

How to give a talk

RSV vaccination as an example



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Preparation

- * Your academic talk is the showcase of your work
- * People are more likely to be exposed to your talk than to read your paper
- * It also showcases your professionalism
 - your reputation will matter in the future
- * Think of it as the big-budget movie version of your paper
 - educate, enlighten and entertain.

Creation

- * Do not create your talk at the last minute
- * NEVER make it during the conference
 - it's rude
 - pay attention to other talks as you would want others to pay attention to yours
 - you will also not know the timing or if some constructions don't work.

Spacing

- * Do not clutter your slides
 - do not use fancy colours or backgrounds
 - simpler is better
- * If you have too much to say on a page, make it two pages
- * Keep points crisp and short
 - no bullet should be more than three lines
- * Pictures, pictures, pictures
 - avoid “reading out” as much as possible.

Biological introduction

- * Be sure to start with something accessible
- * Spend time on the biology
 - a lot of time
- * Explain the background and issues
 - what is the disease?
 - what intervention are you considering?
 - what are the complications?
- * A non-math expert should still understand what problem you are trying to solve.

The Model

- * Introduce the model with a flow diagram
- * Explain everything visually
- * Then write down the equations
- * All parameters should be explained on every page
- * State assumptions clearly
- * The model is the major thing you are creating
 - it needs to be set up carefully.

Analysis

- * Only write down the key results
- * Generally, don't include the details
 - unless you need them elsewhere in the talk
- * Pictures whenever possible
- * Use LaTeXiT to import equations
 - beware of copy and paste signifiers.

Numerical simulations

- * Your pictures tell the story
- * You want a narrative
- * Label everything clearly
- * You can use the slide transition to convey information
- * Use colour
- * Summarise key results in words.

Summary

- * Be concise
- * Identify what we have learned that we didn't know
- * List your limitations
- * Don't introduce new results
- * No equations here, only words.

Conclusion

- * One page only
 - What did we study?
 - Why?
 - What did we learn?
 - What are the recommendations?
- * The audience will see many talks and remember very little
- * You only get to have a few takeaways
 - make them count.

Final page

- * Include a reference to your paper
- * Collaborators can be thanked verbally
- * Include a link to your website with all your publications as free pdfs
 - you have the right to host your work
 - should be accessible to people in developing countries
- * The more easily people can find your work, the more they're likely to read it
 - and cite it.

Worked example

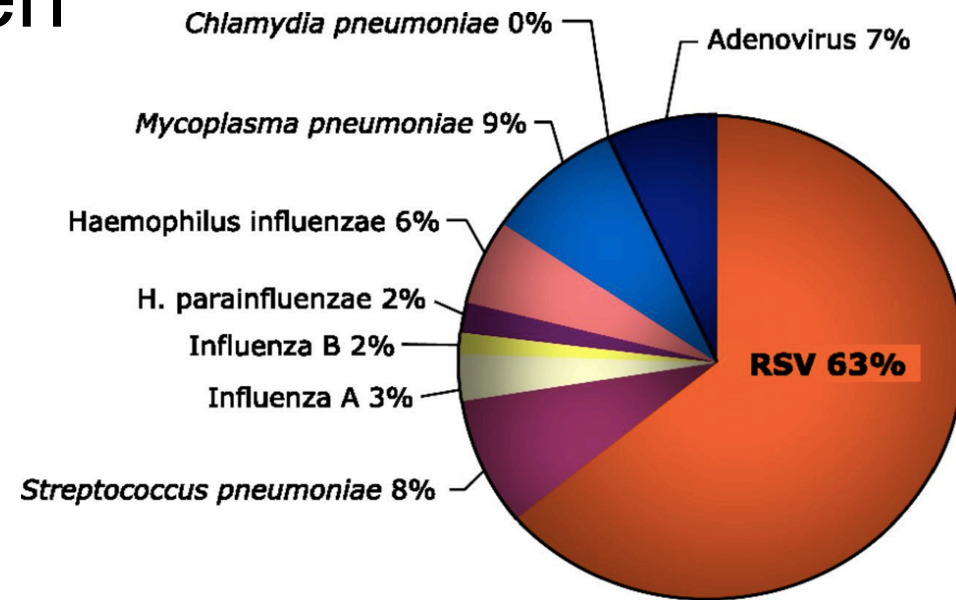
- * RSV vaccination talk
- * Built from the paper
- * But focused on being a talk
- * The talk is visually rich and not exhaustive
 - if they wanted to read the paper, they would
- * Key points highlighted
(not every slide is included).

Respiratory Syncytial Virus (RSV)

- The main cause of acute lower respiratory infections in adults and young children
- Almost all children have been infected by age 2
- About 0.5–2% of infants require hospitalisation due to infection
- In 2005, 3 million new episodes of RSV occurred in children under 5 worldwide.

Animation keeps the audience awake

A period shows my last slide on each page.



Etiology of acute respiratory infections in children.

Symptoms

Express information
consisely

- Mild symptoms:
 - cough
 - runny nose
 - sore throat
 - earache
- Major symptoms:
 - difficulty breathing
 - blue lips
 - lack of energy
 - bronchiolitis
 - pneumonia.

...but they
shouldn't distract.

Pictures add
flavour...



Burden of RSV

- Highest number of observed cases occurs in children aged six weeks to six months
- Morbidity occurs in $<0.1\%$ of cases
- Immunity is short-lasting
- Reinfection is common
- Healthcare costs are substantial
- Infection occurs throughout adult life
 - often a cause of mortality in the elderly
- RSV is a significant economic and healthcare system burden.



Sub-points add extra info, break up the text.

Seasonal patterns

Seasonality informs the model.

- In temperate climates, RSV epidemics exhibit consistent seasonal patterns
- Most infections occur during winter months, whether wet or dry
- Outbreaks typically last 2–5 months
- In tropical climates, RSV is detected throughout the year, with less pronounced seasonal peaks
- The onset of RSV is typically associated with the rainy season.



Prophylaxis

- Immunoprophylaxis with the monoclonal antibody Palivizumab has proven effective in reducing the severity of symptoms

However, it cannot prevent the onset of

Treatment exists but is not a viable option.

- very expensive
- \$1416.48 for a 100mg vial
- generally only administered to high-risk children.



Vaccination

Vaccination is most promising.

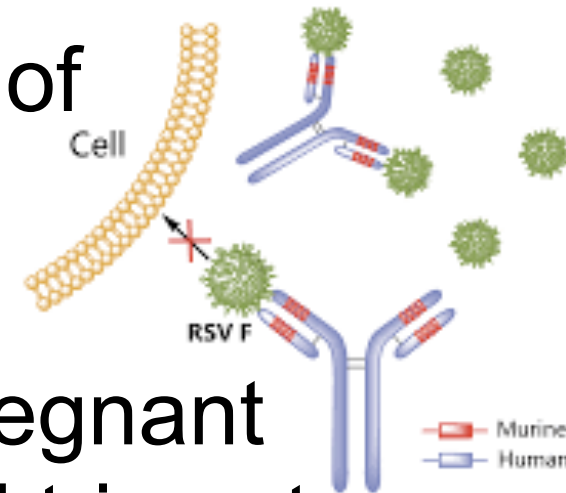
- Research has focused on the development of particle-based, subunit and vectored vaccines
- Several such vaccines are being evaluated in clinical trials
- Other vaccines are in pre-clinical development
- Live attenuated vaccines are also undergoing Phase I trials.



