General conclusions

* Nasm species distribution and prevalence are similar to what has been described previously on conventional farms
  + They act the same way elevating SCC
  + Their overall prevalence in the short comm. Related to other pathogens too
* Similar across facility types – from 40 herd, at bulk tank level
  + Both staph spp and aureus?
* NASM are worthy of consideration as mastitis pathogens, are not “minor” mastitis pathogens, as evidenced by my work
  + 20% quarter level prevalence of NASM – totally dominant
  + 9/10 species elevate above healthy quarters
  + AMR – 33% penicillin resistant in my work
    - Other work point out concerning AMR profiles
  + Ability to be PERSISTENT
  + And also ability to be PERSISTENTLY HIGH
  + Looks like they’re contagious – same RAPD type, dominant ST across farms for limited number of 30 I did MLST for
* Contributed to the understanding of diversity within s chromogenes, the dominant species
  + ID’d new ST
  + Characterized 30 causing persistent IMI into 10 MLST
  + Described vir profile for 30
    - Novel vir factor desc for exo c and coa
  + Described AMR profile for 30
* Although found no link between high and low scc in persistent isolates, DOES seem to point to a genetic basis for virulence and AMR carriage

Bigger significance

* This work generated foundational knowledge about staphylococci causing mastitis on organic dairy farms in Vermont. Mitigating the effect of mastitis caused by staphylococci through targeted prevention and control measures helps dairy producers achieve quality price premiums and results in a higher-quality product for consumers.
* While all dairy producers rely on best management practices to support cow health, mastitis control is of the utmost importance in the prevention of intramammary infections (IMI) on organic farms. Understanding the epidemiology of mastitis pathogens leads to more effective measures which prevent or limit transmission of IMI. This work sought to better understand the epidemiology of the most relevant pathogens causing IMI in organic dairy cows in Vermont.

40 herd/bedded pack stuff

* Do a bigger study, with more BP enrolled, with a more variation in milk quality and mastitis rates, to have the power to ID mgmt. practices which do actually result in good udder health on BP farms
  + Perhaps the biggest limitation of the current study is the small number of farms in each facility type, which limited statistical power
  + A related limitation is that well-established mastitis control practices were widely adapted by participating herds, so we were unable to analyze associations between certain practices and BTM quality, udder health, and hygiene. As group sizes for each facility type were limited, we would caution against making inferences from the findings beyond the source population of this study. The potential still exists for future studies with a larger number of farms enrolled to further characterize milk quality and udder health on BP systems in the Northeastern US. By enrolling farms from a larger geographic area, future studies may be able to enroll a larger number of BP farms, increasing the statistical power needed to identify particular management factors which are beneficial or detrimental on BP specifically. Our data may be used to inform new hypotheses and power calculations for future study design.

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Bayesian analysis of NAS IMI by IMI definition

* Compare what I would have called an IMI with 1 CFU CNS (whatever dohoo recommends) with 1 sample vs. my definition

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Virulence of chromogenes

* **Small scale:** repeat analysis of VF by CATEGORY, like in Naushad et al. to see if I get anything different
  + In a linear regression with all virulence factors considered together, Naushad et al. (2019) did not find that an increase in the overall number of VF for a NAS isolate was associated with an increase in the logSCC of the associated IMI. However, when stratified by type of virulence, the presence of each additional toxin gene for a NAS isolate was associated with a 0.024 increase in logSCC of the associated IMI (Naushad et al., 2019). Similarly, in a logistic regression with ordinal categories for IMI severity (low SCC, medium SCC, high SCC, and clinical mastitis), an overall increase in the number of VF was not associated with increased severity of an IMI (Naushad et al., 2019). In a regression analysis with VF stratified by functional category, the presence of each additional VF gene associated with host immune evasion increased the odds of having a more severe immune response by 0.074 (Naushad et al., 2019). Naushad et al. (2019) applied various approaches in order to determine whether particular VF distributions had any association with SCC category or occurrence of clinical mastitis, but no clustering of isolates representing low SCC, medium SCC, or high SCC or clinical mastitis was identified.
* **Small scale:** do coag testing for all 30 isolates; all had vWbp
  + 2 of ST25 had coa; find other st25 in my datatset and test them

