in odML) and basic python loading routines and simple analysis scripts.

CDP to which Component contributes (if relevant): CDP4, Visuo-Motor Integration

Progress: Utah array recordings (100 electrodes) from the motor/premotor cortex of an awake non-human primate not performing any task were provided by the 'Institut de Neurosciences de la Timone' in Marseille. The data have been recorded beforehand but were prepared and post-processed for our use. Spiking activity, waveforms and local field potentials were measured for 15 min and the monkey was recorded on video in order to separate the data into periods of quietly sitting (rest) and periods that involved limb or head movements. Spikes were already sorted offline so that single unit activities are available.

More resting state recordings with other monkeys are planned and will be available in the late phase of SGA1 and in SGA2.

Quality Control:

Upstream Components:

- Neural Activity Resource Development (API, WebApp, MetaData DB) [added value]: implementation started only recently (T5.7.2) after the DPIT process, and is not yet in place; data will be integrated there
- Compute and data resource co-allocation [important]: unknown
- SP7 Federated HPAC Computing Services [important]: storage service will be needed, but currently not yet used
- Neo [important]: exploratory version available, in use
- odML [essential]: exploratory version available, in use

Downstream Components:

- Elephant [important] (T5.7.1): available, in use
- Workflow for comparison of electrophysiological and simulated data [essential] (T4.5.1): offline available, in use

23.3.3 Structural and functional connectivity at different scales – owner: Viktor Jirsa

Description of Component: Structural connectome (SC) constrains the functional connectivity (FC) in human and in mouse brain. For human and for individualised mouse brain the SC is extracted from diffusion MRI data, while axonal-projections tracing is used for the detailed connectome of the mouse brain from the Allen Institute. We will enable consistent representation at different scales of the human and mouse structural connectivity data from different modalities. Functional connectivity from experimental mouse imaging data will be extracted and represented in a similar manner. The structural data is then used for building macro and mesoscopic model of the brain activity, that can be compared with empirical FC in rest or in pathological conditions, such as epileptic seizure.

CDP to which Component contributes: it should contribute more in the next phase with the application of the same strategy as for human modelling on the mouse) Development of Whole Mouse Brain Model and Mouse Brain Atlas (CDP1), we are responsible for the P3 Virtual imaging lab app

Progress:

We have reconstructed epileptic patients' brain networks, using their specific anatomical properties and used the Epileptor, a neural mass model capturing the temporal evolution of a seizure including on- and offset. Based on clinical hypothesis of EZ, we reproduced seizure propagation *in silico* as observed empirically with SEEG electrodes implanted in the patient. These propagation patterns cannot be captured by functional connectivity due to their inherent non-stationarity. We made the following steps: 1) Proix et al (2016) built the processing chain of all structural images (dMRI, MRI) required to build a virtual brain. 2) Jirsa et al (2016) demonstrated the methodology and proof of concept of how to create personalised models and fit them against empirical neuroimaging data. 3) Proix et al (2017) performed a pilot study (N=15), where we computed a score estimating the difference between the EZ identified by the brain model, and that identified by the clinicians during pre-surgical evaluation. This study demonstrated the favourable correlation between model prediction and surgery outcome. In other words: negative surgery outcome correlates with surgery not performed in line with model predictions.

Modelling can be performed in the Collaboratory. Same strategies can be used for human and for mice, meaning that users can obtain virtual imaging data from selected brain regions, described either as standard atlas partitions (the entire cortex, M1 area, etc.) or in geometrical terms (e.g. arbitrary cut slice). The user can specify the portion of the brain to simulate (e.g. whole brain or a single slice – missing at least part of long range projections), the model to be used (e.g. high-dimensional, spiking point neurons, population level), physiological/pathological conditions, the type of imaging (calcium, VSD, fMRI, PET, electrophysiology), the details of imaging system (resolution, acquisition speed, field of view, spatial orientation effects).

Hired two postdoc for this Project: Dr. Spase Petkoski and Dr. Andreas Spiegler, from 1.4.2016.

Quality Control:

Upstream Components

- SOFTWARE > Algorithm Library > Brain Anatomy [added value]; Not yet implemented (we believe that this platform does not exist and hasn't been needed yet, but it should become relevant in the next phase)
- Model of biologically-realistic network states [added value]; It is not yet needed, we expect it to be implemented in the next phase.
- Neuro-glial model for bursting activity [important]; It is not yet needed, we expect it in the next phase.
- Model of calcium imaging signals [important]; It is not yet needed, we expect it in the next phase.
- Fluorescence imaging of cortical activity after stroke [important]; We have received and started analysing the experimental data and comparing with the model, and these were provided back to the experimentalist. More is expected in the next phase.
- Allen mouse brain reference atlas with white matter structures parcellated [essential]; Actually this Component is more related to the Component 998, Allen Mouse Atlas (AMA) based brain network. It will be used in the next phase when the accent will be on the *modelling* and validation of mouse using the same strategy as for human. It should help

to obtain the 2D mapping from the Allen's structural data, for better correspondence with the calcium imaging

- -Allen Mouse Atlas (AMA) based brain network [essential] (doesn't exist but maybe to be added); The Component has provided the means for performing the same modelling for the mice as for human.

Downstream Components:

- SOFTWARE > Data Factory > Data Storage [essential]; Not yet implemented (we believe that this platform does not exist and hasn't been needed yet, but it should become relevant in the next phase)
- Effective connectivity changes inferred from optogenetic brain interrogation and calcium imaging [added value]: The Component not yet active, we expect to connect with it in the next phase.
- Allen Mouse Atlas (AMA) based brain network; The results from validation of the same strategies in the mouse brain modelling as used for human, have been returned. Will be important in the next phase.

