DOCTORAL CANDIDATE:	Pernille Nilsson
DEGREE:	Philosophiae Doctor
FACULTY:	Faculty of Mathematics and Natural Sciences
DEPARTMENT:	Department of Biosciences
AREA OF EXPERTISE:	Comparative genomics
SUPERVISORS:	Nils Christian Stenseth, Sissel Jentoft, Kjetill S. Jakobsen, Boris V. Schmid & W. Ryan Easterday
DATE OF DISPUTATION:	7th of June 2019
DISSERTATION TITLE:	*The evolutionary arms race between plague and great gerbil – burrowing into host resistance*

The great gerbil (*Rhombomys opimus*) is a desert dwelling rodent distributed throughout Central Asia and is a key reservoir species for several vector-borne diseases including plague (*Yersinia pestis*).

Pernille Nilsson, a PhD candidate at the University of Oslo has explored the genetic basis for the high level of plague resistance in this natural reservoir species uncovering new knowledge and increasing our understanding of plague resistance in a wild, non-model species. The research combined the use of experiments, whole genome data and genomic tools to establish the involvement of multiple genes and pathways of both the immune system and regulation of basic cellular functions to facilitate plague resistance in great gerbils.

In her research, Nilsson uncovered a species-specific duplication of the Major histocompatibility complex class II (*MHCII*) *DRB* locus that could putatively increase the survival of infected animals through faster initiation of the adaptive immune system. Nilsson also revealed genetic differences between surviving and non-surviving individuals that could explain the different outcomes of plague infection in great gerbils.

Lastly, Nilsson developed a molecular tool that is capable of rapidly and specifically detecting and quantifying the plague bacterium in a variety of samples from animal tissues to soil samples. This makes it eligible as a tool for plague surveillance in wildlife, in bioforensics and in studies of disease dynamics within rodent hosts.

Overall, this PhD research demonstrates modifications in the great gerbil both at the genomic level by the species-specific duplication of the *MHCII DRB* locus and at the population level where signs of selection could be connected to the *MHCII* gene duplication as well as the function and regulation of the cells' actin cytoskeleton, signaling pathways and metabolism.