PMI 214 Notes - Final Review with Greg Lanzaro

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• Lec. 11

- EIR: entomological innoculation rate
 - * EIR = $ma\phi\theta$ where
 - \cdot m How many mosquitoes out there? mosq/person
 - · a How many bites? bites/day
 - $\cdot \phi$ How many bites are on humans (how anthropophilic)? human-bites/all-bites
 - \cdot θ How many vectors are infected? transmitting-vectors/all-vectors
- Vectorial Capacity number of infective vector bites that would arise from all the mosquitoes that bite a single host on a single day $C = \frac{ma^2b}{-\ln n}p^n$
 - * a bites/person/day (not the same a as in EIR)
 - * b vector competence
 - * n extrinsiv incubation period
 - * p vector daily survival rate
- Basic Reproductive Rate (R_0) avg. # of future host infections that will arise following introduction of a single infectious host in a susceptible population $R_0 = C/r$ where
 - * r host recovery rate 1/infectious-perioud

• Lec 12

- Integrated Vector Control (IVM) think about all the availble tools for the specific vector and situation "a rational decision-making process for the optimal use of resources for vector control"
- Behavioral Insecticide Resistance ex: mosquitoes feeding over a range of times, kill all the ones who feed at night, going to select for ones who don't feed at night, bed nets stop being effective.
- Source reduction getting rid of some feature of the environment which the mosquito needs filling potholes, getting rid of standing water, covering drainage systems, getting rid of carbohydrates, unneeded vegetation, draining swamps

• Lec 13

- GMM for vector control
- population suppression vs. population replacement
 - * suppression reduce fecundity of the population by killing them or getting rid of all the females
 - * replacement introduce mosquito resistance to malaria through gene drive (turn the vectors into nonvectors)
- transgene (in the lab)
 - * need to have an effector gene (affects the phenotype that kills parasite)
 - * need to have a promoter (turns the gene on at the right time (larval stage, adult stage) in the right place (salivary gland, epidermis lining the stomach, , sex specific, etc))
- transgene in nature
 - * all of the above, and a gene drive (not normal Mendelian inheritance)
- How does a gene drive work?
 - * progeny has one copy of transgene and one copy of wild type.

- * gene drive cuts the wild type gene and repairs it with the transgene.
- * heterozygote turns to homozygote
- * ALL future progeny carries the transgene

• Lec 14

- Ecology of Rickettsiaceae
- hard and soft tick feeding behavior
 - * hard ticks feed once per stage
 - * hard ticks have different hosts for each stage
 - * hard ticks feed for a week at a time they cement themselves to the host, until it gets completely full
 - * soft ticks feed for 20 min at a time
- Rickettsiaceae
 - * bacteria
 - * live inside the cell not exposed to the host immune system
- How do horses get infected with Neorickettsia?
 - * acquatic insects on wet grass (Carter: "I think it's... wet food?")

• Lec 15

- Wolbachia
 - * Wolbachia is very common bacteria in insects often symbiotic
 - * transmitted maternally (vertical transmission)
 - * also horizontally transmitted (between different species) mechanism not understood but good be good for mosquito control, but could cause problems (evolution people say horizontal transmission is between species, not necessarily within species)
- Cytoplasmic incompatibility uninfec/infec female with uninfec/infec male different results.
 - * infected female with whoever, all infected, normal progeny numbers
 - * infected male with uninfected female, all uninfected, reduced progeny numbers
- How are Wolbachia spread?
- Bartonian vs. Fisherian waves
 - * Fisherian (1937) pulled more robust spreading
 - * Bartonian (1979) threshold bistable, pushed easily stopped by barriers to dispersal

• Lec 16

- genome evolution in malaria vectors in response to vector control
- what is a sibling species?
- "genomic island of speciation" in these islands are genes responsible for reproductive isolation
- adaptive introgression is one gene moving into another which imparts a fitness advantage
- shows up in Anopheles coluzii and An. gambiae
 - * movement of a good (adaptive) gene from one species to another, or one diverged population to another
 - * what was the gene that moved from coluzii to gambiae? insecticide resistance.
- selective sweep
 - * if selection is operating on a gene, there is a loss of heterozygosity around that gene

• Lec 17

- Leishmaniasis
- visceral, cutaneous, mucocotaneous
- sand flies
- makes the sand flies regurgitate, making it feed more
- sand fly saliva induces blood flow, preventing coagulation, and suppresses the immune system

• Lec 18

- mechanisms of arbovirus emergence
 - $\ast~1992\mbox{-}93$ vs 1996 outbreaks of VEEV
 - \cdot 92-93 mutation in the virus that changed the pathogenicity to the horses
 - \cdot 1996 caused a switch in the vector making it a really good vector
 - * common causes of alphavirus outbreaks?
 - · either alter pathongenicity, or
 - \cdot alters the vector
 - \ast what determines how frequently these outbreaks occur? environmental conditions
 - * competitive fitness assays?
 - \cdot co-infect a host with a mixture and see which one wins in the individual
 - \cdot if one is over-represented, it is more fit.