24. T4.5.2 - Mouse brain function from structure

24.1 Key Personnel

Task Leader: Viktor JIRSA (AMU, P78)

Other Researcher: Gustavo DECO (UPF, P77)

Other Researcher: Spase PETKOSKI (AMU, P78), Andreas SPIEGLER (AMU, P78), Francesca MELOZZI (AMU, P78)

24.2 DoA Goal(s)

1. To integrate detailed structural data (connectivity, region mapping) of different origins (DTI, Allen Brain Atlas) in whole mouse brain network models. The network node models will be neural population models as developed in SP4 (Task 4.1.3). This Task will mathematically and computationally investigate the non-stationary properties and capacity of the models to propagate activations through the network. These reduced top-down models will be validated against high-dimensional neuronal network models, enabling parameter space explorations to guide high performance computations (SP7). Initial studies will model spontaneous resting state activity, whose understanding forms the basis for later extensions towards behavioural functions and pathologies (SP8).
2. Within the framework of CDP1, the Task will further validate the whole brain mouse network models against empirical data recorded in SP1 using calcium imaging and local field potentials. The top-down models will systematically exploit the effects of the structural connectivity constraints upon network dynamics, and will be compared with empirical cortical and whole-brain activation maps in SP1. These empirical data will be used to constrain the structure-function relationship. This work will be performed with the help of one Postdoc shared between SP4 and SP1.

Changes to DoA Goal(s): No modifications of goals.

24.3 Components Progress

PLA Components:

998. Allen Mouse Atlas (AMA) based brain network – owner: Viktor Jirsa, type: model (DoA Goal 1)

CDP Contributions:

- CDP1 - Development of Whole Mouse Brain Model and Mouse Brain Atlas

Component 998 contributes to CDP1-P2: A virtual anatomy lab app & CDP1-P4: A virtual behaviour lab app

24.3.1 Allen Mouse Atlas (AMA) based brain network

Description of Component: Connectome-based models of the human brain provide insightful information on the structure-function relationship in health and disease. The connectome can be based on diffusion MRI data, for individualised mouse brain modelling, or on the detailed connectome of the mouse brain from the Allen Institute for Brain Science. We will develop clear practical examples, such as the switching of functional connectivity during resting state in health and seizure propagation in epilepsy, which should accelerate our understanding of whole brain dynamics in normal and pathological conditions, as model predictions can be readily tested in the numerous available transgenic mouse lines.

CDP to which Component contributes: CDP1 - Development of Whole Mouse Brain Model and Mouse Brain Atlas / CDP1-P2: A virtual anatomy lab app + CDP1-P4: A virtual behaviour lab app.

Progress:

Partner AMU: We have implemented the open source tracer dataset of the Allen Institute (Oh et al., 2014) into The Virtual Brain (TVB) (Sanz-Leon et al. 2015), thus allowing detailed Structural Connectivity (SC) to be obtained (Melozzi et al 2017). This is then used to build large-scale brain network models for the resting state different modalities of Functional Connectivity, fMRI or calcium imaging.

The resolution of the long-range structural connectivity can be as small as 0.1 mm leading to maximum number of 540 nodes, with 88 belonging to the isocortex. In addition to this, we have also applied homogeneous local connectivity for the isocortex, thus increasing its spatial resolution to several thousand nodes.

Modelling and simulation can be performed in the same way as for the human connectome. Hence, users can obtain virtual imaging data from selected brain regions, described either as standard atlas partitions (the entire cortex, M1 area, etc.) or in geometrical terms (e.g. arbitrary cut slice). The user can specify the portion of the brain to simulate (e.g. whole brain or a single slice – missing at least part of long range projections), the model to be used (e.g. high-dimensional, spiking point neurons, population level), physiological/pathological conditions, the type of imaging (calcium, VSD, fMRI, PET, electrophysiology), the details of imaging system (resolution, acquisition speed, field of view, spatial orientation effects). This part has been put online in the Collaboratory.

For the network nodes we have investigated the dynamics using the reduced Wong Wang model in the bistable regime, as was shown (Hansen et al. 2015) to capture the FC dynamics in humans, but we have also applied different oscillatory models, i.e. Hopf and Kuramoto oscillators. This has allowed the Allen's data to be verified against resting state functional connectivity.

Different lesioning or resection (stroke) strategies and their influence to propagation of excitability are systematically analysed. We have demonstrated proof of concept for the resection of the structural connectivity, and the impact that it has on the observed functional connectivity.

In addition, resting state have been stimulated and propagation patterns (excitability) due to connectivity have been analysed for in line with the experimental data.

Hired two postdoc for this Project: Dr. Spase Petkoski and Dr. Andreas Spiegler, from 1.4.2016.

Contributions also from PhD student Francesca Melozzi.

Partner UPF: Within the CDP1, UPF partner has agreed to estimate the effective connectivity in mice before and after stroke (Effective connectivity changes inferred from optogenetic brain interrogation and calcium imaging). For that we need (i) the structural connectivity in mice (the Allen Atlas which is a Component of T4.5.2 and (ii) whole brain calcium imaging from italy. As a feedback to T4.5.2, we will return to T4.5.2 the estimated EC matrices for the mice, which may be used to constrain the models of the whole brain activity in mice.