AMR of chromogenes

* Doing phenotypic resistance profiles for my isolates, and then seeing how the phenotypic resistance profiles stack up to using WGS data to identify AMR in silico
* **Small scale: do phenotypic AB testing for my 30 isolates; how does blaZ carriage and penicillin resistance stack up?**
  + inconsistencies exist between phenotypic and genotypic resistance results, due either to 1) detection of phenotypic resistance in the absence of expected genotypic determinants, or 2) phenotypic susceptibility despite the presence of genotypic determinants. For isolates of *S. aureus* associated with bovine mastitis, both of these types of discrepancies have been reported for penicillin resistance (Sampimon, 2009; Taponen et al., 2023). This also holds true for the other staphylococci; as summarized by Sampimon (2009), “agreement between phenotypic and genotypic test results for assessment of resistance of CNS of bovine origin to penicillin, oxacillin, and ML [macrolide] antibiotics depended on the antimicrobial compound of interest and on methods used to analyse and interpret test results, but was rarely perfect.” In a study by Taponen et al. (2023) comparing methods of testing for β-lactamase mediated resistance, overall agreement between phenotypic and genotypic resistance tests was moderate to substantial for staphylococci from bovine IMI. However, some inconsistencies were found between phenotypic susceptibility by disk diffusion method, the nitrocefin test to assess β-lactamase production, and PCR to detect the presence of the *blaZ, mecA*, and *mecC* genes encoding the β-lactamase gene.
  + Current status
    - What was cut-off and method used by sam? Think it was plates of a certain concentration
    - 12/30 tested for phenotypic penicillin susceptibility
    - 12/12 agreement between blaZ carriage and phenotype
      * 8 no blaZ, susceptible
      * 4 yes blaZ, resistant
  + Would we find other phenotypic resistance we didn’t identify in silico
* Bla-Z- where is it carried? Plasmid or chromosomally?
  + Where is some of mine carried?
  + Consistent carriage of blaZ from ST originating from different farms may suggest that blaZ is located chromosomally for these S. chromogenes isolates, instead of on a plasmid. Location of blaZ carriage is not well characterized for S. chromogenes, but a study of S. aureus IMI isolates in Finland and Norway found that 26 out of 34 Finnish isolates (76.5%) and 25 out of 44 Swedish isolates (56.8%) carried blaZ on a plasmid (vs. chromosomally) (Bagcigil et al., 2012). They also characterized the diversity of blaZ genes among the S. aureus isolates, identifying 6 different protein signatures. Studies exploring whether blaZ is more likely to be carried chromosomally or on a plasmid for S. chromogenes from bovine IMI, as well as characterizing the genetic diversity of the gene present in this population of isolates, would be useful in understanding transmission of penicillin resistance for this predominant mastitis pathogen.