Model 1

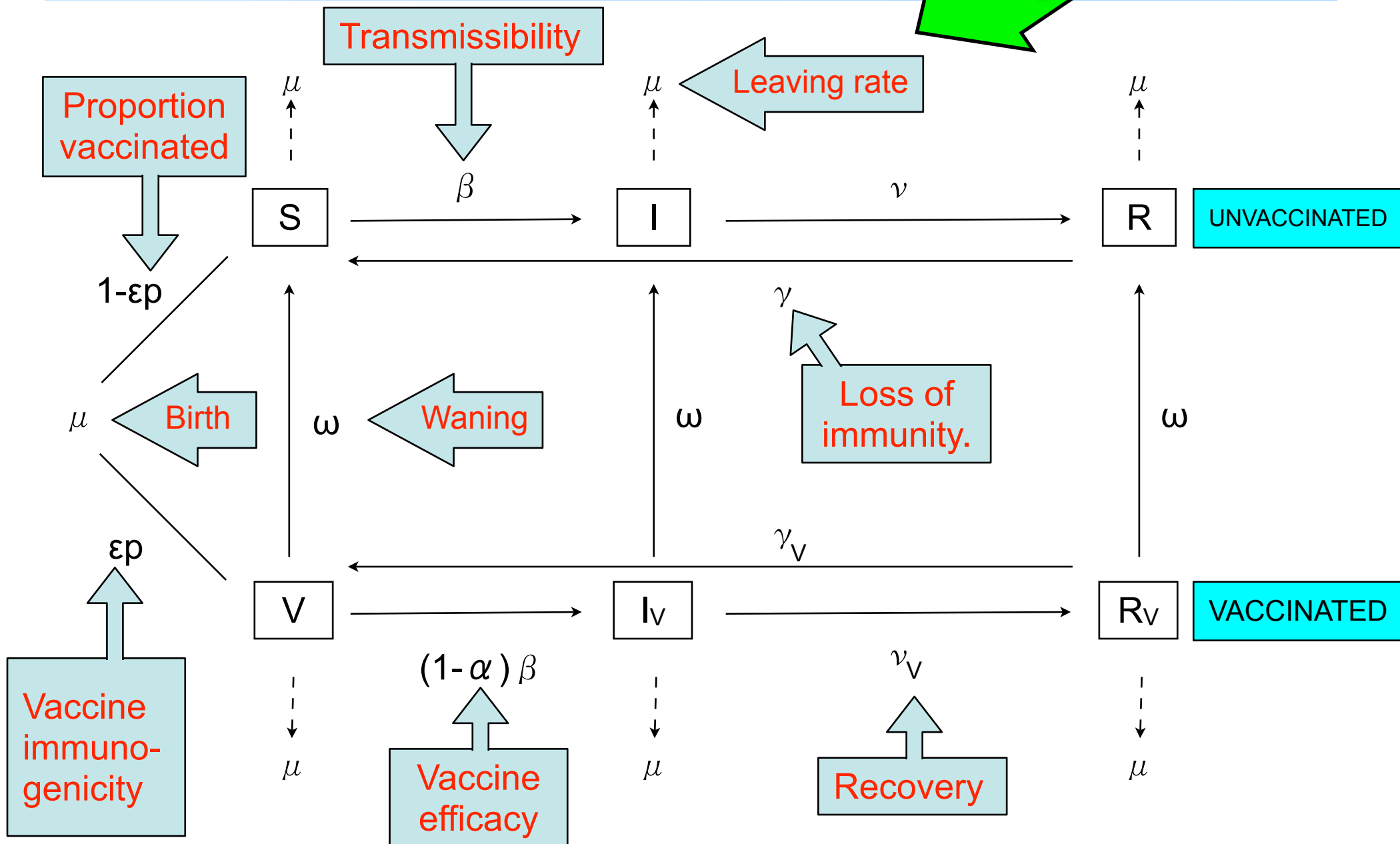
Model assumptions.

- We extend an existing RSV model for a single age cohort to include vaccination
- We first assume a fixed proportion of individuals entering the model are temporarily immune to infection
- This reflects the situation where pregnant women are vaccinated in their third trimester
- Protective maternal antibodies are transferred placentally to the unborn infant
- This confers protection for the first few months of life.



Flow diagram

Every term is explained.



The continuous model

- The model with vaccination is

Indicates another model to come

$$S' = \mu(1 - \epsilon p) - \mu S - \beta(t)S(I + I_V) + \gamma R + \omega V$$

$$I' = \beta(t)S(I + I_V) - \nu I - \mu I + \omega I_V$$

$$R' = \nu I - \mu R - \gamma R + \omega R_V$$

$$V' = \epsilon p \mu - \mu V - \beta_V(t)V(I + I_V) + \gamma_V R_V - \omega V$$

$$I'_V = \beta_V(t)V(I + I_V) - \nu_V I_V - \mu I_V + \omega I_V$$

$$R'_V = \nu_V I_V - \mu R_V - \gamma_V R_V + \omega R_V$$

Parameters are footnoted every time.

with $\beta(t) = \beta_0(1 + \beta_1 \cos(2\pi t + \varphi))$

and $\beta_V(t) = (1 - \alpha)\beta(t)$

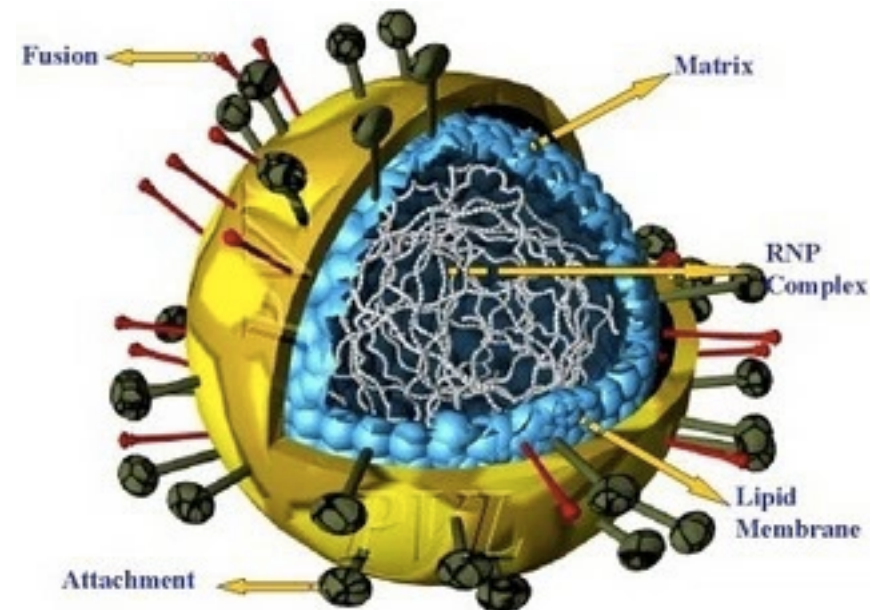
(α may possibly be negative).

*S=susceptible I,I_V=infected
R,R_V=recovered V=vaccinated
 μ =background death ϵ =efficacy
 p =coverage ω =waning
 β, β_V =transmissibility
 ν, ν_V =recovery γ, γ_V =loss of immunity*

Key assumptions

- We assume
 - the leaving rate is unchanged across all classes
 - no disease-specific death
 - entry and leaving rates are scaled so the population is constant
 - transmissibility oscillates seasonally.

Ethically, we need to state the assumptions.



Constant transmission

There is a DFE satisfying

Results are stated,
details omitted

$$(\bar{S}, \bar{R}_V) = \left(\frac{(1 - \epsilon p)\mu + \omega}{\mu + \omega}, 0, 0, \frac{\epsilon p \mu}{\mu + \omega}, 0, 0 \right)$$