Publications (in preprint or in preparation)

Melozzi F, Bergmann E, Kahn I, Jirsa VK, Bernard C. Individual predictability and comparison between different structural connectivities (in preparation)

Melozzi F, Woodman MM, Jirsa VK. Bernard C. The Virtual Mouse Brain: A computational neuroinformatics Platform to study whole mouse brain dynamics (submitted for publication, 2017)

Upstream Components

- SOFTWARE > Algorithm Library > Brain Anatomy [added value]; Not yet implemented (we believe that this platform does not exist and hasn't been needed yet, but it should become relevant in the next phase)
- Model of biologically-realistic network states [added value]; It is not yet needed/Data not yet available, we expect it in the next phase.
- Neuro-glial model for bursting activity [important]; It is not yet needed, we expect it in the next phase.
- Model of calcium imaging signals [important]; It is not yet needed, we expect it in the next phase.
- Hippocampus reconstruction [added value]; Not yet needed, probably it might be used in the next phase.
- Arc-dVenus half mouse brain [added value]; Not used and probably it won't be needed in the next stage either.
- Structural and functional connectivity at different scales [important]; Data from human modelling obtained and the same strategies compared and validated for the mouse.
- Fluorescence imaging of cortical activity after stroke [important]; Data has been received for several mice in resting state, before and after stroke (although only few are about the same subjects). Some of the data analysed and compared/validated with the model. More needs to be done in the next phase.
- Point-neuron model of the whole mouse brain [important]; It is not yet needed, we expect it in the next phase.

Downstream Components:

- SOFTWARE > Data Factory > Data Storage [essential]; Not yet implemented (we believe that this platform does not exist and hasn't been needed yet, but it should become relevant in the next phase)
- SOFTWARE > Algorithm Library > Statistical Analytics > Disease Signatures - concept and methodology [important]: Not yet implemented (we believe that this platform does not exist and hasn't been needed yet, but it should become relevant in the next phase).

- Effective connectivity changes inferred from optogenetic brain interrogation and calcium imaging [added value]: The Component not yet active, we expect to connect with it in the next phase.
- Analysis of meso-scale fluorescence functional data; We have started analysing the experimental data and comparing with the model, and these were provided back to the experimentalist. More is expected in the next phase.

25. T4.7.1 - Scientific coordination

25.1 Key Personnel

Task Leader: Alain DESTEXHE (CNRS, P10)

25.2 DoA Goals

1. coordinate scientific activities within the SP
2. coordinate with other SPs, Partnering Projects and international collaborations;
3. perform quality assurance
4. organise meetings and workshops
5. coordinate reporting.

Changes to DoA Goals: None

25.3 Components Progress

PLA Components:

1016. SP4 scientific Coordination and Management - owner: Alain DESTEXHE, type: service (DoA Goal 1,2,3,4 & 5)

CDP Contribution: N/A

25.3.1 SP4_scientific Coordination and Management

Description of Component: This Task will coordinate scientific activities within the SP; coordinate with other SPs, Partnering Projects and international collaborations; perform quality assurance; organise meetings and workshops, and coordinate reporting.

CDP to which Component contributes (if relevant): N/A

26. Publications

26.1 PREPRINTS:

Gustavo Deco, UPF:

R. G. Bettinardi, G. Deco (UPF), V. M. Karlaftis, T. J. Van Hartevelt, H. M. Fernandes, Z. Kourtzi, M. L. Kringelbach & G. Zamora-Lopez (UPF) **How structure sculpts function: unveiling the contribution of anatomical connectivity to the brain's spontaneous correlation structure**

https://arxiv.org/abs/1612.02243

Marc de Kamps, ULEEDS:

Lai Yi Ming & De Kamps Marc. *Population Density Equations for Stochastic Processes with Memory Kernels (*submitted to Phys Rev E, has passed the first round of review and was resubmitted) https://arxiv.org/abs/1601.07126

Alain Destexhe, CNRS:

Girones Z. and Destexhe A. Enhanced responsiveness in asynchronous irregular neuronal networks. (submitted for publication, 2017) arXiv preprint: https://arxiv.org/abs/1611.09089

Telenczuk B., Kempter R., Curio G. and Destexhe A.. 2017. Encoding Variable Cortical States with Short-Term Spike Patterns. (submitted for publication 2017) BioRxiv preprint: 10.1101/098210.

Zerlaut Y. and Destexhe A. A mean-field model for conductance-based networks of adaptive exponential integrate-and-fire neurons. (submitted for publication, 2017) arXiv preprint:

https://arxiv.org/abs/1703.00698

Romain D. Cazé, Bartosz Telenczuk & Alain Destexhe **Computing threshold functions using dendrites** *ArXiv – NIPS*

https://arxiv.org/abs/1611.03321

Wulfram Gerstner, EPFL:

M. Faraji, K. Preuschoff and W. Gerstner (2017) Balancing New Against Old Information:The Role of Surprise arXiv:1606.05642

Related to **T4.3.2**

T. Schwalger, M. Deger and W. Gerstner (2016) Towards a theory of cortical columns: From spiking neurons to interacting neural populations of finite size arXiv:1611.00294

Related to **T4.3.1**

Moritz Deger, Alexander Seeholzer, Wulfram Gerstner Multi-contact synapses for stable networks: a spike-timing dependent model of dendritic spine plasticity and turnover arXiv:1609.05730v1

Sonja Grün, JUELICH:

