Contrary to the classical mouse inbred strains with unnatural genetic variability, wild-derived strains offer a more suitable model for evolutionary immunology. Toll-like receptors (TLRs) belong to initial detectors of invading pathogens. Although TLRs recognise conserved structures they were shown to be polymorphic. This polymorphism is associated with various diseases. In my thesis, I describe variability of *Tlr1*, *2* and *6* in 24 inbred strains derived from two subspecies of house mouse (*Mus m. musculus* and *M. m. domesticus*). These *Tlrs* exhibit different levels in variability among the strains. In *Tlr1* the polymorphic sites are spread along the whole exodomain. *Tlr6* is quite conserved (a lower amount of substitutions located far from the binding region and with minor modifications in the amino acid residue properties). *Tlr2*, on the contrary, contains some substitutions with substantial alternations of residue properties that are located within or nearby the binding region and the subspecies differ at these sites. All alleles of *M. m. domesticus* and *M. m. musculus*, except for *Tlr1* PWD, *Tlr2* STAIL, are phylogenetically separated. The strains and the subspecies vary in the production of IL-1β, IL-12 a NO after stimulation by TLR1, 2 and 6 ligands. This trend is, however, presumably influenced by the effect of particular lines (e.g. BULS in the case of IL-1β). The results of my thesis imply independent co-evolution of TLR2 with pathogens in these subspecies.