- We can prove that this equilibrium is stable if

$$\lambda^2 + b_1 \lambda + c_1 = 0$$

has roots with negative real part, where

$$b_1 = -\beta \bar{S} + \mu + \nu - \beta_V \bar{V} + \nu_V + \mu + \omega$$

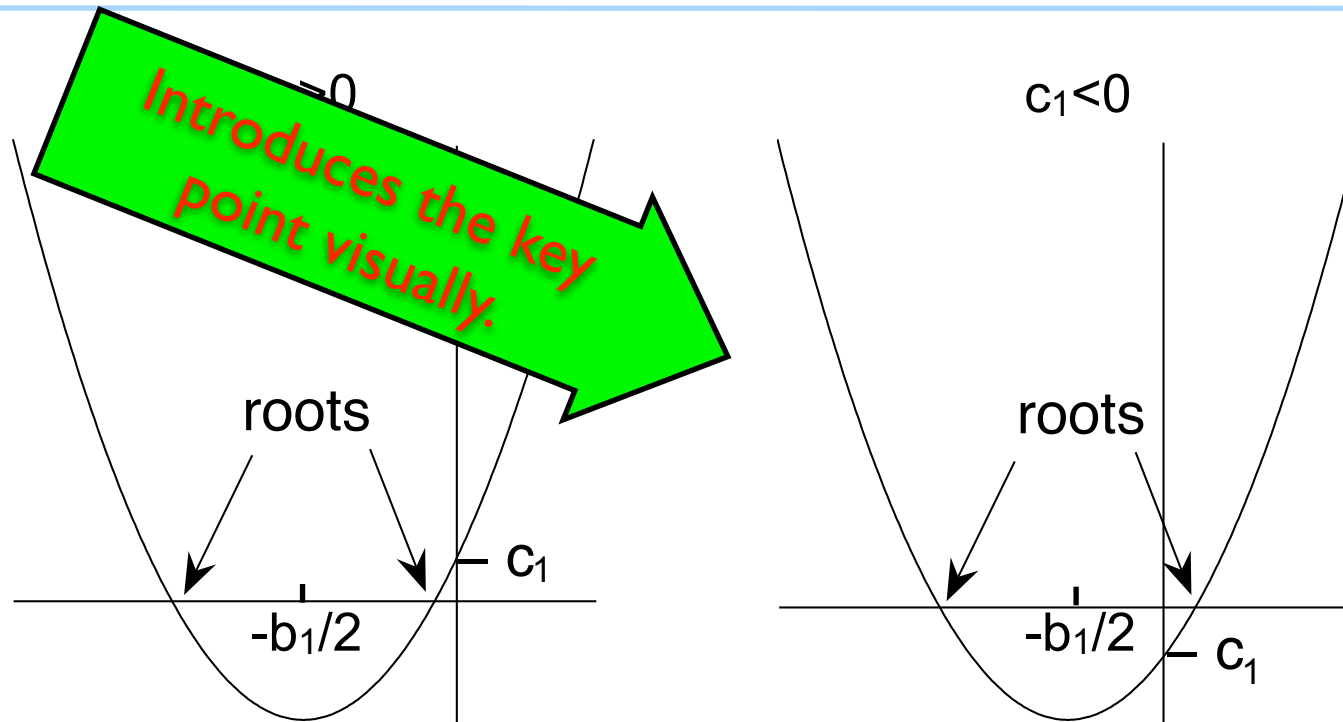
$$c_1 = (\beta \bar{S} - \mu - \nu)(\beta_V \bar{V} - \nu_V - \mu - \omega) - \beta_V \bar{V}(\beta \bar{S} + \omega)$$

$$= \beta \bar{S}(-\nu_V - \mu - \omega)(\mu + \nu)(\beta_V \bar{V} - \nu_V - \mu - \omega) - \beta_V \bar{V} \omega.$$

Audience won't remember
definitions from earlier pages.

*S=susceptible I=infected R=recovered
V=vaccinated susceptible
I_V=vaccinated infected R_V=vaccinated
recovered μ =background death
 ϵ =efficacy p =coverage ω =waning*

Stability of eigenvalues



- If $b_1 > 0$, then c_1 is a proxy for the eigenvalues
- If $b_1 < 0$, then the DFE is unstable and c_1 is not a threshold.

$b_1 = \text{vertex}$
 $c_1 = \text{intercept}$

Basic reproduction number

- Rearranging the constant term leads to

$$R_0 = \frac{\beta_S \bar{S} + \beta_V \bar{V} (\mu + \nu + \omega)}{\mu + \nu_V + \omega}$$

Standard result, to contrast with...

- If $c_1=0$ and $b_1>0$, then we have a bifurcation with the property that the DFE is stable if $R_0<1$ and unstable if $R_0>1$ (as desired)
- However, it is possible that when $c_1=0$, $b_1<0$
- In this case, R_0 is not a threshold, and the disease can persist if $R_0<1$.

...non-standard result

S =susceptible V =vaccinated
 μ =background death ω =waning
 β, β_V =transmissibility
 ν, ν_V =recovery

Positive vertex

Details included only
for original material

- When $c_1=0$, we

$$b_1 \Big|_{c_1=0} = \frac{1}{\nu_V + \mu + \omega} [\beta_V \bar{V}(\nu - \nu_V) + (\nu_V + \mu + \omega)^2]$$

- Note that if $\nu=\nu_V$ (i.e., vaccination does not affect recovery) then $b_1>0$
- However, we expect that vaccinated individuals will recover faster than unvaccinated individuals
- Thus $\nu_V>\nu$
- It follows that b_1 could be negative.

Not for their own
sake; we will use this.

V =vaccinated b_1 =vertex
 c_1 =intercept
 μ =background death
 ω =waning
 β_V =transmissibility
 ν, ν_V =recovery

A possible turning point?

Shows that the result doesn't always happen

this is equivalent to vaccinated individuals covering instantaneously

- In this case,

$$\begin{aligned}\lim_{\nu_V \rightarrow \infty} b_1 &= \lim_{\nu_V \rightarrow \infty} \frac{\beta_V \bar{V}(\nu - \nu_V)}{\omega + \mu + \nu_V} + \omega + \mu + \nu_V \\ &= -\beta_V \bar{V} + \infty > 0\end{aligned}$$

- Defining $f(\nu_V) = \frac{\beta_V \bar{V}(\nu - \nu_V) + (\omega + \mu + \nu_V)^2}{\omega + \mu + \nu_V},$

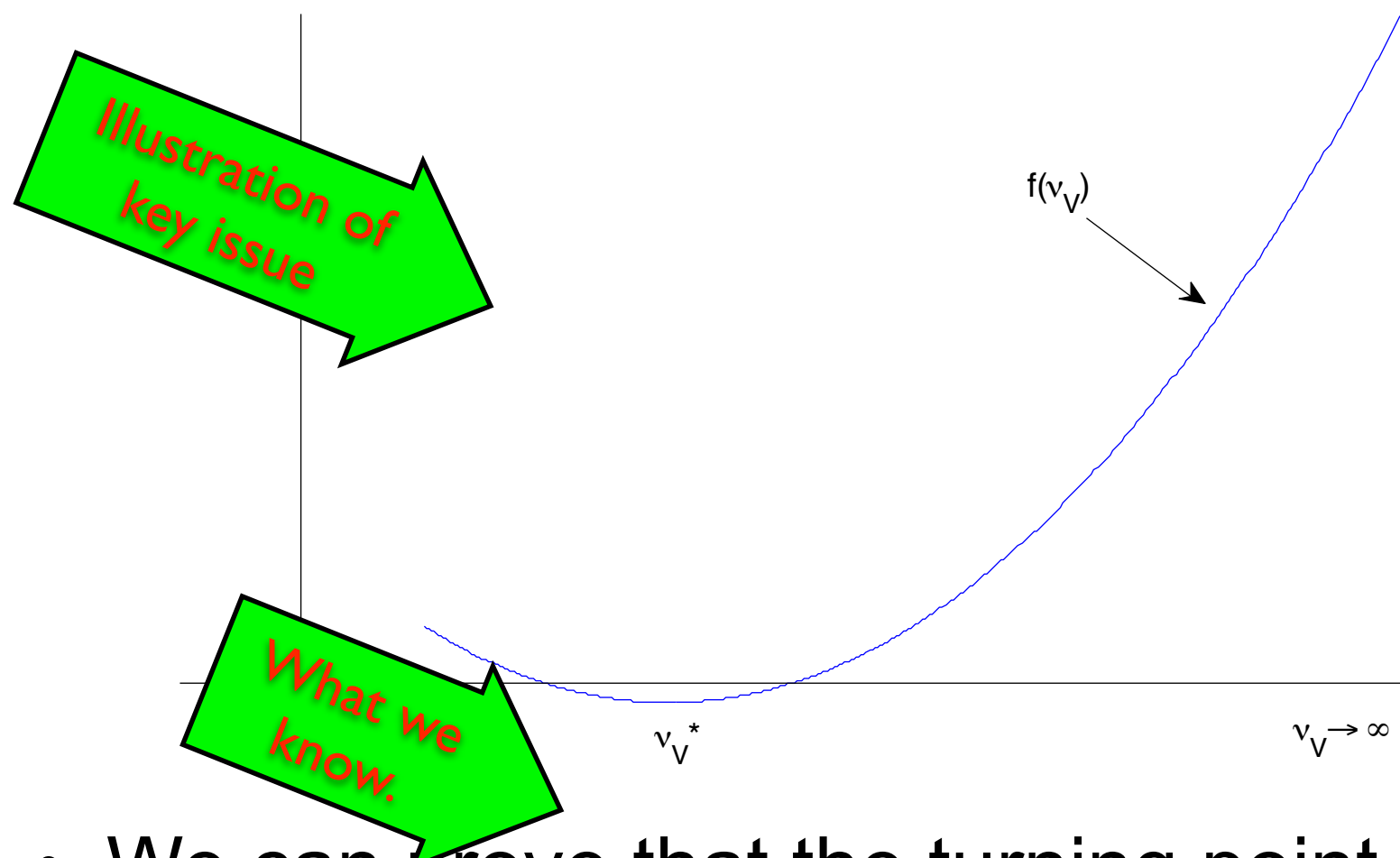
we have $f(\nu_V) > 0$ and $f(\infty) > 0$

