Activity/Susceptibility systems:

Towards a paradigm for propagation of epidemics, collective behaviors, and propagation in excitable media

Samuel Nordmann (in collaboration with Henri Berestycki and Luca Rossi)

Tel-Aviv University. samueln@mail.tau.ac.il
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the SIR epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the SIR epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the SIR epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
**Context:**

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the *SIR* epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

**Goal:**

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the SIR epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
**Context:**

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the *SIR* epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

**Goal:**

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the \textit{SIR} epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe \textit{propagation in excitable media} (e.g. flame propagation) or \textit{collective behaviors} (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with \textit{threshold phenomena} & \textit{spatial propagation}. 
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the SIR epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the SIR epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the SIR epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation
Context:

- Elaborate mathematical models used in epidemiology:
  - Predict complex dynamics (threshold phenomenon, spatial propagation)
  - Testing hypothesis through numerics
- Many instances and refinements of the SIR epidemiology model [Kermack & McKendrik 1927].
- Same kind of equations to describe propagation in excitable media (e.g. flame propagation) or collective behaviors (e.g. riots).

Goal:

- Identify a general paradigm: epidemics, propagation in excitable media, propagation of collective behaviors, etc.
- Provide a unified mathematical framework & a class of model useful to study these phenomena.
- Insights into the core mechanisms and mathematical nature of epidemiology models.
- Robust mathematical tools and general results to deal with threshold phenomena & spatial propagation.
1. Introduction: from SIR model to a general paradigm
2. Epidemiological modeling of collective behaviors: the example of riots
3. Mathematical framework & main results
4. Possible extensions: spatial heterogeneity
5. Conclusion
1. Introduction: from SIR model to a general paradigm
2. Epidemiological modeling of collective behaviors: the example of riots
3. Mathematical framework & main results
4. Possible extensions: spatial heterogeneity
5. Conclusion
Table of contents

1. Introduction: from SIR model to a general paradigm
2. Epidemiological modeling of collective behaviors: the example of riots
3. Mathematical framework & main results
4. Possible extensions: spatial heterogeneity
5. Conclusion
1. Introduction: from SIR model to a general paradigm
2. Epidemiological modeling of collective behaviors: the example of riots
3. Mathematical framework & main results
4. Possible extensions: spatial heterogeneity
5. Conclusion
1. Introduction: from SIR model to a general paradigm
2. Epidemiological modeling of collective behaviors: the example of riots
3. Mathematical framework & main results
4. Possible extensions: spatial heterogeneity
5. Conclusion
Introduction: from *SIR* to general paradigm
**The SIR epidemiology model**

- Population divided in compartments:
  
  Susceptibles $S$; Infected $I$; Recovered/Removed $R$

- Dynamical system $S = S(t)$, $I = I(t)$, $R = R(t)$,

  $$
  \begin{align*}
  \frac{dl}{dt} &= \beta SI - \gamma I, \\
  \frac{dS}{dt} &= -\beta SI, \\
  \frac{dR}{dt} &= \gamma I.
  \end{align*}
  $$

  $\beta, \gamma: \text{coefs} > 0$.

- We omit $R$.

- **Initial conditions.** Typically: $I_0 \approx 0$, $S_0 = \text{constant}$.
The *SIR* epidemiology model

- Population divided in compartments:
  - Susceptibles \( S \); Infected \( I \); Recovered/Removed \( R \)
- Dynamical system \( S = S(t), \quad I = I(t), \quad R = R(t), \)
  \[
  \begin{align*}
  \frac{dI}{dt} &= \beta SI - \gamma I, \\
  \frac{dS}{dt} &= -\beta SI, \\
  \frac{dR}{dt} &= \gamma I.
  \end{align*}
  \]

\( \beta, \gamma : \text{coefs} > 0. \)

- We omit \( R \).
- **Initial conditions.** Typically: \( I_0 \approx 0, \quad S_0 = \text{constant} \).
The SIR epidemiology model

- Population divided in compartments:
  Susceptibles $S$ ; Infected $I$ ; Recovered/Removed $R$
- Dynamical system $S = S(t), I = I(t), R = R(t)$,
  \[
  \begin{aligned}
  \frac{dl}{dt} &= \beta SI - \gamma I, \\
  \frac{dS}{dt} &= -\beta SI, \\
  \frac{dR}{dt} &= \gamma I.
  \end{aligned}
  \]
  $\beta, \gamma$ : coeffs $> 0$.

- Contagion
- Death/Recovery

- We omit $R$.
- Initial conditions. Typically: $I_0 \approx 0$, $S_0 = constant$. 
The SIR epidemiology model

- Population divided in compartments:
  Susceptibles $S$; Infected $I$; Recovered/Removed $R$
- Dynamical system $S = S(t)$, $I = I(t)$, $R = R(t)$,
  \[
  \begin{align*}
  \frac{dI}{dt} &= \beta SI - \gamma I, \\
  \frac{dS}{dt} &= -\beta SI, \\
  \frac{dR}{dt} &= \gamma I.
  \end{align*}
  \]
  \(\beta, \gamma: \text{coefs} > 0\).

- We omit $R$.
- Initial conditions. Typically: $I_0 \approx 0$, $S_0 = \text{constant}$. 

\[\text{Contagion} \quad \beta SI \quad \text{Death/Recovery} \quad \gamma I\]
The SIR epidemiology model

- Population divided in compartments:
  - Susceptibles $S$;
  - Infected $I$;
  - Recovered/Removed $R$

- Dynamical system $S = S(t)$, $I = I(t)$, $R = R(t)$,

$$
\begin{align*}
\frac{dS}{dt} &= -\beta SI, \\
\frac{dI}{dt} &= \beta SI - \gamma I, \\
\frac{dR}{dt} &= \gamma I.
\end{align*}
$$

$\beta, \gamma : \text{coefs} > 0$.

- Contagion $\xrightarrow{\beta SI}$
- Death/Recovery $\xrightarrow{\gamma I}$

- We omit $R$.