Diversity within chromogenes

* ID of chromogenes by MALDI – could maldi be used for ST ID?
  + There were 3 isolates MALDI said NO ID which tuf ended up calling chromogenes
  + All from 1 farm
  + Is this a function of a particular ST, not being well-ID’d by MALDI?
  + None of these were RAPD typed
  + Would need a large number of isolates, with a diversity of MLST identified and represented in this data set, with a good number of isolates belonging to each MLST in order to explore this question
* More work looking at genetic diversity by ST in chromogenes
  + ST6 seems to be the only ST where blaZ carriage was mixed, more diverse group?
  + Looking at dendrogram with publmst isolates, ST6 also seems like most diverse group
* **Virulence or AMR by MLST**: More comprehensive study, using more chromogenes isolates, to understand if a certain strain type is more likely to have particular AMR profiles or virulence capabilities
  + Explore more about diversity at the ST level
    - AMR carriage
    - Persistency
  + Although we did not find any support for an association between carriage of blaZ and the associated SCC category of an IMI, results from the current study suggest that blaZ carriage is likely a function of ST in S. chromogenes. For all but 1 of the 5 MLST identified, blaZ carriage was uniform across a ST. Numerous studies have identified that resistance profiles for NAS are species-specific (Sampimon, 2009; Persson Waller et al., 2011; Taponen et al., 2016; Nobrega et al., 2018; Fergestad et al., 2021; Taponen et al., 2023), so a genetic basis for carriage of particular AMR determinants at the strain level would not be surprising. For S. aureus, carriage of methicillin resistance has been associated with particular clonal complexes both in human medicine (Smith et al., 2021; Garrine et al., 2023) and certain clusters of spa ¬type for bovine clinical mastitis isolates (Freu et al., 2022). Additionally, in a study comparing isolates from persistent and nonpersistent S. aureus IMI, Haveri et al. (2007) found that a particular pulsotype associated more with persistent IMI was significantly more likely to harbor the blaZ gene. An association between genetic grouping and blaZ carriage in S. chromogenes was identified in Persson Waller et. al (2023). In their study, all isolates of ST19, ST102, ST103 carried blaZ. When analyzing clusters of ST, they found that the two clusters comprised primarily of these 3 ST (clusters III and IV) were significantly more likely to be blaZ-positive than other clusters of ST. As isolates belonging to these ST were distributed over different farms and counties in Sweden, the authors suggest that blaZ-mediated penicillin resistance is likely a result of the spread of certain lineages of S. chromogenes, instead of horizontal gene transfer between different strains or species (Persson Waller et al., 2023). Three of the 4 ST which had uniform blaZ carriage in the current study were also distributed over multiple farms.
  + Similar to blaZ, the carriage of VF by isolates in the current study appears to be more a function of phylogeny than a predictor of clinical outcome. The cumulative number of VF identified belonging to the 5 ST with multiple isolates showed little to no variation; total number of VF identified per isolate was uniform for 3 of the 5 ST, 8 of 9 for another ST contained the same number of VF (with the remaining isolate differing by 1 VF), and the remaining ST ranged from 44-48 VF identified per isolate. In a visual assessment of the heat map of VF with isolates organized by ST (Figure 3), many of the limited differences in presence or absence of VF occurred at the ST level. The only isolates lacking fnbA, fnbB, and capN, and possessing coa, capH and capJ both belong to ST25. The only isolates possessing the staphylococcal exotoxin gene set21 were the 2 isolates in ST48. Two isolates of singleton ST (ST177 and ST136) which were not classified as a cluster but were grouped together 42% of the time in the phylogenetic analysis were the only 2 isolates positive for the staphylococcal exotoxin genes set10 and set24. Support for an association between phylogeny and VF presence in S. chromogenes was also found in Persson Waller et al. (2023). When analyzed at the level of ST cluster, isolates belonging to cluster III, IV, and VII had fewer VF compared to isolates belonging to other clusters, and cluster IV had significantly more exoenzyme genes vs. other clusters. At the strain level, they identified ST59 had higher number of adherence genes vs. other ST. The only gene identified to be associated with phylogenetic grouping was atl (autolysin), which was present in clusters V, VI, VII but absent in II, III, and IV (Persson Waller et al., 2023). In the current study, atl was consistently found in all 30 S. chromogenes isolates. When Naushad et al. (2019) applied various clustering approaches in order to determine whether particular VF distributions had any association with SCC category or clinical mastitis, NAS isolates instead grouped together by their respective species. As the pattern of virulence genes carried by NAS isolates likely is species-dependent, a genetic basis for carriage of VF may also extend to the strain level.
  + Our ability to explore if pattern and number of VF vary by ST in the current study was limited both by the relatively small number of isolates assigned to most ST identified, as well as complete uniformity in the number and type of VF carried by a particular ST. The number of S. chromogenes (30) in this study which were submitted for WGS was a function of financial constraint. Future studies with larger isolate collections, isolates collected from a greater number of farms, and the ability to sequence a larger number of S. chromogenes isolates may be better able to explore associations of ARG and VF carriage by ST (as determined by MLST), as larger sample sizes would likely result in a greater diversity of ST and a greater ability to account for the effect of clustering by herd.
* **MLST by geography:** More comprehensive study, using more chromogenes isolates, to understand if a certain strain type predominates geographically (think there HAS been some work on this; but multiple countries/continents)
  + For Huebner et al. (2021), ST1 was the only ST found in all three geographical locations. ST6 was the second most commonly found ST in the current study (9/30 isolates, 30%), and the third most common (15/120, 12.5%) in Huebner et al. (2021). Persson Waller et al. (2023) identified 47 different ST among 105 isolates from Sweden. Huebner et al. (2021) found a similar degree of diversity, with 46 ST identified from 120 isolates from 3 geographical locations. After ST1, ST15 was the second most commonly identified by Hubener et al. (2021), with 17/120 (14.2%) of isolates belonging to this ST. ST15 was primarily identified in isolates from Vermont and Washington State (16/17 isolates), which Huebner et al. (2021) highlight as an example of geographic variation in the distribution of different ST. Interestingly, although all isolates in the current study are from Vermont, only 2 of 30 isolates belonged to ST15
* More exploration of phenotypic trait links
  + linkage between persistency and elevation of SCC?
  + Clinical vs. non clinical chromogenes isolates, compare virulence profiles
  + In a study by Haveri et al. (2005) of 217 S. aureus IMI isolates typed using pulsed-field gel electrophoresis (PFGE), researchers were able to identify that a particular pulsotype was significantly associated with severe clinical mastitis symptoms but reduced persistence when compared to the 4 other commonly identified pulsotypes in the study. This association between a specific genotype and consistent expression of a clinical trait associated with an IMI has not yet been widely described for NAS.