- Does f have a local minimum?
- If so, could it be negative?

The issue boils down to this.

V =vaccinated b_1 =vertex
 μ =background death
 ω =waning
 β_V =transmissibility
 ν, ν_V =recovery

Potential form of $f(v_V)$



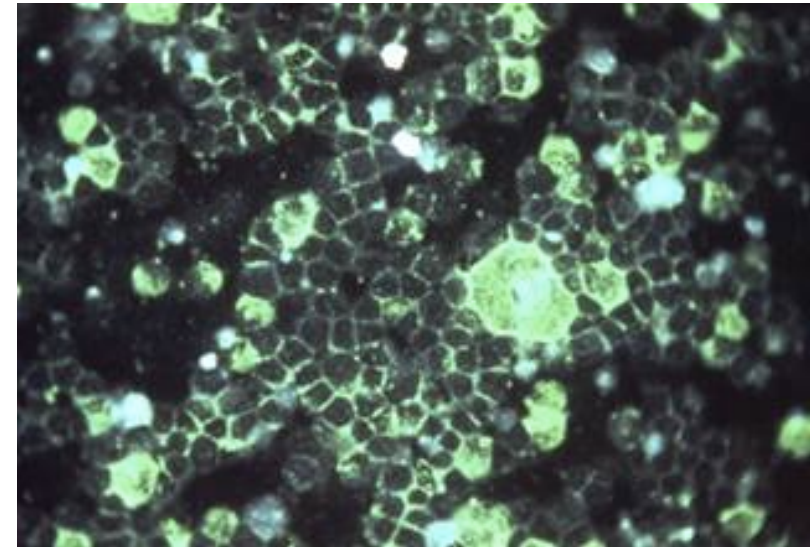
- We can prove that the turning point is a local minimum whenever it exists.

f =vertex
 v, v_V =recovery

Regular vaccinations

- We refine the continuous model
- Vaccination may not occur before birth
- It may also be administered at regular times
 - eg in schools or daycare centres
- We model a vaccine that reduces the susceptible population by a fixed proportion r
- This is described by a series of non-autonomous impulsive differential equations.

Switch to
second model.



r =coverage

The impulsive model

$$\begin{aligned}
 S' &= \mu - \mu S - \beta(t)S(I + I_V), & t \neq t_k \\
 I' &= \beta(t)S(I + I_V) - \nu I - \mu I + \omega I_V, & t \neq t_k \\
 R' &= \nu I - \mu R - \gamma R + \omega R_V, & t \neq t_k \\
 V' &= -\mu V - \beta_V(t)V(I + I_V) + \gamma_V R_V - \omega V, & t \neq t_k \\
 I'_V &= \beta_V V(I + I_V) - \nu_V I_V - \mu I_V - \omega I_V, & t \neq t_k \\
 R'_V &= \nu_V I_V - \mu R_V - \gamma_V R_V - \omega R_V, & t \neq t_k \\
 \Delta S &= -rS, & t = t_k \\
 \Delta V &= rS, & t = t_k,
 \end{aligned}$$

Fundamentally different
kind of model.

where r is the coverage
and t_k are the vaccination times.

S =susceptible I, I_V =infected
 R, R_V =recovered V =vaccinated
 μ =background death ω =waning
 β, β_V =transmissibility
 ν, ν_V =recovery γ, γ_V =loss of
immunity

Susceptible individuals

Assuming transmission is constant, we can show that solutions are bounded below by a positive periodic orbit with endpoints

Outline of proof, results only.

$$S_{\infty}^{-} = \frac{\mu (1 - e^{-(\mu+\beta)\tau})}{(\mu + \beta) (1 - (1 - r)e^{-(\mu+\beta)\tau})}$$

$$S_{\infty}^{+} = \frac{\mu(1 - r) (1 - e^{-(\mu+\beta)\tau})}{(\mu + \beta) (1 - (1 - r)e^{-(\mu+\beta)\tau})}$$

- These correspond to the local maximum and minimum values for the unvaccinated susceptibles after a long time
- Note in particular that $\lim_{\tau \rightarrow 0} S_{\infty}^{-} = 0$.

S=susceptible μ =background death β =transmissibility r =coverage τ =period

Infected individuals

- Assuming infected vaccinated individuals are negligible, we can prove that

$$I' \leq \frac{\beta\mu (1 - e^{-(\mu+\beta)\tau})}{(\mu + \beta) (1 - (1 - r)e^{-(\mu+\beta)\tau})} I - \nu I - \mu I$$

Defining a useful new concept.

we define a new quantity, the *impulsive reproduction number*

$$T_0 = \frac{\beta\mu (1 - e^{-(\mu+\beta)\tau})}{(\nu + \mu)(\mu + \beta) (1 - (1 - r)e^{-(\mu+\beta)\tau})},$$

which has the condition that the disease will be controlled if $T_0 < 1$.

I=infected *μ*=background death
β=transmissibility *ν*=recovery
r=coverage *τ*=period

Impulsive reproduction number

- From the condition $T_0=1$, we can define the maximal period as

$$\hat{\tau} = \frac{1}{\mu + \beta} \ln \frac{(1 - r)(\nu + \mu)(\mu + \beta) - \beta\mu}{(\nu + \mu)(\mu + \beta) - \beta\mu}$$

- This is defined only if

$$r < r^* = \frac{\beta\mu}{(\nu + \mu)(\mu + \beta)}$$

- We can show that $\hat{\tau}$ is increasing as r increases, for $r < r^*$

Determining optimal period

Second case.

- The disease can then be controlled if $\tau < \hat{\tau}$

- For $r > r^*$, $T_0 < 1$ and the disease is always controlled.

T_0 =impulsive reproduction #
 μ =background death
 β =transmissibility ν =recovery
 r =coverage τ =period