- Initial conditions. Typically: $I_0 \approx 0$, $S_0 = \text{constant}$. 
The SIR epidemiology model

- Population divided in compartments:
  - Susceptibles $S$; Infected $I$; Recovered/Removed $R$
- Dynamical system $S = S(t)$, $I = I(t)$, $R = R(t)$,
  \[
  \begin{align*}
  \frac{dI}{dt} &= \beta SI - \gamma I, \\
  \frac{dS}{dt} &= -\beta SI, \\
  \frac{dR}{dt} &= \gamma I.
  \end{align*}
\]
- $\beta, \gamma$: coefs $> 0$.

- Contagion: $\beta SI$
- Death/Recovery: $\gamma I$
- We omit $R$.
- Initial conditions. Typically: $I_0 \approx 0$, $S_0 = \text{constant}$. 
The SIR epidemiology model

- Population divided in compartments:
  - Susceptibles $S$; Infected $I$; Recovered/Removed $R$
- Dynamical system $S = S(t)$, $I = I(t)$, $R = R(t)$,

$$\begin{aligned}
\frac{dI}{dt} & = \beta SI - \gamma I, \\
\frac{dS}{dt} & = -\beta SI, \\
\frac{dR}{dt} & = \gamma I.
\end{aligned}$$

$\beta, \gamma$: coefs $> 0$.

- We omit $R$.
- **Initial conditions.** Typically: $I_0 \approx 0$, $S_0 = constant$. 

\[\]
The spatial SIR epidemiology model

- $S(t, x), I(t, x)$, depend on time $t > 0$ and on space $x \in \mathbb{R}^n$.

- Movement of individuals?
  
  **Assumption:** Brownian motion. \(\Rightarrow\) diffusion.

\[
\begin{align*}
\frac{dI}{dt} &= \beta SI - \gamma I, \\
\frac{dS}{dt} &= -\beta SI,
\end{align*}
\]

\(d_1 > 0, \; d_2 \geq 0\) (motility).

- Other assumptions are possible, to account for migration, heterogeneous interactions, contact matrix, etc.
  
  - Non local diffusion
    \[
    -\Delta_x u \iff \int_{\mathbb{R}^n} K(x - y)(u(x) - u(y)) dy.
    \]
  
  - Diffusion on a graph
    \[
    -\Delta_x u \iff \sum_y q_{yx}(u(x) - u(y)).
    \]
The spatial SIR epidemiology model

- \( S(t, x), I(t, x) \), depend on time \( t > 0 \) and on space \( x \in \mathbb{R}^n \).
- Movement of individuals?
  
  **Assumption**: Brownian motion. \( \Rightarrow \) diffusion.

\[
\begin{align*}
\frac{dI}{dt} &= \beta SI - \gamma I, \\
\frac{dS}{dt} &= -\beta SI,
\end{align*}
\]

\( d_1 > 0, \ d_2 \geq 0 \) (motility).

- Other assumptions are possible, to account for migration, heterogeneous interactions, contact matrix, etc.
  
  - Non local diffusion
    
    \[-\Delta_x u \leftrightarrow \int_{\mathbb{R}^n} K(x - y)(u(x) - u(y)) \, dy.\]
  
  - Diffusion on a graph
    
    \[-\Delta_x u \leftrightarrow \sum_y q_{yx}(u(x) - u(y))\]
The spatial SIR epidemiology model

- $S(t, x), I(t, x)$, depend on time $t > 0$ and on space $x \in \mathbb{R}^n$.
- Movement of individuals?
  
  Assumption: Brownian motion. $\Rightarrow$ diffusion.

\[
\begin{aligned}
  \frac{dl}{dt} &= \beta SI - \gamma I, \\
  \frac{dS}{dt} &= -\beta SI,
\end{aligned}
\]

$d_1 > 0, d_2 \geq 0$ (motility).

- Other assumptions are possible, to account for migration, heterogeneous interactions, contact matrix, etc.
  
  - Non local diffusion
    
    $-\Delta_x u \longleftrightarrow \int_{\mathbb{R}^n} K(x - y)(u(x) - u(y)) \, dy.$
  
  - Diffusion on a graph
    
    $-\Delta_x u \longleftrightarrow \sum_y q_{yx}(u(x) - u(y))$
The spatial SIR epidemiology model

- $S(t, x), I(t, x)$, depend on time $t > 0$ and on space $x \in \mathbb{R}^n$.
- Movement of individuals?
  Assumption: Brownian motion. $\Rightarrow$ diffusion.

\[
\begin{align*}
\frac{dl}{dt} &= \beta SI - \gamma I, \\
\frac{dS}{dt} &= -\beta SI,
\end{align*}
\]

$d_1 > 0, d_2 \geq 0$ (motility).
- Other assumptions are possible, to account for migration, heterogeneous interactions, contact matrix, etc.
  - Non local diffusion
    \[-\Delta_x u \quad \longleftrightarrow \quad \int_{\mathbb{R}^n} K(x - y)(u(x) - u(y)) \, dy.
    \]
  - Diffusion on a graph
    \[-\Delta_x u \quad \longleftrightarrow \quad \sum_y q_{yx}(u(x) - u(y)).\]
The spatial \textit{SIR} epidemiology model

- \( S(t, x), I(t, x) \), depend on time \( t > 0 \) and on space \( x \in \mathbb{R}^n \).
- Movement of individuals?
  
  \textbf{Assumption:} Brownian motion. \quad \Rightarrow \text{diffusion.}

\[
\begin{align*}
\frac{dI}{dt} &= \beta SI - \gamma I, \\
\frac{dS}{dt} &= -\beta SI,
\end{align*}
\]

\( d_1 > 0, \ d_2 \geq 0 \) (motility).

- Other assumptions are possible, to account for migration, heterogeneous interactions, contact matrix, etc.
  
