The mechanisms that maintain and promote bacterial biodiversity are not yet fully understood. Theoretical models have shown that the secretion of narrow spectrum toxins called bacteriocins together with high levels of toxin immunity among bacterial strains could be a mechanism that leads to the origin and maintenance of high bacterial biodiversity. Models predict that communities of coexisting bacterial will be biased towards strains that are immune to most toxins, while producing only very few toxins themselves. To test model predictions, we analysed the pairwise antagonistic interactions between 26 strains of the gram positive bacterial pathogen *Streptococcus pneumoniae*. Our experimental results support the theoretical predictions. Strains from our sample are highly biased towards hyperimmunity while they are very limited in their killing spectrum. Furthermore, we found that the frequency distribution of toxicity and immunity was highly dependent upon the timing of presentation of target strains to putative killers. This implies an important role of intercellular signaling, which induces both bacteriocin production in killer strains and immunity in target strains. To understand this dependence, we sequenced the peptide signal inducing toxins and immunity as well as the peptide receptor. In addition, we developed novel theoretical models to simulate the evolution of toxicity and immunity in systems utilizing quorum dependent bacteriocin production, as is observed in all gram positive bacteria. Simulation results will be discussed in the context of experimental data.