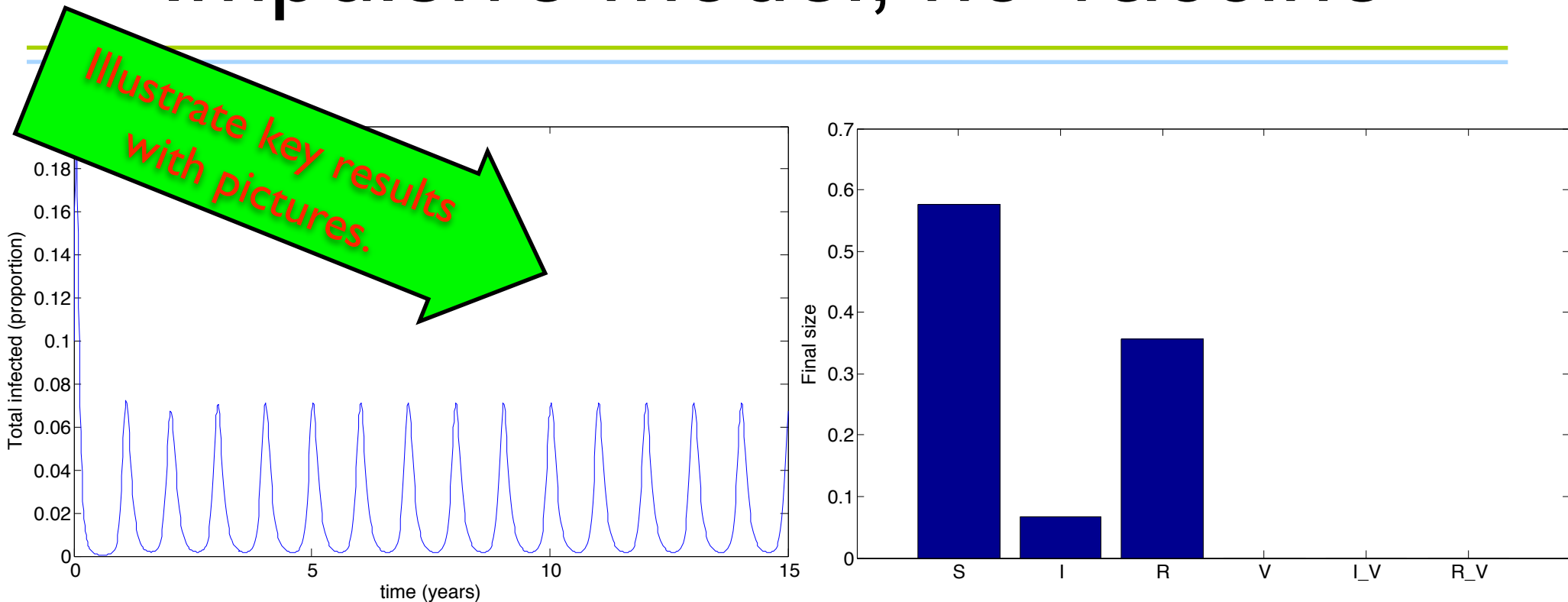
Summarise math results
in practical terms.

Summary of theoretical results

- High coverage can thus control the disease
- If coverage is limited, then sufficiently frequent vaccinations can also achieve control
- Note that the impulsive reproduction number is conditional.



Impulsive model, no vaccine

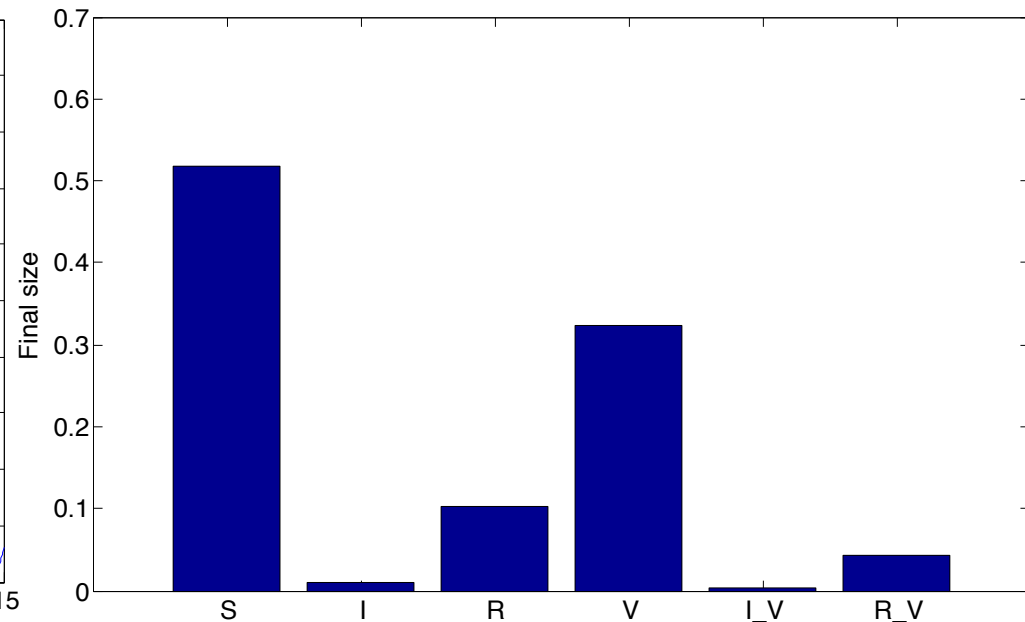
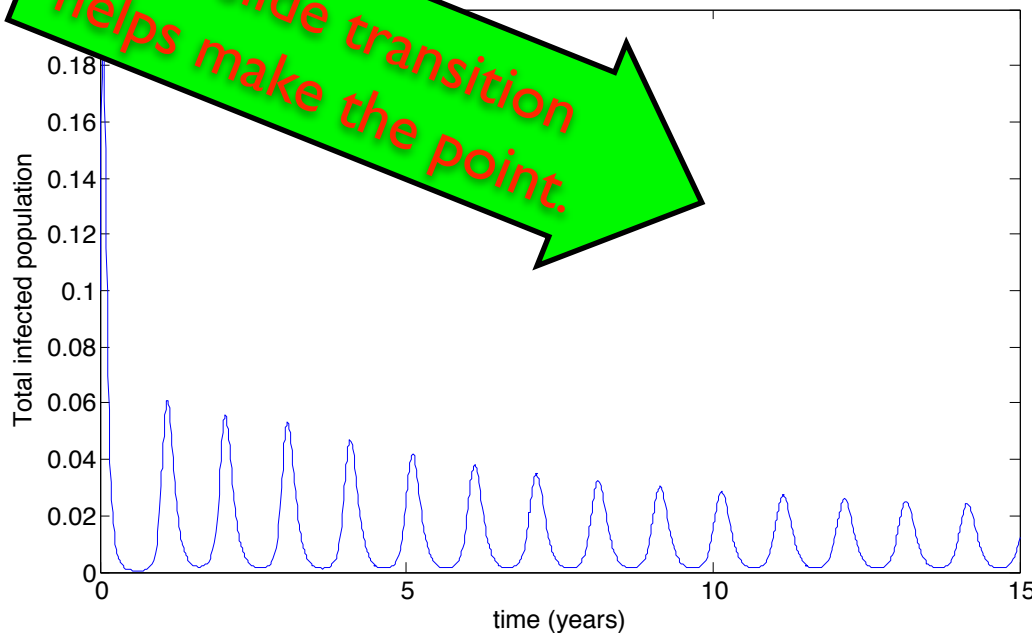


$\mu=1/70$, $\omega=0.1$, $b_0=60$, $b_1=0.16$, $\phi=0.15$,
 $\beta_V=0.5\beta$, $\nu=36$, $\nu_V=1.2\nu$, $\gamma=1.8$, $\gamma_V=0.8\gamma$,
 $r=0$.

μ =background death ω =waning
 b_0 =average transmissibility
 b_1 =seasonal amplitude ϕ =phase
 β_V =transmissibility ν, ν_V =recovery
 γ, γ_V =loss of immunity, r =coverage