  - Non local diffusion

\[
-\Delta_x u \quad \longleftrightarrow \quad \int_{\mathbb{R}^n} K(x - y)(u(x) - u(y)) \, dy.
\]

  - Diffusion on a graph

\[
-\Delta_x u \quad \longleftrightarrow \quad \sum_y q_{yx}(u(x) - u(y))
\]
The spatial SIR epidemiology model

- $S(t, x), I(t, x)$, depend on time $t > 0$ and on space $x \in \mathbb{R}^n$.
- Movement of individuals?
  
  Assumption: Brownian motion. $\Rightarrow$ diffusion.

$$\begin{align*}
\frac{dI}{dt} &= \beta SI - \gamma I, \\
\frac{dS}{dt} &= -\beta SI,
\end{align*}$$

$d_1 > 0, \ d_2 \geq 0$ (motility).

- Other assumptions are possible, to account for migration, heterogeneous interactions, contact matrix, etc.
  - Non local diffusion
    $$-\Delta_x u \quad \longleftrightarrow \quad \int_{\mathbb{R}^n} K(x - y)(u(x) - u(y)) \, dy.$$  

  - Diffusion on a graph
    $$-\Delta_x u \quad \longleftrightarrow \quad \sum_y q_{yx}(u(x) - u(y))$$
The spatial **SIR** epidemiology model

- **S**(*t*, *x*), **I**(*t*, *x*), depend on time *t > 0* and on space *x ∈ R^n*.
- Movement of individuals?
  
  **Assumption:** Brownian motion.  
  
  \[
  \begin{cases}
  \frac{dI}{dt} = \beta SI - \gamma I, \\
  \frac{dS}{dt} = -\beta SI,
  \end{cases}
  \]

  \[d_1 > 0, \quad d_2 \geq 0\text{ (motility).}\]

- Other assumptions are possible, to account for migration, heterogeneous interactions, contact matrix, etc.
  
  - Non local diffusion
  
  \[-\Delta_x u \iff \int_{\mathbb{R}^n} K(x - y)(u(x) - u(y)) \, dy.\]
  
  - Diffusion on a graph
  
  \[-\Delta_x u \iff \sum_y q_{yx}(u(x) - u(y)).\]
The $SI(R)$ epidemiology model: Threshold

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI, \\
l_0(x) &\geq 0 \quad ; \quad S_0(x) \equiv \text{constant}
\end{align*}
\]

→ **Threshold** phenomenon on the sign of $K_0 := \beta S_0 - \gamma$:

- If $K_0 < 0$: no epidemics,
- If $K_0 > 0$: spatial propagation of an epidemics

$\Rightarrow$ Hair-trigger: a little number of infected triggers an epidemic.
The \( SI(R) \) epidemiology model: Threshold

\[
\begin{aligned}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI, \\
I_0(x) &\geq 0 ; S_0(x) \equiv constant
\end{aligned}
\]

→ Threshold phenomenon on the sign of \( K_0 := \beta S_0 - \gamma \):

- If \( K_0 < 0 \): no epidemics,
- If \( K_0 > 0 \): spatial propagation of an epidemics

⇒ Hair-trigger: a little number of infected triggers an epidemic.

\( K_0 < 0 \)
The \( SI(R) \) epidemiology model: Threshold

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI, \\
I_0(x) &\geq 0 \quad ; \quad S_0(x) \equiv \text{constant}
\end{align*}
\]

→ Threshold phenomenon on the sign of \( K_0 := \beta S_0 - \gamma \):

- If \( K_0 < 0 \): no epidemics,
- If \( K_0 > 0 \): spatial propagation of an epidemics

\( K_0 > 0 \) ⇔ Hair-trigger: a little number of infected triggers an epidemic.
The $SI(R)$ epidemiology model: Threshold

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI, \\
I_0(x) &\geq 0 ; S_0(x) \equiv \text{constant}
\end{align*}
\]

→ **Threshold** phenomenon on the sign of $K_0 := \beta S_0 - \gamma$:

- If $K_0 < 0$: no epidemics,
- If $K_0 > 0$: spatial propagation of an epidemics

$\Rightarrow$ Hair-trigger: a little number of infected triggers an epidemic.

$K_0 > 0$
The $SI(R)$ epidemiology model: Threshold

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI, \\
I_0(x) \geq 0; \quad S_0(x) \equiv \text{constant}
\end{align*}
\]

→ Threshold phenomenon on the sign of $K_0 := \beta S_0 - \gamma$:

- If $K_0 < 0$: no epidemics,
- If $K_0 > 0$: spatial propagation of an epidemics
  → Hair-trigger: a little number of infected triggers an epidemic.

The sign of $K_0$ determines the stability of the steady state $(0, S_0)$.

Important remark: $K_0 > 0 \iff R_0 := \frac{\beta}{\gamma} S_0 > 1 \iff S_0 > \frac{\gamma}{\beta}$. 
The \textit{SI(R)} epidemiology model: Threshold

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI,
\end{align*}
\]

\[I_0(x) \geq 0 \quad ; \quad S_0(x) \equiv \text{constant}\]

\rightarrow \textbf{Threshold} phenomenon on the sign of \( K_0 := \beta S_0 - \gamma \):

- If \( K_0 < 0 \): no epidemics,
- If \( K_0 > 0 \): spatial propagation of an epidemics
  \( \rightsquigarrow \) Hair-trigger: a little number of infected triggers an epidemic.

\( \rightsquigarrow \) The sign of \( K_0 \) determines the \textbf{stability} of the steady state \((0, S_0)\).

\textbf{Important remark:} \( K_0 > 0 \quad \Longleftrightarrow \quad R_0 := \frac{\beta}{\gamma} S_0 > 1 \quad \Longleftrightarrow \quad S_0 > \frac{\gamma}{\beta} \).
The $SI(R)$ epidemiology model

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

- $S(t) \times I(t)$: proba. of encounter between $S$ and $I$ (up to normalization). Law of mass action.
- This specific form with $d_2 = 0$ allows for a classical mathematical "trick" that reduces the $SIR$ model to a single $KPP$ equation.
- However, other types of interactions are considered:
  - non-constant total population $\frac{SI}{S + I}$
  - saturation $SI \left(1 - \frac{I}{I_{max}}\right)$
  - other non-linearities $SI^2$, etc.