**Comparing organic and conventional farms** (better understanding how organic farms differ from conventional farms)

* Study directly **comparing NASM species diversity** between conventional and organic farms
  + Mine described what’s on organic farms, but wasn’t designed for head to head comparison (with all other things being “equal”)
  + Other studies comparing these two systems didn’t ID staph to species-level
  + The biggest limitation of most studies comparing resistance profiles of mastitis pathogens between organic and conventional farms is that staphylococci were not identified to the species level. Organisms were primarily grouped as either *S. aureus* or “coagulase-negative staphylococci.” Before MALDI-TOF became more widely available, accurate species-level identification of mastitis-associated staphylococci on a relatively large scale was prohibitively expensive and time-consuming.
* Study comparing STRAIN TYPES between organic and conventional farms for chromogenes isolates causing mastitis (all other things being equal); are there different predominant strain types on these management systems?
  + E coli strains were different in gut of calves
  + Comparison of predominant strain types within a given species causing IMI between organic and conventional farms could further our understanding of the complex interplay between phylogeny and selection pressures resulting from management factors on AMR of mastitis pathogens. Although researchers were studying fecal *E. coli* and not mastitis pathogens, Walk et al. (2007) found that phylogenetic groupings varied between organic and conventional dairies, suggesting there may be differences between lineages of *E. coli* in their ability or likelihood of acquiring resistance genes. Based on their findings, the authors conclude that “organic farming practices not only change the frequency of resistant strains but also impact the overall population genetic composition of the resident *E. coli*flora.”
* Study **comparing AMR, virulence** between organic and conventional farms for isolates causing mastitis (all other things being equal)
  + The biggest limitation of most studies comparing resistance profiles of mastitis pathogens between organic and conventional farms is that staphylococci were not identified to the species level. Organisms were primarily grouped as either *S. aureus* or “coagulase-negative staphylococci.” Before MALDI-TOF became more widely available, accurate species-level identification of mastitis-associated staphylococci on a relatively large scale was prohibitively expensive and time-consuming. As resistance profile varies by species, additional work comparing AMR for NASM isolates (while controlling for species) may give further insight into whether resistance profiles differ between management systems for these bacteria.
  + Can this be used to start answering the question, where does resistance in these bacteria come from, and why are they maintaining AB genes in the absence of drug use?
    - Not only about drug use- selective advantage over other microorganisms (fungi, other bacteria)
  + Where are these AMR determinants located in the genetic info? Close to genes transcribing things necessary for survival, so get “carried along?”
  + How “expensive” is it to maintain AMR determinants during replication?
* Study looking at **AMR, over time, from old isolates from organic farms to newer isolates**; trends in AMR presence/absence/diversity over time
  + Additionally, few studies have described resistance patterns of mastitis pathogens before and after transitioning to organic status, and most were limited in both the number of herds enrolled and the amount of time farms were followed. Although likely logistically difficult and expensive, a long-term, larger study of farms transitioning from conventional to organic status would be incredibly valuable in understanding what types of AMR are maintained in organic dairy herds and for how long.
  + Do different types of AMR persist LONGER in dairy farm environment?
    - In almost all studies summarized in this review, some degree of AMR was found in isolates despite decreased (EU) or absence (US) of selective pressure of antimicrobial use; organic farms in McDougall et al. (2021) had no antimicrobial usage for a range of 7-19 years, with a median of 12 years of organic certification. Assuming there is a fitness cost to bacteria for maintaining AMR genes (Vanacker et al., 2023), this certainly begs the question of why resistance genes have been maintained to any degree in the absence of selective antimicrobial pressures. A rather extreme example of AMR persistence in cattle farms is a study comparing bacteria isolated from retail ground beef raised in conventional and “raised without antibiotics” operations. LeJeune and Christie (2004) identified resistance against chloramphenicol in isolates from both systems, an antimicrobial that had been banned from use in US food animals since 1986.
