Abstract (eng)

The phenomenon of hybrid sterility represents one of the evolutionary mechanisms that enables speciation. Only a few speciation genes have been uncovered. The only one found in mammals is *Prdm9* (PR-domain 9). Data in the literature on the involvement of *Prdm9* in decreased fertility of various semifertile hybrid males of house mouse subspecies were scarce before the results of this thesis were completed, despite that such males are much more frequent in nature than the fully sterile ones. Utilizing a panel of genetic tools and a battery of phenotyping tests, this thesis shows a central role of *Prdm9* in fecundity of hybrids, including many fertility disorders and age dependency. Both increasing and reducing the *Prdm9* gene dosage significantly elevated fertility parameters. Surprisingly, even the allele that in one copy causes full hybrid sterility increased F1 hybrid fertility when present in multiple copies. The PRDM9 protein also plays a role in identifying the sites of meiotic recombination. This study also points out the principles of allelic competition in determination of the sites of preferred recombination (hotspots), which suggests a possible link between both previously described *Prdm9* roles. This thesis summarizes a set of three logically interconnected publications with the ambition to become the key piece of puzzle illustrating the mechanism of speciation. The presented results should have an impact on studies dealing with decreased fertility of the mouse laboratory strains as well as on analysis of the wild mouse populations. Thus, they should be taken into an account when designing such experiments.