Questions:

- What does it change? What remains true?
- What model can be used? Is there a general framework?
The \textit{SI(R)} epidemiology model

\[
\begin{cases}
\partial_t I - d_1 \Delta_x I = \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S = -\beta SI.
\end{cases}
\]

- \(S(t) \times I(t)\) : proba. of encounter between \(S\) and \(I\) (up to normalization). \textit{Law of mass action}.
- This specific form with \(d_2 = 0\) allows for a classical mathematical “trick” that reduces the \textit{SIR} model to a single \textit{KPP} equation.
- However, other types of interactions are considered:
  - non-constant total population \(\frac{SI}{S + I}\)
  - saturation \(SI \left(1 - \frac{I}{I_{\text{max}}}\right)\)
  - other non-linearities \(SI^2\), etc.

\textbf{Questions :}

- What does it change? What remains true?
- What model can be used? Is there a general framework?
The $SI(R)$ epidemiology model

$$\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}$$

- $S(t) \times I(t)$: proba. of encounter between $S$ and $I$ (up to normalization). Law of mass action.
- This specific form with $d_2 = 0$ allows for a classical mathematical "trick" that reduces the $SIR$ model to a single $KPP$ equation.
- However, other types of interactions are considered:
  - non-constant total population $\frac{SI}{S + I}$
  - saturation $SI \left( 1 - \frac{I}{I_{max}} \right)$
  - other non-linearities $SI^2$, etc.

Questions:

- What does it change? What remains true?
- What model can be used? Is there a general framework?
The $SI(R)$ epidemiology model

\[
\begin{aligned}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{aligned}
\]

- $S(t) \times I(t)$: proba. of encounter between $S$ and $I$ (up to normalization). Law of mass action.
- This specific form with $d_2 = 0$ allows for a classical mathematical “trick” that reduces the $SIR$ model to a single $KPP$ equation.
- However, other types of interactions are considered:
  - non-constant total population $\frac{SI}{S + I}$
  - saturation $SI \left(1 - \frac{I}{I_{\text{max}}} \right)$
  - other non-linearities $SI^2$, etc.

Questions:

- What does it change? What remains true?
- What model can be used? Is there a general framework?
The $SI(R)$ epidemiology model

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

- $S(t) \times I(t)$: proba. of encounter between $S$ and $I$ (up to normalization). **Law of mass action.**
- This specific form with $d_2 = 0$ allows for a classical mathematical “trick” that reduces the $SIR$ model to a single $KPP$ equation.
- However, other types of interactions are considered:
  - non-constant total population $\frac{SI}{S + I}$
  - saturation $SI \left(1 - \frac{I}{I_{max}}\right)$
  - other non-linearities $SI^2$, etc.

**Questions:**

- What does it change? What remains true?
- What model can be used? Is there a general framework?
The SI(R) epidemiology model

\[
\begin{aligned}
\partial_t I - d_1 \Delta_x I &= \beta S I - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta S I.
\end{aligned}
\]

- \( S(t) \times I(t) \): proba. of encounter between \( S \) and \( I \) (up to normalization). Law of mass action.
- This specific form with \( d_2 = 0 \) allows for a classical mathematical “trick” that reduces the SIR model to a single KPP equation.
- However, other types of interactions are considered:
  - non-constant total population \( \frac{SI}{S + I} \)
  - saturation \( SI \left( 1 - \frac{I}{I_{\text{max}}} \right) \)
  - other non-linearities \( SI^2 \), etc.

Questions:

- What does it change? What remains true?
- What model can be used? Is there a general framework?
The \( SI(R) \) epidemiology model

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

- \( S(t) \times I(t) \): proba. of encounter between \( S \) and \( I \) (up to normalization). Law of mass action.
- This specific form with \( d_2 = 0 \) allows for a classical mathematical “trick” that reduces the \( SIR \) model to a single \( KPP \) equation.
- However, other types of interactions are considered:
  - non-constant total population \( \frac{SI}{S + I} \)
  - saturation \( SI \left(1 - \frac{I}{I_{max}}\right) \)
  - other non-linearities \( SI^2 \), etc.

Questions:

- What does it change? What remains true?
- What model can be used? Is there a general framework?
**SI(R) model** \(\rightarrow\) **General paradigm**

\[
\begin{align*}
I(t, x) & : \text{fraction of Infected}, \\
S(t, x) & : \text{fraction of Susceptibles}
\end{align*}
\]

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= \Phi(u, v), \\
\partial_t v - d_2 \Delta_x v &= \Psi(u, v).
\end{align*}
\]
$SI(R)$ model $\rightarrow$ General paradigm

\[
\begin{align*}
I(t, x) & : \text{fraction of Infected}, \\
S(t, x) & : \text{fraction of Susceptibles}
\end{align*}
\]

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

\[
\begin{align*}
u(t, x) & : \text{level of Activity}, \\
v(t, x) & : \text{level of Susceptibility}
\end{align*}
\]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= \Phi(u, v), \\
\partial_t v - d_2 \Delta_x v &= \Psi(u, v).
\end{align*}
\]
\( SI(R) \) model \hspace{2cm} \rightarrow \hspace{2cm} \text{General paradigm}

\[
\begin{align*}
I(t, x) & : \text{fraction of Infected,} \\
S(t, x) & : \text{fraction of Susceptibles}
\end{align*}
\]

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

\[
\begin{align*}
I(t, x) & : \text{fraction of Infected,} \\
S(t, x) & : \text{fraction of Susceptibles}
\end{align*}
\]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= \Phi(u, v), \\
\partial_t v - d_2 \Delta_x v &= \Psi(u, v).
\end{align*}
\]
$SI(R)$ model $\rightarrow$ General paradigm

\[
\begin{align*}
I(t,x) &: \text{fraction of Infected}, \\
S(t,x) &: \text{fraction of Susceptibles}
\end{align*}
\]