  + Some could be linked to locally advantageous traits and when that fitness advantage leaves bc something in the env changes, then would expect those to disappear
    - In a study where feedlot steers were fed subtherapeutic levels of antibiotics, Alexander et al. (2008) found that ampicillin-resistant *E. coli* in the control group (no antibiotics) increased due to an evident clonal expansion of an environmental strain (detected by PFGE) during the latter part of this longitudinal study. This environmental strain outcompeted other strains of *E. coli* present in the intestinal tract of the steers in the control group, suggesting that fitness traits beyond carriage of AMR genes play an important role in the prevalence of AMR bacteria. Specifically, the authors suggest that one environmental factor related to the level of AMR was diet, as the prevalence of steers shedding tetracycline-resistant *E. coli* was higher in animals fed grain-based vs. silage-based diets in both treatment and control groups. Although specifically looking at commensal *E. coli* in dairy calves and not mastitis pathogens, one group of researchers set out to explore which factors beyond antimicrobial usage may explain the persistence of an *E. coli* strain (SSuT) in the GI tract which was resistant to streptomycin, sulfonamide and tetracycline (Khachatryan et al., 2004, 2006a, 2006b, 2008; as summarized in Call et al., 2008). Their first study asked if direct antimicrobial selection pressure was maintaining a high prevalence of SSuT *E. coli* strains in calves, and they found that it was not; a clinical trial showed that addition or removal of oxytetracycline from the diet had no effect on the prevalence of SSuT strains in fecal samples over a period of 3 months. Their next step was to ascertain if SSuT traits themselves provide a secondary but unrecognized fitness advantage to these particular strains of *E. coli* by generating null mutants for the SSuT traits (now susceptible to these antibiotics). On average, they found that the null mutant strains retained a competitive advantage over the other susceptible strains, and concluded that the specific genes conferring the SSuT phenotype were not responsible for providing any secondary fitness advantages. At some point between studies, the farm stopped feeding a medicated milk replacer. The researchers observed that after only a short time frame, the SSuT strain had suddenly declined in prevalence. This was unexpected, given that their previous work demonstrated that the SSuT strains had an obvious advantage compared to the susceptible strains. This unexplained decline prompted an additional study, which hypothesized that the milk supplement itself (comprised of dried milk powder, vitamin A and D) was somehow providing an advantage to the SSuT strains. When the milk supplement was reintroduced (both with and without tetracycline), the prevalence of SSuT *E. coli* strains nearly doubled for both groups of animals receiving the milk supplement vs. those that received none. This work highlights an example of a positive selective force (a dietary supplement) in a dairy farm system either directly or indirectly favoring strains of resistant *E. coli,* which was completely unrelated to antimicrobial exposure.
  + Ones that were energetically expensive to maintain and conferred no advantage would expect to see disappear fast
    - Call et. al (2008) summarize the 3 possible outcomes after exposure to antimicrobials in an individual animal produces a transient increase in AMR prevalence in a population of bacteria, as has been documented to occur in fecal bacteria. Once the negative selective pressure of antimicrobial usage is removed, the first possible outcome is subsidence of AMR in the population, assuming there is a fitness cost to maintaining the AMR traits. Alternatively, if there is no additional fitness cost to maintaining AMR, we would expect to see “eventual displacement in the face of natural turnover of clonal types at the level of individual animals.” A third possibility, as seen in the work from Khachatryan et al., is that there is no (or limited) change in the level of AMR prevalence after selective pressure from antimicrobials is removed. This could occur if AMR traits have been coupled with other some other locally beneficial traits which provide the bacteria possessing them an advantage in their specific environmental niche. Call et al. (2008) illustrate