**THE POSSIBLE CONTROL IMPLICATIONS OF THE INFERIOR OLIVE – DEEP CEREBELLAR NUCLEI PATHWAY IN A DISTRIBUTED PLASTICITY CEREBELLAR MODEL**

Niceto R. Luque¹, Jesús A. Garrido², Richard R. Carrillo³, Eduardo Ros¹

¹Dept. Computer Architecture and Technology, University of Granada, Spain; ²Consorzio Interuniversitario per le Scienze Fisiche della Materia (CNISM), Pavia, Italy; ³Dept. of Computer Architecture and Electronics, University of Almería, Spain.

**MOTIVATION**

The cerebellum is involved in controlling and learning smooth coordinated movements, therefore an accurate understanding of how this control engine works should have a strong impact on the control of biomorphic robots.

- We have studied the possible control implications that the inferior olive - deep cerebellar nuclei cell connections (IO–DCN) may present in a distributed-synaptic-plasticity cerebellar model (activity conveyed by this connection seems to control plasticity at DCN synapses (Bengtsson & Hexaslow 2006) (Ruigrok & Voogd 2000)).
- The Marr and Abus model hypothesized that parallel fibers (PFs) presented LTD and LTP at PC and DCN connections. We have studied a model where the IO–DCN pathway plays a fundamental role by refining the cerebellar learning performance.
- We have developed a firing-rate cerebellar model (Garrido et al. 2012) with plasticity mechanisms at PF–PC and at DCN synaptic inputs (from Mossy Fibers (MFs), Purkinje Cells (PCs) and IO). Therefore, we present a model where the IO–DCN pathway plays a fundamental role by refining the cerebellar learning performance.

**CEREBELLAR MODEL**

Our cerebellar model includes the following plasticity mechanisms:

- **IO-driven LTD** at PC–DCN connections.
- **IO-driven LTP** at PF–PC connections.
- **IO-driven LTD** and **LTP** at PC–DCN connections.
- **PC-driven LTD** and **LTP** at MF–DCN connections.
- **PC-driven LTD** and **LTP** at MF–PC connections.

**DIFFERENT CONTROL PATHWAYS DURING LEARNING PROCESS**

**CONTROL ARCHITECTURE**

The adaptive cerebellar module delivers corrective add-on torque values to compensate for deviations in the inverse dynamic module when manipulating an object of mismatched weight.

**RESULTS**

The plasticity mechanisms that determine the synaptic strength in MF→DCN/PC→DCN/IO→DCN connections were driven by the activity from PCs which is responsible for the balance (homeostasis) between all these plasticity laws. This PC activity makes MF→DCN and PC→DCN synaptic strength increase while makes the IO→DCN synaptic strength decrease. During the first learning stages the IO→DCN corrective action predominates (Fig.5.C and 5.D). Afterwards, this corrective action is gradually decreased while the corrective action provided by MF→DCN and PC→DCN connections is gradually increased. The transition between these two control actions is regulated by the PC activity which in turn, is working in a constant range of frequencies thanks to MF→DCN and PC→DCN connections. We obtain a system able to self-adapt by means of a distributed learning where all the learning sites are complementary working together.

**CONCLUSIONS**

The results suggest that the cerebellar gain control is a consequence of the MF→DCN and PC→DCN synaptic plasticity working in balance with IO→DCN connection. Thus, this balance (homeostasis), which is implemented through different learning, enhances the cerebellar learning performance. IO→DCN connection ensures stability in the very early learning stages, that is, while the weights of MF→DCN and PC→DCN connections have yet to stabilize. Once the learning process is finished, the IO→DCN connection effect ceases.

**ACKNOWLEDGMENTS**

This work was supported in part by the EU project REALNET (IST-270434).