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

\[
\begin{align*}
u(t,x) &: \text{level of Activity}, \\
v(t,x) &: \text{level of Susceptibility}
\end{align*}
\]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= \Phi(u,v), \\
\partial_t v - d_2 \Delta_x v &= \Psi(u,v).
\end{align*}
\]
$SI(R)$ model $\rightarrow$ General paradigm

\[
\begin{align*}
I(t, x) : \text{fraction of Infected,} \\
S(t, x) : \text{fraction of Susceptibles}
\end{align*}
\]

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= \Phi(u, v), \\
\partial_t v - d_2 \Delta_x v &= \Psi(u, v).
\end{align*}
\]
\[ SI(R) \text{ model} \quad \rightarrow \quad \text{General paradigm} \]

\[
\begin{align*}
I(t, x) : & \text{ fraction of Infected,} \\
S(t, x) : & \text{ fraction of Susceptibles}
\end{align*}
\]

\[
\begin{align*}
\partial_t I - d_1 \Delta_x I &= \beta SI - \gamma I, \\
\partial_t S - d_2 \Delta_x S &= -\beta SI.
\end{align*}
\]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= \Phi(u, v), \\
\partial_t v - d_2 \Delta_x v &= \Psi(u, v).
\end{align*}
\]
General paradigm: same kind of equations in various contexts.

- **Epidemiology**: $u =$ Infected, $v =$ Susceptibles.
- Propagation in excitable media: flame in solid combustion:
  $u =$ temperature, $v =$ chemical fuel
- Population dynamics: Lotka-Volterra.
  $u =$ predators, $v =$ preys
- Bass model in marketing: $u =$ fraction of adopters of a product in a potential market; $v =$ fraction of non-adopters.
- Other contexts: Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

All these situations are instances of propagation in excitable media.

Another example is the epidemiological approach to model collective behaviors in social sciences.
General paradigm: same kind of equations in various contexts.

- **Epidemiology**: $u = $ Infected, $v = $ Susceptibles.
- **Propagation in excitable media**: flame in solid combustion:
  $u = $ temperature, $v = $ chemical fuel

\[
\begin{align*}
\frac{du}{dt} &= \beta F(u)v, \\
\frac{dv}{dt} &= -F(u)v.
\end{align*}
\]

- **Arrhenius law**: $F(u) \times v$; $\beta > 0$; $F(u < \theta) = 0$, $F(u > \theta) > 0$.
- **Population dynamics**: Lotka-Volterra.
  $u = $ predators, $v = $ preys
- **Bass model** in marketing: $u = $ fraction of adopters of a product in a potential market; $v = $ fraction of non-adopters.
- **Other contexts**: Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

⇝ All these situations are instances of propagation in excitable media.
General paradigm: same kind of equations in various contexts.

- **Epidemiology**: \( u = \text{Infected}, \ v = \text{Susceptibles} \).
- **Propagation in excitable media**: flame in solid combustion:
  \( u = \text{temperature}, \ v = \text{chemical fuel} \)
  \[
  \begin{align*}
  \frac{du}{dt} &= \beta F(u) v, \\
  \frac{dv}{dt} &= -F(u) v.
  \end{align*}
  \]
  \( F(u) \times v \text{ Arrhenius law} \); \( \beta > 0 \); \( F(u < \theta) = 0, \ F(u > \theta) > 0 \).
- **Population dynamics**: Lotka-Volterra.
  \( u = \text{predators}, \ v = \text{preys} \)
- **Bass model** in marketing: \( u = \text{fraction of adopters of a product in a potential market} \); \( v = \text{fraction of non-adopters} \).
- **Other contexts**: Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

\( \Rightarrow \) All these situations are instances of propagation in excitable media
General paradigm: same kind of equations in various contexts.

- **Epidemiology**: $u = \text{Infected}$, $v = \text{Susceptibles}$.
- **Propagation in excitable media**: flame in solid combustion:
  $u = \text{temperature}$, $v = \text{chemical fuel}$
- **Population dynamics**: Lotka-Volterra.
  $u = \text{predators}$, $v = \text{preys}$
  \[
  \begin{align*}
  \frac{du}{dt} &= \beta uv - \omega u, \\
  \frac{dv}{dt} &= \alpha u - \delta uv.
  \end{align*}
  \]
  Predation; intrinsic death; intrinsic growth
- **Bass model** in marketing: $u = \text{fraction of adopters of a product in a potential market}$; $v = \text{fraction of non-adopters}$.
- **Other contexts**: Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

⇝ All these situations are instances of propagation in excitable media → another example is the epidemiological approach to model collective behaviors in social sciences.
**General paradigm:** same kind of equations in various contexts.

- **Epidemiology:** $u = \text{Infected}, \ v = \text{Susceptibles}.$
- **Propagation in excitable media:** flame in solid combustion:
  - $u =$ temperature, $v =$ chemical fuel
- **Population dynamics:** Lotka-Volterra.
  - $u =$ predators, $v =$ preys
  \[
  \begin{align*}
  \frac{du}{dt} &= \beta uv - \omega u, \\
  \frac{dv}{dt} &= \alpha u - \delta uv.
  \end{align*}
  \]
  - Predation; intrinsic death; intrinsic growth
- **Bass model** in marketing: $u =$ fraction of adopters of a product in a potential market; $v =$ fraction of non-adopters.
- **Other contexts:** Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

\[\Rightarrow\text{All these situations are instances of propagation in excitable media}\]
**General paradigm:** same kind of equations in various contexts.