this with a hypothetical model illustrating the effect of antimicrobial exposure in an individual animal (Figure 1). First, a transient increase occurs in the relative number of resistant bacteria within a population after exposure to an antimicrobial. During this time of increased replication, there is an increased probability for a genetic event to occur which links AMR carriage to some other trait providing increased fitness in that specific environment. Organisms with the linked AMR carriage and locally advantageous trait survive better in the population, but in the absence of antimicrobial exposure, there is nothing to actively suppress the susceptible strains in the population. Although the relative proportion of bacteria with AMR may decline gradually over time, linkage of AMR to some other advantageous trait could also lead to a gradual increase or maintenance of a baseline prevalence of AMR, even in systems devoid of antimicrobial exposure. So far, work exploring this question has been limited to studying the effect of antimicrobials on resistant bacteria present in the GI tract of cattle. The potential exists for research focused on exploring why maintenance of AMR genes occurs in mastitis pathogens from organic dairies, years after the selective pressure of antimicrobial use has been removed.
  + May vary by type of carriage – on circular genome? Plasmid?
  + Compare VERSIONS of blaZ present in staphylococci from a dairy 30ish years ago and currently to better understand if particular versions of ARG maintained
    - blaZ – might make sense from my findings that that ST seemed to determine carriage IF it was chromosomally carried
      * + future direction: where is blaZ carried in these chromogenes isolates
        + In 26 out of 34 Finnish isolates (76.5%) and in 25 out of 44 Swedish isolates (56.8%) the *blaZ* gene was localized on a plasmid. Six different protein signatures were found.
        + why penicillin-resistance is clearly more common in Finland than in the neighbouring Nordic countries with similar conditions for milk production. One explanation could be the more common plasmid location of the *blaZ* gene and plasmid-mediated spread of penicillin-resistance.
        + Genetic basis of penicillin resistance of S. aureus isolated in bovine mastitis; Arzu Funda Bagcigil,1,2 Suvi Taponen,corresponding author1 Joanna Koort,3 Björn Bengtsson,4 Anna-Liisa Myllyniemi,5 and Satu Pyörälä1
      * ***from bagcigil paper:***
        + Resistance to benzylpenicillin is mainly caused by the *blaZ* gene encoding production of beta-lactamases, which hydrolytically destroy beta-lactams [[13](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B13)]. The *blaZ* gene can be located chromosomally or on plasmids [[14](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B14)]. This type of penicillin resistance in *S. aureus* may thus emerge via two mechanisms: spread of resistant clones or through horizontal dissemination of mobile elements containing the *bla*Z gene [[15](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B15),[16](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B16)]. Location of the resistance determinants on transferable elements generally promotes efficient spread [[16](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B16)]. In Denmark the *blaZ* gene of penicillin resistant *S. aureus* isolates has been predominately located chromosomally [[17](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B17)]…. ***Possession of the blaZ gene was partly linked to pulsotype, which may indicate a clonal spread of resistance.***
        + Certain genotypes of mastitis causing *S. aureus* can become dominant in the dairy herds [[25](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B25)]. In the three most common pulsotypes here *bla*Z-negative isolates were over-represented, indicating that penicillin-resistance was partly related to pulsotype. An association between certain pulsotypes and penicillin susceptibility has also been shown previously [[32](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B32),[33](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B33)]. Penicillin-resistance may be linked to other virulence factors of *S. aureus*, which may facilitate the spread of resistant clones [[33](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B33)]. Intramammary infection remained significantly more often chronic if it was caused by *bla*Z positive (61.0% remained persistent) than *blaZ* negative (25.0%) strains [[34](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575348/#B34)].
  + Likely drug dependent – in my isolates, seems like carriage of blaZ was genetic – based more on ST with limited data

