S1. ***Population structure of S equi***

Sequencing of the genomes of over 200 isolates of *S equi* recovered from outbreaks in the UK, Australia, Belgium, Canada, Ireland, New Zealand, Saudi Arabia, Sweden, and the USA has provided a global snapshot of the genetic diversity of *S equi* and the complex epidemiology of some individual outbreaks.[21] The sequence diversity observed in the global collection of isolates utilized by Harris et al. was surprisingly low, suggesting that the currently circulating worldwide strains of *S equi* share a common ancestor sometime between 1819 and 1946.[21] This period of history was marked by global conflicts during which horses performed critical roles.[22]

Persistence of *S equi* in the guttural pouch has been shown to drive both the diversification and decay of its genome, and potentially explains the path to host restriction.[21] During persistent infection the rate of mutation increases and genes that are not required for survival in the guttural pouch can be lost.[21] If the loss of genes affects the ability of *S equi* to cause strangles, such as those required for the production of the hyaluronic acid capsule or the equibactin siderophore, then those less virulent mutant strains have become an evolutionary dead-end. [21] However, if the lost genes – such as those required for lactose fermentation - are not required for the induction of strangles, then those losses are likely to become fixed in the population over time, reducing the ancestral capabilities of *S equi*, ultimately leading to its host restriction.[23]