- **Epidemiology:** $u =$ **Infected**, $v =$ **Susceptibles**.
- **Propagation in excitable media:** flame in solid combustion: $u =$ temperature, $v =$ chemical fuel
- **Population dynamics:** Lotka-Volterra. $u =$ predators, $v =$ preys
- **Bass model** in marketing: $u =$ **fraction of adopters** of a product in a potential market; $v =$ **fraction of non-adopters**.

\[
\begin{align*}
\frac{du}{dt} &= \beta uv + \alpha v, \\
\frac{dv}{dt} &= -\beta uv - \alpha v.
\end{align*}
\]

$\beta uv$ adoption via word-of-mouth; $\alpha v$ adoption via **advertisement**

- **Other contexts:** Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

⇝ All these situations are instances of **propagation in excitable media**
General paradigm: same kind of equations in various contexts.

- **Epidemiology:** $u = $ Infected, $v = $ Susceptibles.
- **Propagation in excitable media:** flame in solid combustion: $u =$ temperature, $v =$ chemical fuel
- **Population dynamics:** Lotka-Volterra.
  $u =$ predators, $v =$ preys
- **Bass model** in marketing: $u =$ fraction of adopters of a product in a potential market ; $v =$ fraction of non-adopters.

$$\begin{align*}
\frac{du}{dt} &= \beta uv + \alpha v, \\
\frac{dv}{dt} &= -\beta uv - \alpha v.
\end{align*}$$

$\beta uv$ adoption via word-of-mouth ; $\alpha v$ adoption via advertisement

- **Other contexts:** Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

⇝ All these situations are instances of propagation in excitable media
**General paradigm:** same kind of equations in various contexts.

- **Epidemiology:** \( u = \text{Infected}, \ v = \text{Susceptibles}. \)
- **Propagation in excitable media:** flame in solid combustion:
  \( u = \text{temperature}, \ v = \text{chemical fuel} \)
- **Population dynamics:** Lotka-Volterra.
  \( u = \text{predators}, \ v = \text{preys} \)
- **Bass model** in marketing: \( u = \text{fraction of adopters of a product in a potential market}, \ v = \text{fraction of non-adopters}. \)
- **Other contexts:** Belousov-Zhabotinski biochemical reaction,
  host/parasite cooperative system, etc.

\[\text{⇝ All these situations are instances of propagation in excitable media}\]

\[\rightarrow \text{another example is the epidemiological approach to model collective behaviors in social sciences.}\]
General paradigm: same kind of equations in various contexts.

- Epidemiology: \( u = \text{Infected}, \ v = \text{Susceptibles} \).
- Propagation in excitable media: flame in solid combustion: \( u = \text{temperature}, \ v = \text{chemical fuel} \).
- Population dynamics: Lotka-Volterra. \( u = \text{predators}, \ v = \text{preys} \).
- Bass model in marketing: \( u = \text{fraction of adopters} \) of a product in a potential market; \( v = \text{fraction of non-adopters} \).
- Other contexts: Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

\( \Rightarrow \) All these situations are instances of propagation in excitable media.

\( \Rightarrow \) another example is the epidemiological approach to model collective behaviors in social sciences.
General paradigm: same kind of equations in various contexts.

- **Epidemiology**: \( u = \text{Infected}, \ v = \text{Susceptibles} \).
- **Propagation in excitable media**: flame in solid combustion: \( u = \text{temperature}, \ v = \text{chemical fuel} \).
- **Population dynamics**: Lotka-Volterra. \( u = \text{predators}, \ v = \text{preys} \).
- **Bass model** in marketing: \( u = \text{fraction of adopters of a product in a potential market}; \ v = \text{fraction of non-adopters} \).
- **Other contexts**: Belousov-Zhabotinski biochemical reaction, host/parasite cooperative system, etc.

\[ \Rightarrow \] All these situations are instances of propagation in excitable media.

\[ \rightarrow \] another example is the epidemiological approach to model collective behaviors in social sciences.
Epidemiological modeling of collective behaviors: the example of riots
Epidemiological models for collective behaviors.

- Analogy between contagion and social imitation. 
  [Granovetter 1978, Threshold models of collective behaviors]

- Many instances:
  - Spreading of memes - [Wang & Wood 2011]
  - Spreading of hashtags - [Skaza & Blais 2017]
  - Spreading of ideas - [Bettencourt et al. 2006]
  - Spreading of rumors - [Kawachi 2008]
  - Spreading of extreme ideologies - [Santonjaa et al. 2008]
  - ...

- Dynamics of riots and social unrest
Epidemiological models for collective behaviors.

- Analogy between contagion and social imitation.  
  [Granovetter 1978, Threshold models of collective behaviors]

- Many instances:
  - Spreading of memes - [Wang & Wood 2011]
  - Spreading of hashtags - [Skaza & Blais 2017]
  - Spreading of ideas - [Bettencourt et al. 2006]
  - Spreading of rumors - [Kawachi 2008]
  - Spreading of extreme ideologies - [Santonjaa et al. 2008]
  - ...

- Dynamics of riots and social unrest
Epidemiological models for collective behaviors.

- Analogy between contagion and social imitation. 
  [Granovetter 1978, Threshold models of collective behaviors]

- Many instances:
  - Spreading of memes - [Wang & Wood 2011]
  - Spreading of hashtags - [Skaza & Blais 2017]
  - Spreading of ideas - [Bettencourt et al. 2006]
  - Spreading of rumors - [Kawachi 2008]
  - Spreading of extreme ideolologies - [Santonjaa et al. 2008]
  - ...

- Dynamics of riots and social unrest
Epidemiological models for collective behaviors.

- Analogy between *contagion* and *social imitation*.
  
  \[\text{Granovetter 1978, Threshold models of collective behaviors}\]

- Many instances:
  - Spreading of memes - \[\text{Wang & Wood 2011}\]
  - Spreading of hashtags - \[\text{Skaza & Blais 2017}\]
  - Spreading of ideas - \[\text{Bettencourt et al. 2006}\]
  - Spreading of rumors - \[\text{Kawachi 2008}\]
  - Spreading of extreme ideologies - \[\text{Santonjaa et al. 2008}\]
  - ...
Epidemiological models for collective behaviors.

- Analogy between **contagion** and **social imitation**.
  