Michael Denker, Lyuba Zehl, Bjørg E. Kilavik, Markus Diesmann, Thomas Brochier, Alexa Riehle, Sonja Grün (2017) LFP beta amplitude is predictive of mesoscopic spatio-temporal phase patterns. arXiv:1703.09488 [q-bio.NC]

Olivier Faugeras, INRIA:

Pierre Guiraud, Etienne Tanré, Stability of synchronization under stochastic perturbations in leaky integrate and fire neural networks of finite size, submitted arXiv:1609.07103v1

Olivier Marre, UPMC:

Ferrari U, Gardella C, Marre O, Mora T (2016). Closed-loop estimation of retinal network sensitivity reveals signature of efficient coding arxiv: 1612.07712 [q-bio.NC]. Submitted

Lefebvre B, Yger P, Marre O (2016). Recent progress in multi-electrode spike sorting methods Journal of Physiology Paris, in press. Biorxiv: 086991

Deny S, Ferrari U, Mace E, Yger P, Caplette R, Picaud S, Tkacik G, Marre O (2016). Multiplexed computations in retinal ganglion cells of a single type biorxiv: 080135. In revision

Yger P, Spampinato GLB, Esposito E, Lefebvre B, Deny S, Gardella C, Stimberg M, Jetter F, Zeck G, Picaud S, Duebel J, Marre O (2016). Fast and accurate spike sorting *in vitro* and *in vivo* for up to thousands of electrodes biorxiv: 067843. Submitted

Botella-Soler V, Deny S, Marre O, Tkacik G (2016). Nonlinear decoding of a complex movie from the mammalian retina. arXiv: 1605.03373 [q-bio.NC]. In revision.

Ulisse Ferrari, Tomoyuki Obuchi & Thierry Mora **Random versus maximum entropy models of neural population activity** *ArXiv & bioRxiv*

https://doi.org/10.1101/092973

Shimon Ullman, Weizmann:

Ben-Yosef, G. Assif, L., Shimon Ullman, S. Full interpretation of minimal images. Submitted, 2017

Ben-Yosef, G. Yachin, A., Shimon Ullman, S. A model for interpreting social interactions in local image regions. Submitted, Submitted, 2017

26.2 Publications M1-M12

Gustavo Deco, UPF:

P281 Córdova-Palomera A., Tornador C., Falcón C., Bargalló N., Brambilla P., Crespo-Facorro B., **Deco G.**, and Fañanás L., "Environmental factors linked to depression vulnerability are associated with altered cerebellar resting-state synchronization." Scientific Reports (2016) 6, 37384.

P280 Insabato A, Pannunzi M, Deco G (2017) Multiple Choice Neurodynamical Model of the Uncertain Option Task. PLoS Comput Biol 13(1):e1005250. doi:10.1371/journal.pcbi.1005250

M6 **Zamora-López, G.**, Chen, Y., **Deco, G.**, Kringelbach, M.L. and Zhou, C.S. "Functional complexity emerging from anatomical constraints in the brain: the significance of network modularity and rich-clubs." Scientific Reports (2016) 6, 38424.

Alain Destexhe, CNRS:

M6 Zerlaut, Y., Telenczuk, B., Deleuze, C., Bal, T., Ouanounou, G. and Destexhe, A. Heterogeneous firing rate response of mice layer V pyramidal neurons in the fluctuation-driven regime. Journal of Physiology 594: 3791-3808, 2016 Jul 1.

M6 Le Van Quyen, M., Muller, L., Telenczuk, B., Cash, S.S., Halgren, E., Hatsopoulos, N.G., Dehghani, N. and Destexhe, A. High-frequency oscillations in human and monkey neocortex during the wake-sleep cycle. Proc. Natl. Acad. Sci. USA 113: 9363-9368, 2016.

M6 Barbieri, F., Trauchessec, V.. Caruso, L., Trejo Rosillo, J., Telenczuk, B., Paul, E., Bal, T., Destexhe, A., Fermon, C., Pannetier-Lecoeur, M. and Ouanounou, G. Local recording of biological magnetic fields using Giant Magneto Resistance-based micro-probes. Nature Scientific Reports 6: 39330, 2016 Dec 19, Open Access.

M6 Telenczuk, B., Dehghani, N., Le Van Quyen, M., Cash, S., Halgren, E., Hatsopoulos, N.G. and Destexhe, A. Local field potentials primarily reflect inhibitory neuron activity in human and monkey cortex. Nature Scientific Reports 7: 40211, 2017 Jan 11, Open Access.

Related to **T4.1.4**

Bedard, C., Gomes, J-M., Bal, T. and Destexhe, A. A framework to reconcile frequency scaling measurements, from intracellular recordings, local-field potentials, up to EEG and MEG signals. DOI: 10.3233/JIN-160001 Journal of Integrative Neurosci. 16: 3-18, 2017.

P282 Touboul, J. and Destexhe, A. Power-law statistics and universal scaling in the absence of criticality. Physical Review E 95: 012413, 2017 January 31.

