***Introduction***

* Evidence for variation in ST generally for NASM
  + should have stuff from grant narrative, SCC DIM paper
  + Souza 2016
    - **CHRONIC IMI isolate… like mine …** S. chromogenesisolated from a chronic IMIhad greater ability to adhere to bovine mammary epithelial cellscompared to a strain isolated from the teat apex
  + Haveri et al 2005 Bacterial Genotype Affects the Manifestation andPersistence of BovineStaphylococcus aureusIntramammary Infection
    - Persistence = cows wererevisited 2 and 4 weeks posttreatment for follow-up samplingand clinical examination
    - Finland
    - IMI graded as sublicnical, mild clinical, or sever clinical
    - Two-hundred seventeenStaphylococcus aureusisolates from 116 dairy cows with intramammary infectionswere analyzed by pulsed-field gel electrophoresis to study the association between symptom severity, persis-tence of infection, and bacterial genotype. Among five main genotypes infecting 90% of the cows, one wasassociated with severe clinical symptoms but reduced persistence
  + Haveri 2007
    - To screen putative virulence genes in Staphylococcus aureus causing persistent and nonpersistent bovine intramammary infections (IMI) with different clinical characteristics. To examine, whether a possible relationship exists between genetic profile and infection persistence, clinical signs of infection, clonal type determined by pulsed-field gel electrophoresis (PFGE), and antimicrobial resistance
    - Our results suggest that certain genetic elements are over-representative in S. aureus isolates especially from persistent bovine mastitis. This phenomenon seems to be in connection with clonal type and is often concomitant with penicillin resistance
    - **Isolates from persistent mastitis typically harboured blaZ (P < 0Æ01**
    - **More common pulsotypes had genes encoding PTSAg VF**
    - **a common clonal type carrying a set of genes appeared to predominate in isolates from persistent bovine mastitis**
* Species level, see if gene number associated with SCC – from naushad
  + We also computed the difference in gene associations among NAS species and forisolates from low, medium, and high SCC and CM. Differences in associations forindividual NAS species and isolates from various inflammatory responses suggestcomplex interplay among virulence genes in causing disease. Unraveling these inter-actions will be important to elucidate distinctive pathogenic mechanisms of individualNAS species and assessing species-specific effects on udder health
* AMR in NASM
  + Presence of this AMG confers resistance to benzylpenicillin by the production of beta-lactamases which hydrolytically destroy β-lactam antibiotics.
* Avall: Virulence factors are seen as properties (i.e., gene products) that enable a microorganism to establish itself on or within a host of a particular species and enhance its potential to cause disease (Virulence Factor Database). Thus, any property of the microorganism which enhances its’ potential to survive within a host can be seen as a virulence factors.

The objectives of the current study were to: 1) identify if distinct strain types of *S. chromogenes* are associated with IMI where quarter SCC is consistently elevated (HIGH SCC phenotype) vs. consistently low (LOW SCC phenotype), 2) identify if *S. chromogenes* from HIGH SCC phenotype are more likely to carry genes encoding for antimicrobial resistance (as determined by whole-genome sequencing) vs. LOW SCC phenotype, and 3) identify if *S. chromogenes* from HIGH SCC phenotype possess a higher number of genes encoding previously-described staphylococcal virulence factors vs. LOW SCC phenotype.