  [Granovetter 1978, *Threshold models of collective behaviors*]

- Many instances:
  - Spreading of memes - [Wang & Wood 2011]
  - Spreading of hashtags - [Skaza & Blais 2017]
  - Spreading of ideas - [Bettencourt et al. 2006]
  - Spreading of rumors - [Kawachi 2008]
  - Spreading of extreme ideologies - [Santonjaa et al. 2008]
  - ... 

- **Dynamics of riots and social unrest**
Epidemiological models for collective behaviors.

- [Granovetter 1978, *Threshold models of collective behaviors*]

“There is also some similarity between the present models and models used in epidemiology (Bailey 1976), the diffusion of information (Bartholomew 1967) and innovations (Hamblin, Jacobsen, and Miller 1973), and the evolution of behavior in groups over time (Coleman 1965). To develop these analogies in more detail would require that (1) my models [...] be translated into differential equations in continuous time, and that (2) some way be found to introduce the "threshold" concept into these other models, which generally do not stress individual differences.”
Epidemiological modeling of riots

- [Burbeck et. al. (1978), *The dynamics of riot growth: an epidemiological approach*]

  “Patterns within three major riots suggest that the dynamics of the spread of riot behavior during a riot can be fruitfully compared to those operative in classical epidemics. We therefore conceptualize riots as behavioral epidemics, and apply the mathematical theory of epidemics [...].”

- Incidentally, it points to the social nature of epidemics.
  → Institute of Development Studies [MacGregor et. al. 2020]:

  “As the COVID-19 pandemic rages across the world, one thing is clear: this epidemic, like all others, is a social phenomenon. The dynamics of the virus, infection and immunity, not to mention on-going efforts to revise and improve clinical care [...], are a critical part of the unfolding story. So, too, are peoples’ social responses to the disease and interactions with each other.”
Epidemiological modeling of riots

• [Burbeck et. al. (1978), The dynamics of riot growth: an epidemiological approach]

“Patterns within three major riots suggest that the dynamics of the spread of riot behavior during a riot can be fruitfully compared to those operative in classical epidemics. We therefore conceptualize riots as behavioral epidemics, and apply the mathematical theory of epidemics [...].”

• Incidentally, it points to the social nature of epidemics.
  → Institute of Development Studies [MacGregor et. al. 2020]:

“As the COVID-19 pandemic rages across the world, one thing is clear: this epidemic, like all others, is a social phenomenon. The dynamics of the virus, infection and immunity, not to mention on-going efforts to revise and improve clinical care [...], are a critical part of the unfolding story. So, too, are peoples’ social responses to the disease and interactions with each other.”
Epidemiological modeling of riots

- [Burbeck et. al. (1978), The dynamics of riot growth: an epidemiological approach]

“Patterns within three major riots suggest that the dynamics of the spread of riot behavior during a riot can be fruitfully compared to those operative in classical epidemics. We therefore conceptualize riots as behavioral epidemics, and apply the mathematical theory of epidemics [...]”

- Incidentally, it points to the social nature of epidemics.
  → Institute of Development Studies [MacGregor et. al. 2020]:

“As the COVID-19 pandemic rages across the world, one thing is clear: this epidemic, like all others, is a social phenomenon. The dynamics of the virus, infection and immunity, not to mention on-going efforts to revise and improve clinical care [...], are a critical part of the unfolding story. So, too, are peoples’ social responses to the disease and interactions with each other.”
Epidemiological modeling of riots

- [Burbeck et. al. (1978), The dynamics of riot growth: an epidemiological approach]

“Patterns within three major riots suggest that the dynamics of the spread of riot behavior during a riot can be fruitfully compared to those operative in classical epidemics. We therefore conceptualize riots as behavioral epidemics, and apply the mathematical theory of epidemics [...].”

- Incidentally, it points to the social nature of epidemics.
  → Institute of Development Studies [MacGregor et. al. 2020]:

“As the COVID-19 pandemic rages across the world, one thing is clear: this epidemic, like all others, is a social phenomenon. The dynamics of the virus, infection and immunity, not to mention on-going efforts to revise and improve clinical care [...], are a critical part of the unfolding story. So, too, are peoples’ social responses to the disease and interactions with each other.”
Epidemiological modeling of riots

- [Burbeck et. al. (1978), *The dynamics of riot growth: an epidemiological approach*]

  “*Patterns within three major riots suggest that the dynamics of the spread of riot behavior during a riot can be fruitfully compared to those operative in classical epidemics. We therefore conceptualize riots as behavioral epidemics, and apply the mathematical theory of epidemics […].*”

- Incidentally, it points to the social nature of epidemics.
  → Institute of Development Studies [MacGregor et. al. 2020]:

  “*As the COVID-19 pandemic rages across the world, one thing is clear: this epidemic, like all others, is a social phenomenon. The dynamics of the virus, infection and immunity, not to mention on-going efforts to revise and improve clinical care […], are a critical part of the unfolding story. So, too, are peoples’ social responses to the disease and interactions with each other.*”
Epidemiological modeling of riots: literature.

- [Berestycki, Rodriguez, Nadal 2015]
- [Bonasse-Gahot, Berestycki, Nadal et. al. Scientific Reports 2018]:
  - data of 2005 French riot show a “wave-like” spatial propagation of rioting activity.
  - the dynamics can be reproduced by an epidemiology model with few parameters.
  - Spatial dynamics is crucial.

Main modeling features:

- Social movements are triggered by a single (small) triggering event in a context of high social tension.
  
  "The stroke that broke the camel’s back."

- Self-reinforcement mechanism: social imitation.
  
  "The more people on the street, the more prone one is to join."

- Spatial propagation
  - movement of rioters
  - diffusion of social tension through direct communication or media
Epidemiological modeling of riots: literature.

- [Berestycki, Rodriguez, Nadal 2015]
- [Bonasse-Gahot, Berestycki, Nadal et. al. Scientific Reports 2018]:
  - data of 2005 French riot show a “wave-like” spatial propagation of rioting activity.
  - the dynamics can be reproduced by an epidemiology model with few parameters.
  - Spatial dynamics is crucial.