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Table 1. Agreement between *blaZ* gene carriage and phenotypic susceptibility to penicillin by strain type (ST; as determined by MLST) for 30 *Staphylococcus chromogenes* isolates associated with persistent bovine intramammary infections. N = no *blaZ* carriage; Y = positive for *blaZ* carriage; S = susceptible to penicillin; R = resistant to penicillin. | | | | |
| Isolate | ST | ST cluster | *blaZ* carriage | Susceptibility phenotype (penicillin) |
| assembly\_3 | 175 | 1 | **N** | S |
| assembly\_2 | 174 | 1 | **N** | S |
| assembly\_20 | 176 | 1 | **N** | - |
| assembly\_21 | 176 | 1 | **N** | - |
| assembly\_22 | 176 | 1 | **N** | - |
| assembly\_23 | 176 | 1 | **N** | - |
| assembly\_14 | 176 | 1 | **N** | - |
| assembly\_15 | 176 | 1 | **N** | - |
| assembly\_16 | 176 | 1 | **N** | - |
| assembly\_17 | 176 | 1 | **N** | - |
| assembly\_19 | 176 | 1 | **N** | - |
| assembly\_10 | 5 | 5 | **Y** | - |
| assembly\_24 | 5 | 5 | **Y** | - |
| assembly\_12 | 5 | 5 | **Y** | R |
| assembly\_1 | 6 | 6 | **N** | S |
| assembly\_6 | 6 | 6 | **N** | S |
| assembly\_9 | 6 | 6 | **N** | S |
| assembly\_13 | 6 | 6 | **N** | S |
| assembly\_30 | 6 | 6 | **N** | - |
| assembly\_25 | 6 | 6 | **Y** | - |
| assembly\_26 | 6 | 6 | **Y** | - |
| assembly\_27 | 6 | 6 | **Y** | - |
| assembly\_29 | 6 | 6 | **Y** | - |
| assembly\_5 | 25 | 25 | **N** | S |
| assembly\_7 | 25 | 25 | **N** | S |
| assembly\_4 | 48 | 48 | **Y** | R |
| assembly\_11 | 48 | 48 | **Y** | R |
| assembly\_8 | 51 | 51 | **Y** | R |
| assembly\_28 | 136 | 136 | **N** | - |
| assembly\_18 | 177 | 177 | **N** | - |