01.25.2017

\*Info update presenters must update glossary with relevant words before lecture

**Info Update:**

1. What are QTN/QTN Programme?
   1. QTN – **q**uantitative **t**rait **n**ucleotides
      1. Individual SNPs that add to the variation and may be under the influence of selection
      2. Ex: flowering time, color, thermal tolerance, venom potency, defense compounds, drought tolerance, altitude tolerance (continuous traits, NOT discrete/Mendelian)
      3. Mendelian traits are major-effect traits while QTNs are quantitative traits
      4. Alpha – average effect
2. Quantitative genetic theory of adaptive traits
   1. VA=Sum(αipiqi)
   2. h2=VA/Vp
3. Methods
   1. Linkage mapping (QTL mapping)
   2. GWAS
   3. Selection scans

**Rockman, 2011**

* Rarely sampling from the pool
* Looking for tiny gold pieces (analogy)
* 3 basic arguments
  + QTNs to date are large-effect alleles/Mendelian (biased in ignoring small-effect alleles)
  + Theory doesn’t support large-effect alleles being the basis for adaptive evolution
  + Small-effect alleles do not act the same as large-effect alleles
* Asking the right questions is the most important thing
  + Forward vs. reverse approach (Forward approach may be less biased)
  + Don’t just do it *because*, have formulated questions, hypotheses, etc.
  + When do you need to know the genetic mechanisms vs. when can you use alternative studies
  + (Dough Schempske paper – speciation)
* Are these findings showing interaction?

**Rockman, 2011 vs. Lee et al. 2014**

**Lee et al. 2014**

**-**These tools should be used in conjunction with other tools (different types of study)

**-**Still a lot of questions and results that can be addressed that haven’t been yet

**-**potentially a hypothesis-generating approach

**Project (Data) Overview: (info on BB)**

Seastar wasting disease:

* 50-100% mortality (typically in the 90% range)
* 1970s and 1980s found in isolated cases
* 2012 first bout, increased in severity since
* 2016 less severe
* Disease has never been this severe and pervasive
* Some species more resistant to SWD
  + Presentation of pathogens similar across species
* Progression of disease occurs within hours
* Limbs fall off, insides explode out, etc.
  + Begins with loss of turgor or lesions
  + Limbs pull off (behavioral/gravity)
* Pathogen is not known
  + **ssDNA virus**
  + No viral load studies
  + Densovirus implicated (Hewson et al. 2014 PNAS)
  + Present in 70 year old museum speciments
  + All individuals have been exposed to the virus (AK to CA)
  + Could be multiple viruses
  + Symptoms also occur when they die (maybe can’t activate catch collagen)
  + Healthy and sick in same populations
  + Slight association between SWD and warmer water temperatures
  + Epidemics peaks during warmest months
  + Appears more prevalent in interdially than subtidally (warmer water, less constant🡪stressor)
    - Potentially greater transmission in INT because of water current
  + Could most vulnerable species be large predators?
* Focusing on: *Pisaster ochraceus*
  + Purple and orange
  + Found from Alaska to Baja, California
  + Vulnerable species (not as vulnerable as the sunflower star)
  + Cannot maintain them in lab (die)
  + Found in both tidal zones and generally the same diet (albeit maybe in different proportion)
* What is causing the tipping point?
  + Disruption in environment
  + Microbiome
* Our samples:
  + *Pisaster ochraceus*
  + Our samples collected in Monterey area
* Hypotheses:
  + Genetic difference between seastars in interdal vs. subtidal and related to susceptibility
  + Gene expression differences between tidal regions
  + Microbiome (H v S)
  + Microbiome vs time
  + Relatedness v microbiome
  + Expression of immune response GE
    - E.g. RNAi
    - Reverse pathology