Impulsive model, 10% vaccination

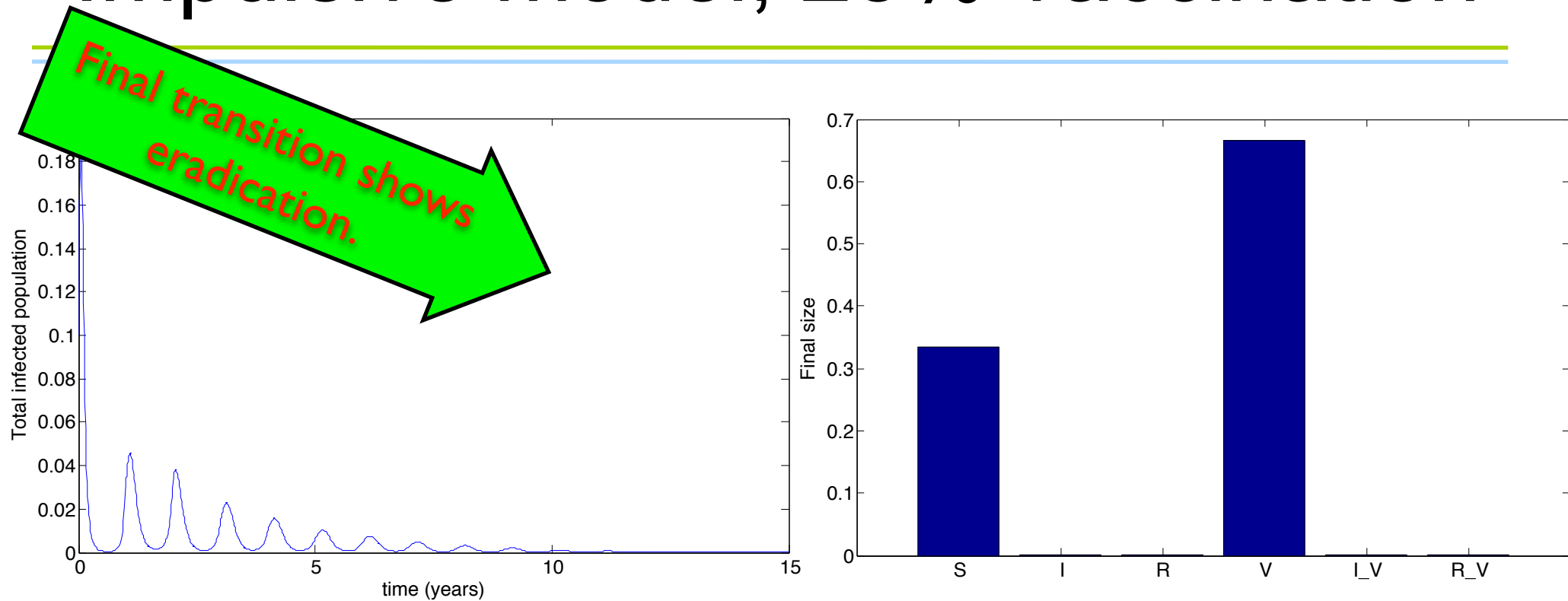
The slide transition helps make the point.



$\mu=1/70$, $\omega=0.1$, $b_0=60$, $b_1=0.16$, $\varphi=0.15$,
 $\beta_V=0.5\beta$, $\nu=36$, $\nu_V=1.2\nu$, $\gamma=1.8$, $\gamma_V=0.8\gamma$,
 $r=0.1$.

μ =background death ω =waning
 b_0 =average transmissibility
 b_1 =seasonal amplitude φ =phase
 β_V =transmissibility ν, ν_V =recovery
 γ, γ_V =loss of immunity, r =coverage

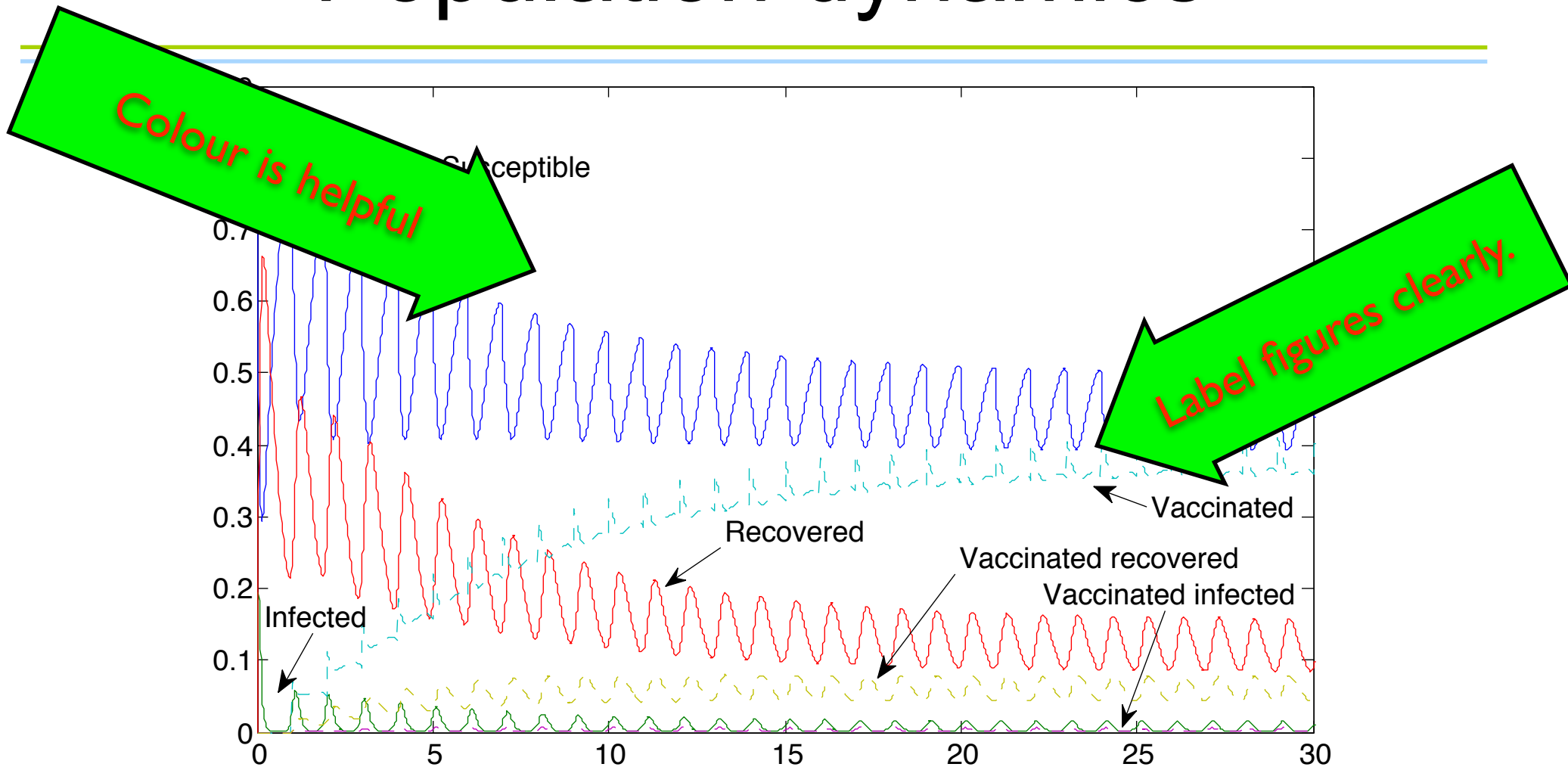
Impulsive model, 25% vaccination



$\mu=1/70$, $\omega=0.1$, $b_0=60$, $b_1=0.16$, $\varphi=0.15$,
 $\beta_V=0.5\beta$, $\nu=36$, $\nu_V=1.2\nu$, $\gamma=1.8$, $\gamma_V=0.8\gamma$,
 $r=0.25$.

