

EVOLUTION of VIRULENCE

recognition vs effectors
(RQ)

↑
virulence (parasite)
tolerance } (host)
resistance }

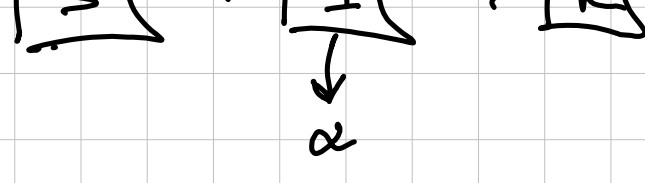
VIRULENCE

- loss of host fitness due to parasite infection -

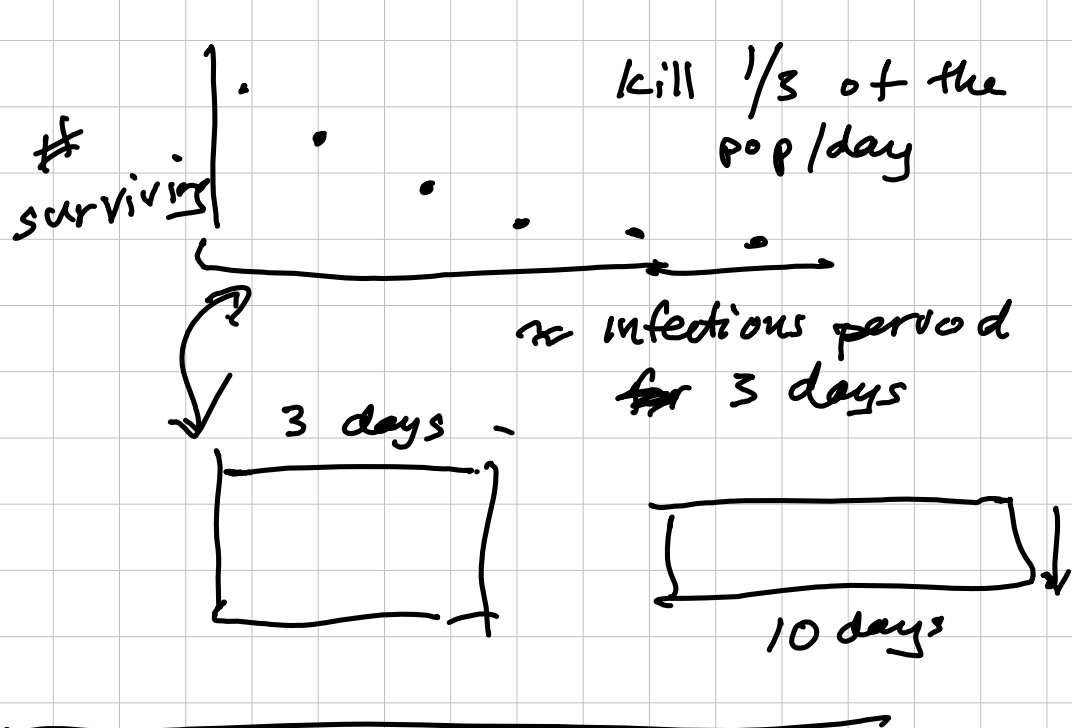
- host killing
- loss of fecundity
- castration
- loss of resources

PROXIES

↳ narrow-sense
rate of host-killing



≠ case fatality rate.
infection



transmission rate ↔ virulence rate
↙ ↘
parasite load / replication rate / use of host resources

Classical dogma (1970s)

parasites always evolve toward commensalism (lower virulence)

- newer host-parasite associations would be more virulent

Syphilis

1495 'great pox'

over 50 years, virulence decreased.

? host evolution?

