7 Summary

Detection and characterisation of Shigatoxin-producing *Escherichia coli* (STEC) in pigeons

366 feces samples of pigeons from three different habitats (racing pigeons, showing pigeons, feral pigeons) were investigated for STEC. The samples were from healthy animals and flocks, only three pigeons showed diarrhea. The samples were sent for investigation of *Salmonella* sp. and parasites to the veterinary laboratory of the university of Göttingen. A part of them were collected at a exhibition of showing pigeons. Initially a screening for stx with polymerase chain reaction and DNA-DNA-hybridisation was made. At this step the stx1 and stx2 and the variants of stx2 made distinctions. From a part of the positive stx-samples STEC were isolated with blotting technics. STEC were investigated for biochemical reactions and for the virulence markers *eae* and *hly*EHEC.*

67% of the feces samples harbored stx. The prevalence differed a lot within the habitats. Racing and showing pigeons, which had a close relationship to human beings, showed the following data: 15,1% stx1, 27,1% stx2, and 25,6% stx2f in showing pigeons respectivley 45,6% stx1, 32,2 stx2, and 33,2% stx2f in racing pigeons. The distribution of prevalence in the feral pigeons were 2% stx1, 16% stx2, and 76% stx2f.

21 of the 27 STEC, which were isolated from 13 feces samples, were tested positive for stx2f and 6 positive for stx1. The samples named B92 (racing pigeons) harbored 10 different STEC. The factor for adhesion Intimin (*eae*) was detected in 66% of the stx1-STECL and in 90,5% of the stx2f-STECL. The gene *hly*EHEC coding for the virulence marker enterohemolysin could only be detected in one single stx1-STECL. Three stx1-STECL were detected from racing pigeons, which showed clinical signs. Two of them harbored additionally the *eae*-gene.

The high prevalence of the stx-genes and the existence of the virulence markers *eae* and *hly*EHEC seemed to be a high risk for human beings to become infected with STEC from pigeons. But there was no experience of toxicity of the Stx2f, which was mostly detected in feral pigeons. Interestingly there was a higher prevalence of stx1 and stx2 in racing and showing pigeons than in feral pigeons. 60% of investigated feral pigeons harbored stx2f, which could not yet be detected in men. Therefore the risk of men to get infected by STEC-harboring pigeons depends on were the animals live.

Avian disease due to STEC-infection could not longer be excluded. Wether or not pigeons might play a role in epidemiology of edema disease of piglets could not be proved in this work.