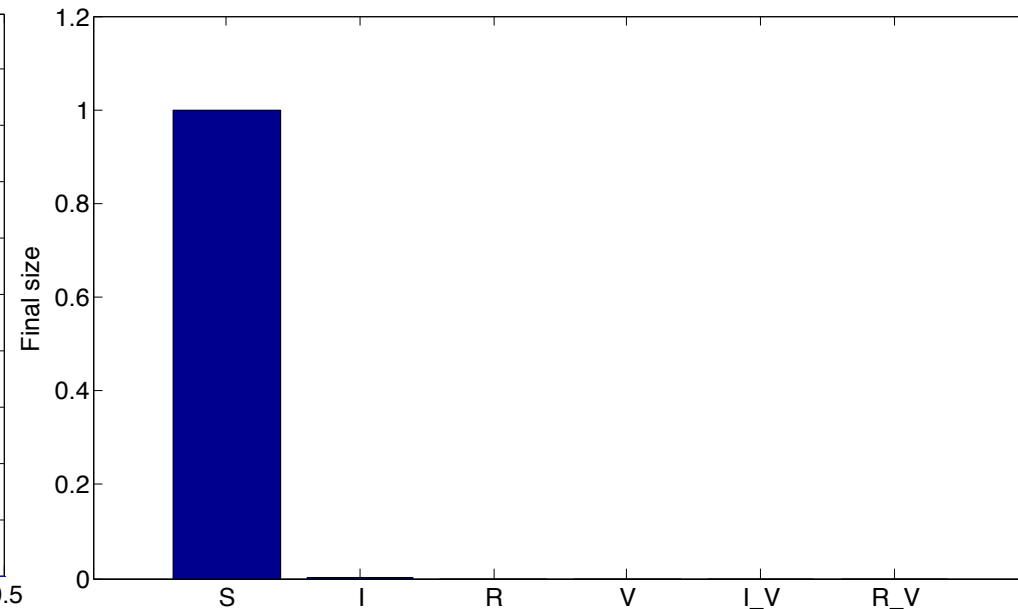
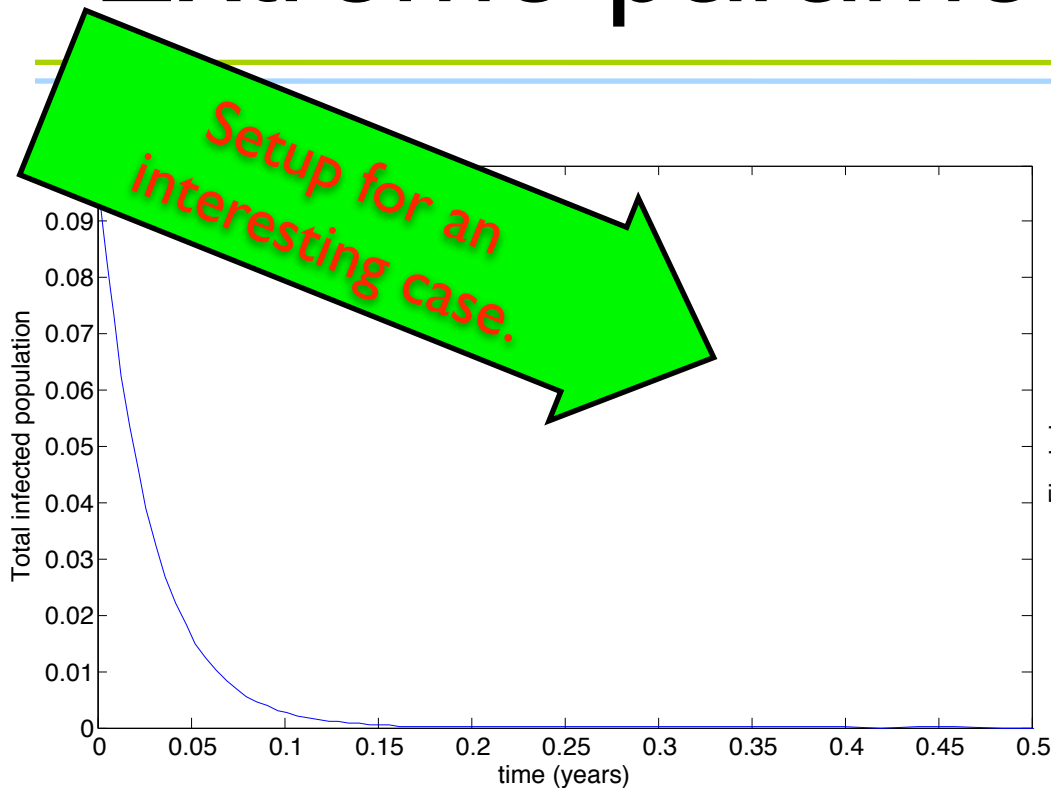
μ =background death ω =waning
 b_0 =average transmissibility
 b_1 =seasonal amplitude φ =phase
 β_V =transmissibility ν, ν_V =recovery
 γ, γ_V =loss of immunity, r =coverage

Population dynamics



- 10% vaccination
- Note the low-level oscillations in both infected classes.

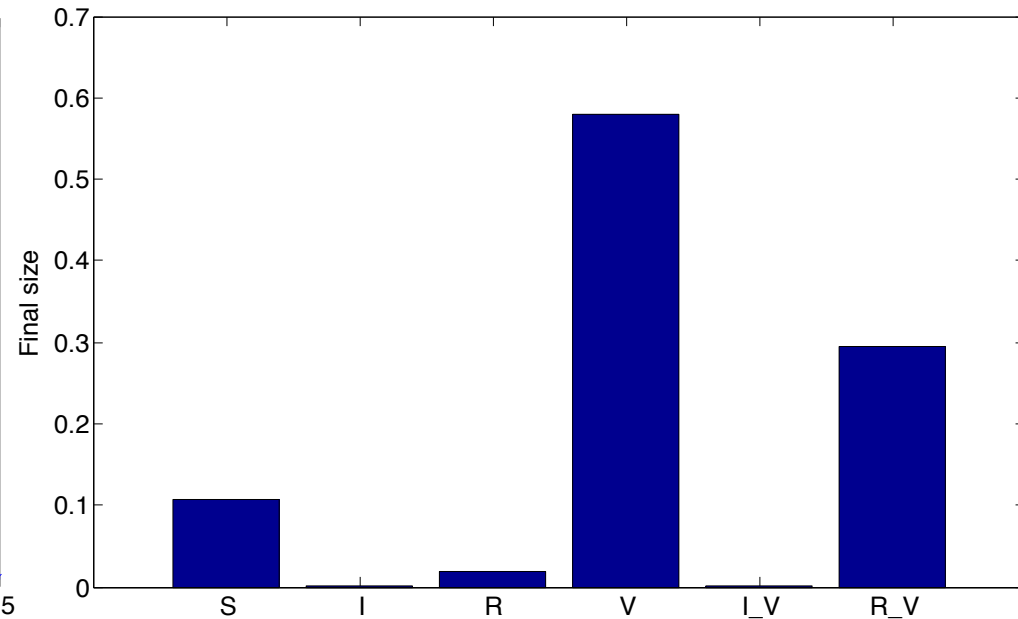
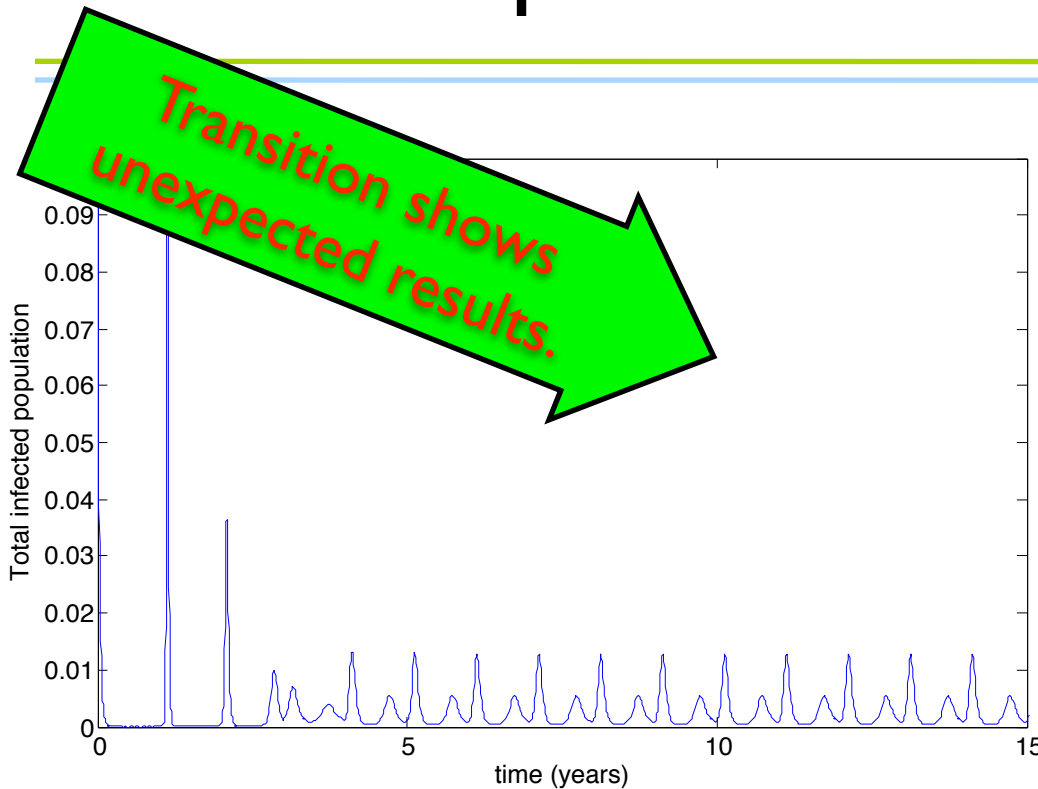
Extreme parameters, no vaccine



$\mu=1/70$, $\omega=0.1$, $\beta=0.03$, $\beta_V=300$, $\nu=36$, $\nu_V=177$,
 $\gamma=1.8$, $\gamma_V=0.8\gamma$, $r=0$.

