***Notes for conclusion (May 2024)***

* Notes from Committee Meeting 2024 about **Conclusion section**
  + What are the *big take-home messages* from each manuscript
  + What’s *new* to science? What have *you* contributed?
  + What kind of directions could people go in; based off your research findings; from where you left off with your work; what are both near and far/short and long-term *next steps to continue the research questions* covered in your thesis (both others in your lab, and bigger picture)
    - What are the *next steps* following your research, both in your lab and more grandiose?
  + What are pieces of *actionable information* gained from your thesis, and what do we need to get there?
  + Speciation of NASM could be important with regard to antimicrobial susceptibility; are there certain species that have more or less resistance, does knowing susceptibility pattern inform decisions on how producers (or advisors) treat cases of species-specific NASM mastitis?
  + What is the *benefit of your work* to the dairy industry generally?
  + Acknowledge any *challenges or issues you see in the field*
  + What lessons do we learn from your work – this can include what NOT to do!
    - What were some things that you tried, that didn’t work – can you save someone else the work of trying the same thing that won’t work?
  + Conclusions will be similar to your “Discussion” sections of manuscripts… kind of *the long version* of it
* Future directions
  + Study directly comparing 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”)
  + Study comparing AMR, virulence between organic and conventional farms for isolates causing mastitis (all other things being equal)
    - 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 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?
  + Study looking at AMR, over time, from old isolates from organic farms to newer isolates; trends in AMR presence/absence/diversity over time
    - Do different types of AMR persist LONGER in dairy farm environment?
    - 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
    - Ones that were energetically expensive to maintain and conferred no advantage would expect to see disappear fast
    - 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
    - Likely drug dependent – in my isolates, seems like carriage of blaZ was genetic – based more on ST with limited data -
  + 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
  + 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
  + Clinical vs. non clinical chromogenes isolates, compare virulence profiles
  + 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)
* Notes from 7.29.24
  + 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
  + MALDI identification
    - There were 4 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
  + Explore more about diversity at the ST level
    - AMR carriage
    - Persistency
    - Like we had written for DIM SCC paper, is there some unknown linkage between persistency and elevation of SCC?
* 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
* Virulence and AMR manuscript
  + Look where blaZ carried
  + Test association of increased SCC and number of vir factors BY CATEGORY (like Naushad)
  + Test for phenotypic resistance to penicillin, include that- corresponds to blaZ carriage?
  + Analyze vwp and coagulase results (and coa) – genotype and phenotype link