Markus Diesmann, JUELICH:

M6 David Dahmen, Hannah Bos, and Moritz Helias (2016) Correlated Fluctuations in Strongly Coupled Binary Networks Beyond Equilibrium. Phys. Rev. X 6, 031024

Related to T4.1.3

M6 Bos H, Diesmann M, Helias M (2016) Identifying Anatomical Origins of Coexisting Oscillations in the Cortical Microcircuit. PLoS Comput Biol 12(10): e1005132. doi:10.1371/journal.pcbi.1005132

Related to T4.1.3

M6 Andrei Maksimov, Sacha J. van Albada and Markus Diesmann **[Re] Cellular and Network Mechanisms of Slow Oscillatory Activity (<1 Hz) and Wave Propagations in a Cortical Network Model** Rescience, 2(1). http://doi.org/10.5281/zenodo.161526 Open Access

Related to **T4.2.1**

P43 Hagen E, Dahmen D, Stavrinou ML, Linden H, Tetzlaff T, van Albada SJ, Grün S, Diesmann M, Einevoll GT (2016) Hybrid Scheme for *Modelling* Local Field Potentials from Point-Neuron Networks. Cereb Cortex. 26 (12) 4461-4496. DOI: 10.1093/cercor/bhw237

P272 Dmytro Grytskyy, Markus Diesmann, and Moritz Helias (2016) Reaction-diffusion-like formalism for plastic neural networks reveals dissipative solitons at criticality. Phys. Rev. E 93, 062303 https://doi.org/10.1103/PhysRevE.93.062303

Related to T4.1.3

P149 Schuecker J, Schmidt M, van Albada SJ, Diesmann M, Helias M. Fundamental activity constraints lead to specific interpretations of the connectome (2017) PLoS CB 13(2):e1005179, doi:10.1371/journal.pcbi.1005179.

Related to T4.2.1

Einevoll, NMBU

M6 Ness, Remme, Einevoll. *Active subthreshold dendritic conductances shape the local field potential*. J Physiol (2016) 594:3809–3825.

Involved in T4.1.4 - Improved LFP model with quasi-active conductances. Acknowledgements: RUP – 604102

M6 Halnes, Mäki-Marttunen, Keller, Pettersen, Andreassen, Einevoll. *Effect of Ionic Diffusion on Extracellular Potentials in Neural Tissue*. PLoS Comput Biol (2016) 12(11): e1005193. Involved in T4.1.4 - Improved LFP model with quasi-active conductances. Acknowledgements: RUP - 604102

P270 Miceli, Ness, Einevoll, Schubert. *Impedance Spectrum in Cortical Tissue: Implications for Propagation of LFP Signals on the Microscopic Level*. eNeuro (2017) 4(1) 0291-16.2016. Involved in T4.1.4 - Simplified EEG models, and T4.1.4 - Improved LFP model with quasi-active conductances. Acknowledgements: SGA1 - 720270

Olivier Marre, UPMC:

M6 Ulisse Ferrari (UPMC) Learning maximum entropy models from finite-size data sets: A fast data-driven algorithm allows sampling from the posterior distribution *Published in Physical Review E, 2016 August 1*

Related to **T4.4.2,** Model of the retina responding to complex stimuli

P271 Lefebvre B, Yger P, Marre O (2016). Recent progress in multi-electrode spike sorting methods Journal of Physiology Paris, in press. http://dx.doi.org/10.1016/j.jphysparis.2017.02.005

Related to **T4.4.2,** Model of the retina responding to complex stimuli

Olivier Faugeras, INRIA:

P276 Audric Drogoul & Romain Veltz, Hopf bifurcation in a nonlocal nonlinear transport equation stemming from stochastic neural dynamics, Chaos: 27, 021101 (2017); doi: 10.1063/1.4976510

Wulfram Gerstner, EPFL:

P284 Zenke F, Gerstner W. 2017, Hebbian plasticity requires compensatory processes on multiple timescales. Phil. Trans. R. Soc. B 372: 20160259. http://dx.doi.org/10.1098/rstb.2016.0259

Sonja Grün,JUELICH:

M6 Zehl L, Jaillet F, Stoewer A, Sobolev A, Wachtler T, Brochier T, Riehle A, Denker M, Grün S. (2016) Handling Metadata in a Neurophysiology Laboratory. Frontiers in Neuroinformatics 10, 26. DOI:10.3389/fninf.2016.00026

André Grüning, SURREY:

M6 Gardner, Brian and Grüning, Andre: Supervised Learning in Spiking Neural Networks for Precise Temporal Encoding. PLoS ONE 2016(11) http://dx.doi.org/10.1371%2Fjournal.pone.0161335.

Jeanette Hellgren Kotaleski, KTH:

P278 Lindahl M and Hellgren Kotaleski J (2017) Untangling basal ganglia network dynamics and function – role of dopamine depletion and inhibition investigated in a spiking network model, DOI: 10.1523/ENEURO.0156-16.2016

Related to **T4.4.3**

M6 Ekaterina Brocke, Upinder S. Bhalla, Mikael Djurfeldt, Jeanette Hellgren Kotaleski (KTH) and Michael Hanke. Efficient Integration of Coupled Electrical-Chemical Systems in Multiscale Neuronal Simulations *Published in Frontiers in Computational Neuroscience, 2016 September 12, Open Access*

Viktor Jirsa, AMU:

P 52 Jirsa, V. K. et al. The Virtual Epileptic Patient: Individualized whole-brain models of epilepsy spread. Neuroimage (2017). doi:10.1016/j.neuroimage.2016.04.049

Related to **T4.5.2 & T4.2.1**

M12 Proix, T. et al. How do parcellation size and short-range connectivity affect dynamics in large-scale brain network models? Neuroimage (2016, November 15). doi:10.1016/j.neuroimage.2016.06.016

Related to **T4.5.2**

P 51 Spase Petkoski, Andreas Spiegler, Timothée Proix, Parham Aram, Jean-Jacques Temprado, and Viktor K. Jirsa. Heterogeneity of time delays determines synchronization of coupled oscillators Phys. Rev. E 94, 012209 – Published 11 July 2016

P277 Proix T, Bartolomei F, Guye M, Jirsa V. Individual brain structure and modelling predict seizure propagation. Brain 140, 641–654 (2017).