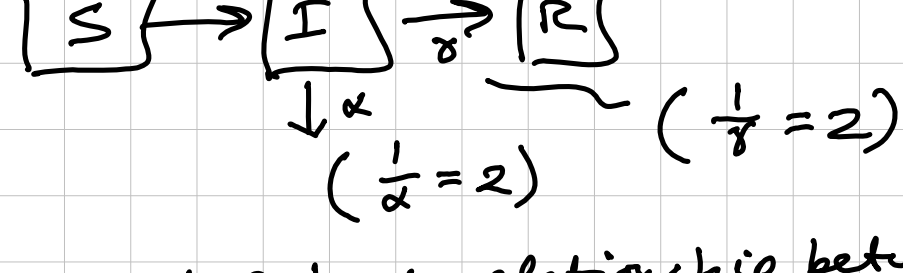
group selection

tradeoff theory
host-level selection

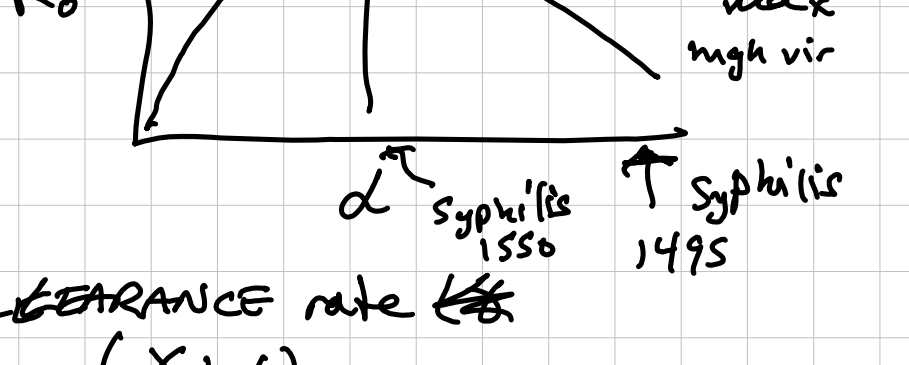


high relatedness

$R_0 = \text{transmission rate } (\beta) \cdot \text{infectious period } \left(\frac{1}{\alpha + \gamma}\right)$



LEADS to a relationship betw R_0 and α with an intermediate max



CLEARANCE rate $(\gamma + \alpha)$

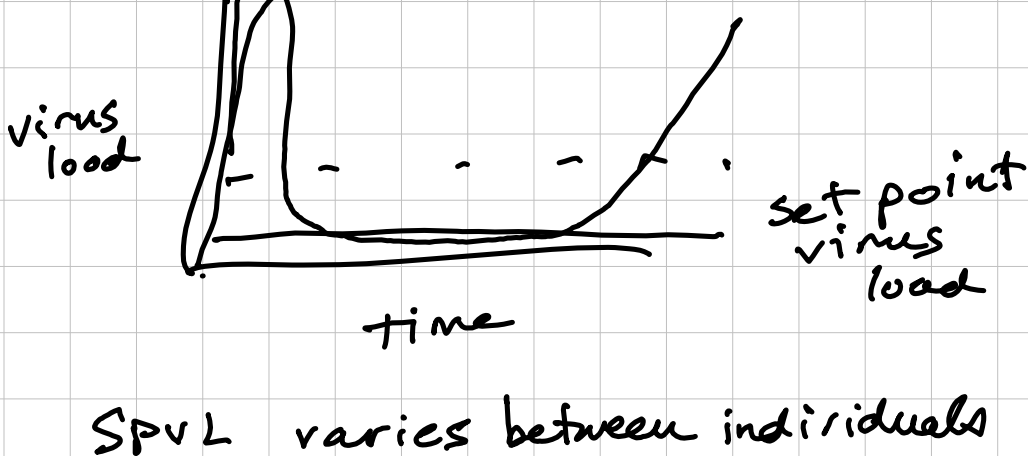
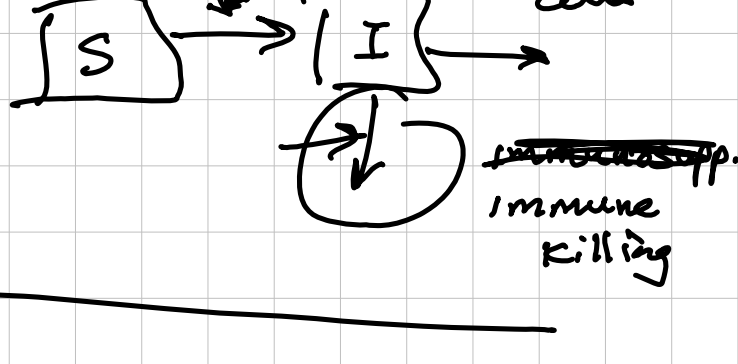
classical dogma: from sampling bias?

MECHANISM

- myxo: Australia, Europe

genomic analysis

- lower replication rate?
- immunosuppression
- 'deleterious' mutations



SPVL varies between individuals

RAKA1, Uganda

measured SPVL

- time to transmission
- time to progression

classical dogma → tradeoff theory → ??
1970s ~ 2000

within-host competition



mutation? superinfection
increases virulence

Short-sighted evolution

- paralytic polio strains
- CNS - dead end

in HIV