Most *Escherichia coli* strains live in the human intestine and cause no harm. However, pathogenic strains, such as enterohaemorrhagic *E. coli*, also exist, and pose a growing danger to human health. These strains may cause haemorrhagic colitis, which in some people develops into a severe and often fatal complication called hemolytic-uremic syndrome. During the 2011 outbreak in Germany 2987 people fell ill with haemorrhagic colitis, 53 of whom died. Children and elderly people are at the greatest risk of developing severe complications. The main virulence factors produced by enterohaemorrhagic *E. coli* are Shiga toxins, which bind to specific receptors on endothelial and epithelial cells in the intestine and kidney, as well as on red blood cells, causing cell death.

Ruminants (especially cattle) are the main reservoir of enterohaemorrhagic *E. coli*, but they have also been found in other wild and domestic animals, i.e. dogs. Recent data suggest that urban birds (i.a. pigeons) also can harbor these microbes and, like ruminants, show no signs of disease. Importantly, urban birds live in populated areas and forage in spaces frequented by humans, such as waste collection or playgrounds, thus creating opportunities for transmission of pathogenic bacteria.

In this project, we aim to find out why birds are refractory to Shiga toxins despite having the receptors on endothelium and red blood cells. The preliminary data show that birds have two types of receptors (glycolipid and glycoprotein), the synthesis of which is controlled by two genes, in contrast to only one gene in mammals. Elucidation of this phenomenon may help design strategies of mitigating complications in people infected with enterohaemorrhagic *E. coli*.