Related to **T4.5.2**

M6 Viktor Müller, Dionysios Perdikis, Timo von Oertzen, Rita Sleimen-Malkoun, Viktor Jirsa (AMU) and Ulman Lindenberger Structure and Topology Dynamics of Hyper-Frequency Networks during Rest and Auditory Oddball Performance Published in Frontiers in Computational Neuroscience, 2016 October 17, Open Access

Related to **T4.5.2**

M6 Andreas Spiegler (AMU), Enrique C.A. Hansen, Christophe Bernard, Anthony R. McIntosh and Viktor K. Jirsa (AMU) Selective activation of resting state networks following focal stimulation in a connectome-based network model of the human brain Published in eNeuro, 2016 September 21, Open Access

Related to **T4.5.1 & T4.5.2**

Marja-Leena Linne, TUT:

P283 Manninen T., Havela R., Linne M.-L. (2017b) Reproducibility and comparability of computational models for astrocyte calcium excitability. Frontiers in Neuroinformatics 11:11. https://doi.org/10.3389/fninf.2017.00011

Related to **T4.2.2**

M6 Heidi Teppola, Jertta-Riina Sarkanen, Tuula O. Jalonen and Marja-Leena Linne. Morphological Differentiation Towards Neuronal Phenotype of SH-SY5Y Neuroblastoma Cells by Estradiol, Retinoic Acid and Cholesterol. Neurochem Res (2016) 41: 731. doi:10.1007/s11064-015-1743-6, Open Access

Idan Segev, HUJI:

P13 Eyal G, Verhoog MB, Testa-Silva G, Deitcher Y, Lodder JC, Benavides-Piccione R, Morales J, DeFelipe J, de Kock CP, Mansvelder HD, Segev I. (2016) Unique membrane properties and enhanced signal processing in human neocortical neurons. Elife. 2016 Oct 6;5. pii: e16553. doi: 10.7554/eLife.16553.

Misha Tsodyks, WEIZMANN:

P293 Romani S, Katkov M, Tsodyks M. Practice makes perfect in memory recall. Learning & Memory. 2016;23(4):169-173. doi:10.1101/lm.041178.115.

P279 Y. Mi, M. Katkov & M. Tsodyks. Synaptic correlates of working memory capacity. Neuron, 93:323-330 (2017).

Related to **T4.3.2**

26.3 RUP Publications

The two following publications were not reported yet.

Walter Senn, UBERN:

Schiess M, Urbanczik R and Senn W: **Somato-dendritic Synaptic Plasticity and Error-backpropagation in Active Dendrites.** PLoS Comput Biol. 2016 Feb 3;12(2):e1004638. doi: 10.1371/journal.pcbi.1004638. eCollection 2016.

Viktor Jirsa, AMU:

Tim Kunze, Alexander Hunold, Jens Haueisen, Viktor Jirsa, Andreas Spiegler Transcranial direct current stimulation changes resting state functional connectivity: A large-scale brain network *modelling* study. NeuroImage 140 (2016) 174–187 http://dx.doi.org/10.1016/j.neuroimage.2016.02.015

26.4 Conference papers

Markus Diesmann, Juelich:

Hagen E, Senk J, van Albada SJ, Diesmann M: Local field potentials in a 4 × 4 mm² multi-layered network model. BMC Neuroscience 2016, 17(Suppl 1):P167

Related to T4.2.1

Van Albada SJ, Rowley AG, Hopkins M, Schmidt M, Senk J, Stokes AB, Galluppi F, Lester DR, Diesmann M and Furber SB (2016). Full-scale simulation of a cortical microcircuit on SpiNNaker. *Front. Neuroinform. Conference Abstract: Neuroinformatics 2016.* doi: 10.3389/conf.fninf.2016.20.00029.

Denker M, Grün S (2016) Designing workflows for the reproducible Analysis of Electrophysiological Data. in: Brain Inspired Computing, eds Amunts K, Grandinetti L, Lippert T, Petkov N. Springer Series Lecture Notes in Computer Science, Vol 10087,pp. 58-72. DOI:10.1007/978-3-319-50862-7_5

P59 Yegenoglu A, Quaglio P, Torre E, Grün S, Enders D. (2016) Exploring the Usefulness of Formal Concept Analysis for Robust Detection of Spatio-Temporal Spike Patterns in Massively Parallel Spike Trains. In: Graph-Based Representation and Reasoning 22nd International Conference on Conceptual Structures, ICCS 2016, Annecy, France. pp 3-16. DOI:10.1007/978-3-319-40985-6_1 ISBN: 978-3-319-40984-9

Related to T4.2.1

The conference paper below also relates to Sonja Grün

P203 Johanna Senk, Alper Yegenoglu, Olivier Amblet, Yury Brukau, Andrew Davison, David Roland Lester, Anna Lührs, Pietro Quaglio, Vahid Rostami, Andrew Rowley, Bernd Schuller, Alan Barry Stokes, Sacha Jennifer van Albada, Daniel Zielasko, Markus Diesmann, Benjamin Weyers, Michael Denker, Sonja Grün (2017) A Collaborative Simulation-Analysis Workflow for Computational Neuroscience Using HPC. In: Di Napoli E., Hermanns MA., Iliev H., Lintermann A., Peyser A. (eds) High-Performance Scientific Computing. JHPCS 2016. Lecture Notes in Computer Science, vol 10164. Springer, Cham, doi:10.1007/978-3-319-53862-4_21

