In my thesis I am dealing with human endogenous retroviruses (HERVs), which are involved in placenta development, and with porcine endogenous retroviruses (PERVs) in the context of the risk of their transmission to a patient xenotransplanted with a pig organ.
We have shown DNA methylation to be an important silencing mechanism regulating HERV as well as PERV. (1) Whereas in placenta the demethylation of HERVs ERVWE1 and ERVFRDE1 is crucial for its correct function, in the testis it is connected with seminoma development. (2) It seems that methylation is partially responsible for low PERV expression in tissues and notably reduces the risk of zoonotic transmission during xenotransplantation. (3) However, the risk remains because in contrast with mouse and rat cells, some human cells are permissive to porcine retroviruses in vitro thanks to functional receptors and their inability to efficiently silence the integrated PERVs.