Modeling cerebellar learning for spatial cognition

Jean-Baptiste Passot[1,2], Laure Rondi-Reig[1,2], Angelo Arleo[1,2]

[1] UPMC Univ Paris 6, UMR 7102, F-75005 Paris, France

[2] CNRS, UMR 7102, F-75005 Paris, France

E-mail: jean-baptiste.passot@upmc.fr

Recent experimental findings have begun to unravel the implication of the cerebellum in high-level functions such as spatial cognition [1,2]. We focus on behavioural genetic data showing that L7-PKCI mice (lacking LTD at parallel fibres–Purkinje cell synapses) have a spatial learning impairment in the Morris Watermaze (MWM), whereas they exhibit normal performances in the Starmaze, a paradigm that reduces the procedural demand of the task [3]. These results suggest that cerebellar learning may prominently contribute to the procedural component of spatial learning [3].

We model the main information processing components of the cerebellar microcomplex via a large-scale network of spiking neurons. We test the performances of artificial L7-PKCI mice in simulated MWM and Starmaze environments. Importantly, we isolate the purely procedural component of the learning process by endowing simulated controls and mutants with identical declarative learning capabilities. The model reproduces most of the experimental results on the learning impairments of L7-PKCI mice: in the MWM, the mean escape latency and the mean angular deviation between the optimal direction to the target and the actual motion direction of the animal are both significantly larger compared to controls. These differences are not due to swim capability deficits. Furthermore, consistent with experimental data, simulated mutants and controls exhibit comparable learning capabilities in the Starmaze paradigm. On the other hand, our simulations cannot reproduce the experimentally observed difference between the goal-searching behaviour of mutants and controls in the MWM (measured by the ratio between the time spent within the target quadrant and the duration of the trial [2]). In fact, our results suggest that a purely procedural impairment cannot explain this latter deficit, and they raise the hypothesis of a concurrent declarative learning impairment of L7-PKCIs occurring upstream the cerebellum, e.g. at the level of the hippocampal–cortical interaction.

Finally, we address the issue of how multiple internal cerebellar models [4] could cooperate to optimise procedural spatial learning. We show that our cerebellar network can simultaneously learn an internal representation of the motor apparatus – the forward model – and the online generation of motor-correction signals – the inverse model. We show that the slow learning process undertaken by the inverse model may benefit from the fast learning property of the forward model, which supports the working hypothesis that these two types of models coexist and are functionally coupled in the cerebellum [4].

References

1. L. Rondi-Reig, E. Burguière . **Is the cerebellum ready for navigation ?** *Prog Brain Res.* 2004,**148**:199-212. .
2. L. Petrosini, M. G. Leggio, M. Molinari. **The cerebellum in the spatial problem solving : a co-star or a guest star?** *Progress in Neurobiology* 1998,**56**:191-210,
3. E. Burguière , A. Arleo, C.I. De Zeeuw, A. Berthoz, L. Rondi-Reig. **Spatial navigation impairment in mice lacking LTD: a motor adaptation deficit ?** *Nat Neurosci.* 2005,**10**:1292-1294.
4. D. Wolpert, R.C. Miall, M. Kawato. **Internal models in the cerebellum.** *Trends in Cognitive Sciences.* 1998:**2(9)**:338-347