Colicin K belongs to the group of ColE1 pore-forming colicins which destroy the electrochemical potential of the cytoplasmic membrane. Production of the bacteriocin is encoded by three genes: *cka* encoding the colicin, *cki* encoding immunity and *ckl* encoding lysis.

Our studies have shown that synthesis of colicin K is highest in the stationary phase due to a depletion of nutrients and that the alarmone ppGpp is an activator of colicin K synthesis. We further found that ppGpp stimulates Cka synthesis by enhancing its translational efficiency.

As a number of other colicins, colicin K is released semi-specifically by cell lysis. Therefore, only a part of the population should express the colicin activity and lysis genes. We have shown that in the stationary phase, transcription from the *cka* promoter is derepressed in only approximately 3% of the colicinogenic population and that the LexA protein is a decisive regulatory element repressing expression at the level of transcription in the large majority of the population.

Recently, we have shown that the IscR global transcription repressor is responsible for delaying *cka* expression following DNA damaging treatment. As LexA is additionally involved in antibiotic resistance emergence and virulence, we have also focused on the mechanism of LexA binding to operator sequences. Our studies have revealed that rotation of the LexA DNA binding domain, with respect to the dimerised C-terminal domain, is required for selective DNA binding.