# ABSTRACT

Acquisition, maintenance and loss of neuronal functions depend on still poorly understood epigenetic mechanisms. We aimed to identify genes potentially involved in the time control of behaviour in the nematode *C.elegans*. The responsive behaviour to odorants, named chemotaxis, is associated to AWC chemosensory neurons whereas long term memory of early exposure to odors, named imprinting, is associated to AIY interneurons. In *C.elegans*, those behaviours are both under a precise temporal regulation. Chemotaxis is only observable from the larval stage 2 (L2); it peaks during early adulthood and then vanishes.

Olfactory imprinting is acquired possible during a very short post-natal time window, named critical period, corresponding to the larval stage 1 (L1). We analysed the consequences of mutations for the genes *spr-1*, *spr-5* and *hbl-1* on the time control of chemotaxis and imprinting. Those genes are orthologues of genes known for being involved in the time control of neurogenesis in mammals and Drosophila. *spr-1* corresponds to the Corest repressor, *spr-5* is the partner of CoRest, lsd1 (lysine demethylase), and *hbl-1* corresponds to hunchback in Drosophila. We analysed the expression of *hbl-1* in the AWC ans AIY neurons.

We observed that the loss of function of *hbl-1*, *spr-1*/CoRest and *spr-5*/lsd1 genes induces an earlier acquisition of the chemotaxis competence.

The same mutants showed both advanced chemotaxis during L1 and imprinting defect. We hypothesize that SPR-1, SPR-5 and *hbl-1* which inactivation leads to the same effects, could form a histone-modifying complex as the mammal REST-CoREST-LSD1 complex. Those genes could control the length of the competence window in the AWC and AIY neurons by maintaining their plasticity.

Key-words: imprinting; critical period; epigenetic; chemotaxis; neuron maturation; neoteny.