μ =background death ω =waning
 b_0 =average transmissibility
 b_1 =seasonal amplitude ϕ =phase
 β_V =transmissibility ν, ν_V =recovery
 γ, γ_V =loss of immunity, r =coverage

Extreme parameters, 100% vaccination



$\mu=1/70$, $\omega=0.1$, $\beta=0.03$, $\beta_V=300$, $\nu=36$, $\nu_V=177$,
 $\gamma=1.8$, $\gamma_V=0.8\gamma$, $r=1$.

μ =background death ω =waning
 b_0 =average transmissibility
 b_1 =seasonal amplitude ϕ =phase
 β_V =transmissibility ν, ν_V =recovery
 γ, γ_V =loss of immunity, r =coverage

Unexpected infection spikes

- We used extreme vaccination parameters
- Transmission due to vaccinated individuals was extremely high
- But recovery was fast
- This allowed low-level infection spikes to occur in vaccinated populations
- Note that this is not a backward bifurcation
- Rather, it is a destabilisation of the DFE.

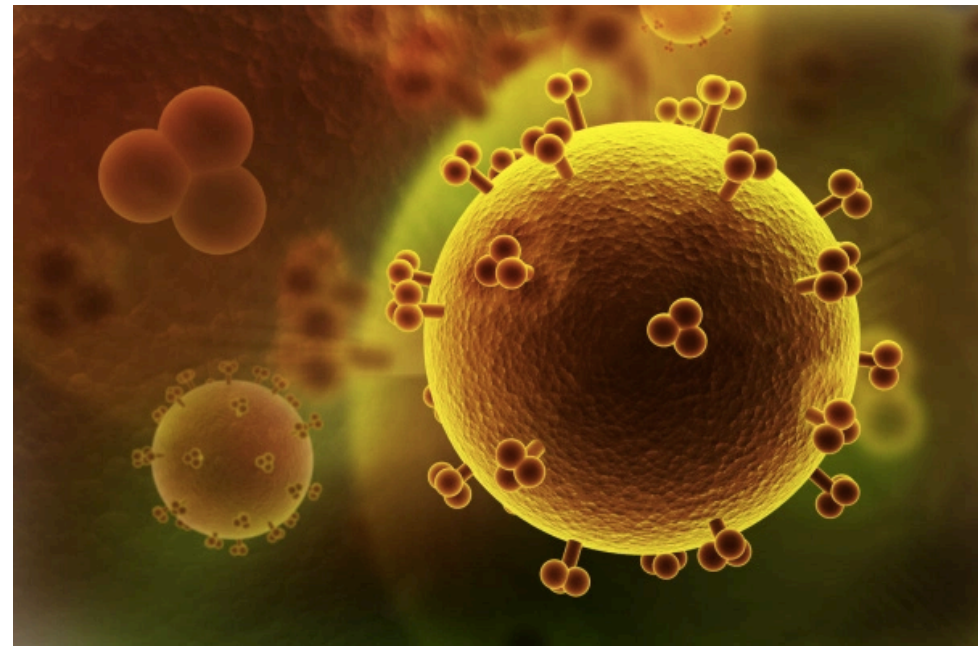


*Eliminate conclusion
most people will make.*

Summary

Summary is
concise.

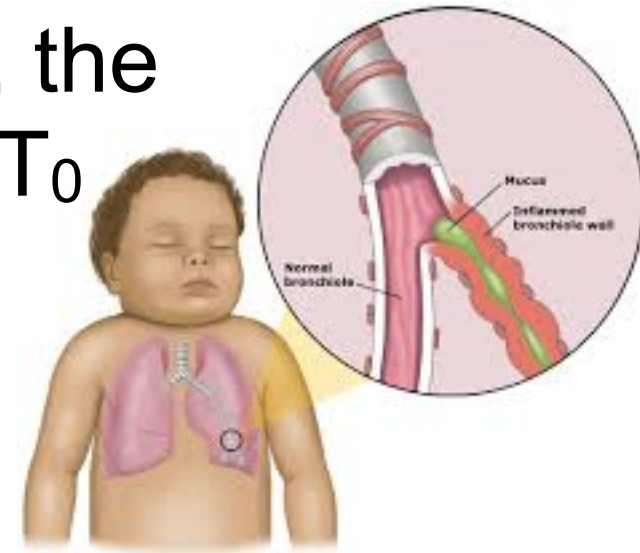
- We considered two forms of vaccination:
 - single administration before infection
 - e.g., a maternal vaccine
 - periodic vaccination
- Using impulsive differential equations, we were able to formulate conditions on the period and strength of vaccination to allow for disease control.



Impulsive reproduction number

Highlights the new quantity.

- We also defined a new quantity, the impulsive reproduction number T_0
- This is a sufficient (but not necessary) condition that ensures eradication if $T_0 < 1$
- In this case, the infected population is contracting within each impulsive cycle
- The result is eventual eradication of the infection.



T_0 =impulsive reproduction number

Infection spikes

Notes the unexpected numerical result.

Infection spikes occur when vaccine-induced transmission is extremely high but recovery is extremely fast

- They occur even when the transmission function is not oscillating
- They are unlikely to occur in reality with the parameters we chose
- Nevertheless, we have shown proof-of-concept that such an outcome is possible.



Limitations

Always list your limitations.

We assumed:

- The time to administer the vaccine was significantly shorter than the time between vaccinations
- A well-mixed population
- A single age cohort
- A population of fixed size
- Constant birth and death
- Maternal vaccination in the first model.



Conclusions

Reminder of what we studied

- A vaccine that targets RSV infection has the potential to significantly reduce the overall incidence of the disease

One audience takeaway

- Long-term, periodic vaccination can theoretically control the disease, but coverage needs to be high, or administration sufficiently frequent

Second audience takeaway

What does this mean for the real world?

- Some parameters have the potential to significantly reduce the expected infection spike
- Care should be taken to understand long-term effects when introducing new vaccines.

Only one page for the conclusion.

Acknowledge
collaborators verbally

Key reference

- R.J. Smith?, A.B. Hogan, G.N. Mercer *Unexpected infection spikes in a model of Respiratory Syncytial Virus vaccination* (Vaccines, 2017, 5:12).

<http://mysite.science.uottawa.ca/rsmith43>

Have a website where people
can find you and your work.



Summary

- * You should be telling a story
- * It should have a beginning, middle and end
- * Make it appealing to an audience who will be both reading and listening
 - needs to work on both levels
 - they should not be the same
 - try not to “read out” your slides
- * Talks should have pizzazz.

Practice, practice, practice

- * Practice every talk three times
 - once for saying the words
 - once for timing
 - once for a polish
- * The talk is the major way you will get other people interested in your work
- * It needs to be deep, dramatic and digestible
- * Do not go over time
 - leave time for questions.

The take-home message

- * If you're junior, bring hard copies of your papers to hand out to interested parties
 - people are more likely to read a hard copy at a conference or on the train
- * This is your moment in the sun
- * Don't waste it through poor preparation
- * The audience will take away 1-2 points from your talk, no more
 - make sure they're the points you want them to remember.

Final page

- * People will be looking at this page a lot
 - far longer than any other in your talk
- * Make sure they have something to look at.

mysite.science.uottawa.ca/rsmith43/TalkGiving.pdf