Related to T4.2.1

Sonja Grün,Juelich:

P239 Denker M. and Grün S. Designing workflows for the reproducible analysis of electrophysiological data. in: Brain Inspired Computing, Amunts et al. (Eds.), Lecture notes in computer science, vol 10087. pp. 58–72. doi:10.1007/978-3-319-50862-7_5. 2016

Yegenoglu A, Quaglio P, Torre E, Grün S, Enders D. (2016) Exploring the Usefulness of Formal Concept Analysis for Robust Detection of Spatio-Temporal Spike Patterns in Massively Parallel Spike Trains. In: Graph-Based Representation and Reasoning 22nd International Conference on Conceptual Structures, ICCS 2016, Annecy, France. pp 3-16. DOI:10.1007/978-3-319-40985-6_1 ISBN: 978-3-319-40984-9

Marja-Leena Linne, TUT:

Lehtimäki M., Paunonen L., Pohjolainen S., Linne M.-L. (2017) Order reduction for a signaling pathway model of neuronal synaptic plasticity. IFAC2017 Conference (accepted).

Idan Segev, HUJI

Eyal et al. An analytical and accurate model for reducing neuron- model complexity. Gordon conference on dendrites (March 2017).

27. Dissemination

In addition to the EITN dissemination, SP4 was involved in the science market where we presented our work to non-HBP public.

For EITN dissemination activity please refer to Deliverable D4.6.1 of SGA1.

Marc de Kamps, ULEEDS:

NIPS workshop on: Representation Learning in Artificial and Biological Neural NetworksMartin Perez-Guevara*, Marc De Kamps, Christophe Pallier *Blackboard Architecture simulation with simple biological networks captures the behavior of diverse neuroimaging measurements during language processing (https://sites.google.com/site/mlini2016nips/schedule)*

Wulfram Gerstner , EPFL:

Poster presentation at CoSyne 2017, Holing Li and W. Gerstner, A unified neural network model for reconsolidation and extinction of fear memory

Related to T4.3.1

Markus Diesmann, JUELICH:

Related to T4.2.1:

van Albada SJ, Deco G, Gilson M. Organised workshop "Multi-area models of cortex", CNS*2016, Jeju, South Korea.

Schmidt M, talk “A multi-scale spiking network model of macaque visual cortex,” workshop “Multi-area models of cortex,” CNS*2016.

Schuecker J, Schmidt M, van Albada SJ, Diesmann M, Helias M (2016) talk “Global stability reveals critical Components in the structure of multi-scale neural networks,” 80th DPG annual conference, Regensburg, Germany.

Schmidt M, talk “A multi-scale spiking network model of macaque visual cortex,” International workshop “Vision over vision: man, monkey, machine, and network models”, Osaka, Japan

Van Albada SJ (June 10, 2016) talk “Multiscale *modelling* of cortex at cellular resolution,” PASC16 minisymposium “Level of Detail in Brain Modeling: Common Abstractions and Their Scientific Use”, Lausanne, Switzerland.

Organization of the 9th Bernstein Sparks workshop by Farzad Farkhooi (TU Berlin), Guillaume Lajoie (U Washington), and Moritz Helias (INM-6/IAS-6) on the topic “Recent advances in recurrent network theory: fluctuating correlated dynamics across scales” in Goettingen.

Diesmann, M (March 13-16, 2016) Talk “Multi-area models at cellular resolution” Workshop “Brain Manifesto 2.0”, Rheinfelden, Germany

Diesmann, M (February 15, 2016) Talk “Brain-scale simulations of cortical networks at cellular and synaptic resolution” SFB 936 „Multi-Site Communication in the Brain“, Hamburg, Germany

Diesmann, M (June 22-24, 2016) Talk “Progress and challenges in bottom-up network modeling”, MONA2 – Modelling Neural Activity, Waikoloa, Hawaii

Diesmann, M (February 22-23, 2016) Talk “Necessity and feasibility of brain-scale simulations at cellular and synaptic resolution”, 6th AICS International Symposium, RIKEN AICS, Kobe, Japan

Diesmann, M (Aug 29 – Sep 2, 2016) Talk “Multi-area model of macaque visual cortex at cellular and synaptic resolution” 12th International Neural Coding Workshop, Cologne, Germany

Diesmann, M (February 24th, 2016) Talk” Simulations of macaque cortical networks at cellular and synaptic resolution”, Osaka, Japan

Diesmann, M (October 3-4, 2016) Talk “Brain-scale simulations at cellular and synaptic resolution” Workshop “Vision over vision: man, monkey, machine, and network models”, Osaka, Japan

Diesmann, M (April 24-29 2016) Talk “Necessity and feasibility of brain-scale simulation at cellular and synaptic resolution” Workshop on High-Performance Computing, Stochastic *Modelling* and Databases in Neuroscience, São Paulo, Brazil

van Albada SJ, Deco G, Gilson M. Workshop "Multi-area models of cortex", CNS*2016.

Related to T4.1.3:

Organization of the 9th Bernstein Sparks workshop by Farzad Farkhooi (TU Berlin), Guillaume Lajoie (U Washington), and Moritz Helias (INM-6/IAS-6) on the topic “Recent advances in recurrent network theory: fluctuating correlated dynamics across scales” in Goettingen. Olivier Faugeras gave an invited talk.

Gustavo Deco,UPF:

– Gustavo Deco. Oral presentation (invited speaker). BrainModes 2016: coordinated brain activity: foundations and applications. Royal Flemish Academy of Belgium for Science and the Arts, Brussels, Belgium. December 1-2, 2016. Title of presentation: "*Towards a Whole-Brain Model: Lessons from the Human Connectome*."

– Gustavo Deco. Oral presentation (invited speaker). Alpine Brain Imaging Meeting 2017. Champéry, Switzerland. 8th – 12th January 2017. Title of presentation: "*Novel concept of intrinsic ignition characterises the broadness of communication underlying different brain states*."

- Gustavo Deco. Oral presentation (keynote speaker). MICCN Computational Neuroscience Symposium 2017. Monash Biomedical Imaging Auditorium, Monash, Australia. February 3rd 2017. Title of presentation: "*Towards a global model of brain activity: How to identify brain states?*"

Einevoll, NMBU:

- Ness, Remme, Einevoll. *Active subthreshold dendritic conductances shape the Local Field Potential*. Poster presented at Young Researchers Event, April 2016

- Ness, Remme, Einevoll. *Active subthreshold dendritic conductances shape the local field potential*. Poster presented at HBP summit, October 2016

- Næss, Ness, Dale, Einevoll, *Understanding EEG with Biophysical Modeling*. Poster presented at NRSN PhD Conference, September 2016

- Næss, Ness, Halnes, Halgren, Dale, Einevoll. *Biophysical modelling of single-neuron contributions to EEG and ECoG signals*. Poster presented at SfN, November 2016

Olivier Faugeras, INRIA:

Organization of the 9th Bernstein Sparks workshop by Farzad Farkhooi (TU Berlin), Guillaume Lajoie (U Washington), and Moritz Helias (INM-6/IAS-6) on the topic “Recent advances in recurrent network theory: fluctuating correlated dynamics across scales” in Goettingen. Olivier Faugeras gave an invited talk.

Sonja Grün, JUELICH:
All related to T451

Grün, participated at the booth at the Brain Initiative Meeting in Bethesda, Dec 2016

Gruen, Talk `Analysis of large-scale recordings of neural activity *in vivo* and *in silico*`, Intern. Workshop ‘Introduction to the HBP Collaboratory’, Copenhagen, Denmark, FENS, July 2016

Senk et al, Talk ‘Integrating HPC into a Collaborative Simulation-Analysis Workflow for Computational Neuroscience’, JARA-HPC Symposium, Aachen, Germany Oct 2016

Denker, Poster: ‘Challenges in Designing Workflows for Reproducible Analysis of Electrophysiological Data – Usage of Community Tools’, INCF Neuroinformatics 2016, UK, Sept 2016

28. Education

SP4 and EITN help in the dissemination of the HBP education programme and activities when contacted and also facilitate the hosting of events related to education programme.

Gaute Einevoll, NMBU:

Computational Neuroscience: Bridging brain scales with mathematics. HBP Curriculum – Neurobiology for non-specialists. Lecture 13 , held at the EITN, Paris, 2017 June 21-23: Computational Neuroscience: Bridging brain scales with mathematics https://www.youtube.com/watch?v=8KJLE4j92QM

Alain Destexhe, CNRS:

3rd HBP Winter School "Future Neuroscience _ The Multiscale Brain: from genes to Behaviour" on the 1st of December 2016, in Obergurgl, Austria. Lecture on Multiscale modelling.

Sonja Grün, JUELICH:

organisation of "Advanced course on Neural Data Activity", held in Haus Overbach, Jülich, from March 26 -April 8 2017

29. Ethics

At the beginning of SGA1 the Ethics Rapporteur (ER) Gorka Zamora-López, from UPF, took over the ER responsibilities for SP4. He has since then participated in all Ethics and Society activities requested, including the joint ER – Ethics Advisory Board meeting organised by SP12 at the HBP Summit (October 2016, Florence).

In June 2016, prior to the SIB meeting SP4 prepared an internal evaluation of Ethical issues as requested by the Ethics Management team. As a theoretical SubProject, none of the work packages in SP4 involves carrying out experiments with neither animals nor humans. Nevertheless, for model validation and analysis, SP4 researchers use and manipulate third party animal and human data acquired within and outside the HBP. However, several deficits were identified in what respects the storage and manipulation, in particular non-HBP and 3rd country data. Action has been taken to inform the SP4 partners about correct storage and manipulation of empirical data, e.g. the need to request always, together with the data, the third-party re-use consents.

SP4 has raised several ethical and legal questions which are currently being discussed by the competent organs within HBP.

- While HBP encourages the public release of data acquired and software generated by the Project members, the legal requirements and support to do so need clarification because it may remain unclear to whom legally belongs the data acquired with HBP funding and who is the legal owner of the software developed within HBP.
- Communications between HBP partners, data storage and HBP-related documentation needs the establishment of criteria and policies to guarantee privacy and confidentiality. Unfortunately, due to the lack of secure alternatives HBP members are often pushed to make use of unsecure means of communication, data transfer and cooperative documentation tools, e.g. the use of Google Docs for the creation of classified documentation.

30. Innovation

No innovation to report.

31. Open Research Data

Markus Diesmann, Juelich:

Kunkel S et al. (2017). NEST 2.12.0. Zenodo. 10.5281/zenodo.259534.

Related to **T4.2.1**

Please refer to publications