Main modeling features:

- Social movements are triggered by a single (small) triggering event in a context of high social tension.
  - “The stroke that broke the camel’s back.”
- Self-reinforcement mechanism: social imitation.
  - “The more people on the street, the more prone one is to join.”
- Spatial propagation
  - movement of rioters
  - diffusion of social tension through direct communication or media
Epidemiological modeling of riots: literature.

- [Berestycki, Rodriguez, Nadal 2015]
- [Bonasse-Gahot, Berestycki, Nadal et. al. Scientific Reports 2018]:
  - data of 2005 French riot show a “wave-like” spatial propagation of rioting activity.
  - the dynamics can be reproduced by an epidemiology model with few parameters.
  - Spatial dynamics is crucial.

Main modeling features:

- Social movements are triggered by a single (small) triggering event in a context of high social tension.
  “The stroke that broke the camel’s back.”
- Self-reinforcement mechanism: social imitation.
  “The more people on the street, the more prone one is to join.”
- Spatial propagation
  - movement of rioters
  - diffusion of social tension through direct communication or media
Epidemiological modeling of riots: literature.

- [Berestycki, Rodriguez, Nadal 2015]
- [Bonasse-Gahot, Berestycki, Nadal et. al. Scientific Reports 2018]:
  - data of 2005 French riot show a “wave-like” spatial propagation of rioting activity.
  - the dynamics can be reproduced by an epidemiology model with few parameters.
  - Spatial dynamics is crucial.

Main modeling features:

- Social movements are triggered by a single (small) triggering event in a context of high social tension.
  “The stroke that broke the camel’s back.”
- Self-reinforcement mechanism: social imitation.
  “The more people on the street, the more prone one is to join.”
- Spatial propagation
  - movement of rioters
  - diffusion of social tension through direct communication or media
Epidemiological modeling of riots: literature.

- [Berestycki, Rodriguez, Nadal 2015]
- [Bonasse-Gahot, Berestycki, Nadal et. al. Scientific Reports 2018]:
  - data of 2005 French riot show a “wave-like” spatial propagation of rioting activity.
  - the dynamics can be reproduced by an epidemiology model with few parameters.
  - Spatial dynamics is crucial.

Main modeling features:

- Social movements are triggered by a single (small) triggering event in a context of high social tension.
  “The stroke that broke the camel’s back.”

- Self-reinforcement mechanism: social imitation.
  “The more people on the street, the more prone one is to join.”

- Spatial propagation
  - movement of rioters
  - diffusion of social tension through direct communication or media
Epidemiological modeling of riots: literature.

- [Berestycki, Rodriguez, Nadal 2015]
- [Bonasse-Gahot, Berestycki, Nadal et. al. Scientific Reports 2018]:
  - data of 2005 French riot show a “wave-like” spatial propagation of rioting activity.
  - the dynamics can be reproduced by an epidemiology model with few parameters.
  - Spatial dynamics is crucial.

Main modeling features:

- Social movements are triggered by a single (small) triggering event in a context of high social tension.
  - “The stroke that broke the camel’s back.”
- Self-reinforcement mechanism: social imitation.
  - “The more people on the street, the more prone one is to join.”
- Spatial propagation
  - movement of rioters
  - diffusion of social tension through direct communication or media
Epidemiological modeling of riots: literature.

- [Berestycki, Rodriguez, Nadal 2015]
- [Bonasse-Gahot, Berestycki, Nadal et. al. Scientific Reports 2018]:
  - data of 2005 French riot show a “wave-like” spatial propagation of rioting activity.
  - the dynamics can be reproduced by an epidemiology model with few parameters.
  - Spatial dynamics is crucial.

Main modeling features:

- Social movements are triggered by a single (small) triggering event in a context of high social tension.
  “The stroke that broke the camel’s back.”
- Self-reinforcement mechanism: social imitation.
  “The more people on the street, the more prone one is to join.”
- Spatial propagation
  - movement of rioters
  - diffusion of social tension through direct communication or media
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/rioting activity $u$
    (corresponds to $I$ in the $SI$ model)
  - Social tension/susceptibility $v$
    (corresponds to $S$ in the $SI$ model)
- $u$ is explicit (intensity of the riot) ; $v$ is implicit
- Triggering event perturbs the system at equilibrium at $t = 0$.
- Dynamics of $u$: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- Retroaction on $v$. 3 cases:
  - Inhibiting: rioting consumes social tension.
    \( \Rightarrow \) Describes ephemeral movements (Paris 2005, London 2011)
    Formally contains $SIR$ (infection consumes susceptibles).
  - Enhancing: rioting increases social tension.
    \( \Rightarrow \) Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    Formally contains cooperative systems.

\( \Rightarrow \) Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/rioting activity $u$ (corresponds to $I$ in the $SI$ model)
  - Social tension/susceptibility $v$ (corresponds to $S$ in the $SI$ model)

- $u$ is explicit (intensity of the riot) ; $v$ is implicit

- Triggering event perturbs the system at equilibrium $a t = 0$.

- Dynamics of $u$: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.

- Retroaction on $v$. 3 cases:
  - Inhibiting: rioting consumes social tension.
    - Formally contains $SIR$ (infection consumes susceptibles).
  - Enhancing: rioting increases social tension.
    - Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    - Formally contains cooperative systems.

Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - **Level of unrest/rioting activity** $u$
  - **Social tension/susceptibility** $v$
- $u$ is explicit (intensity of the riot); $v$ is implicit
- **Triggering event** perturbs the system at equilibrium at $t = 0$.
- **Dynamics of** $u$: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- **Retroaction** on $v$. 3 cases:
  - **Inhibiting**: rioting consumes social tension.
    $\rightarrow$ Describes ephemeral movements (Paris 2005, London 2011)
    Formally contains $SIR$ (infection consumes susceptibles).
  - **Enhancing**: rioting increases social tension.
    $\rightarrow$ Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    Formally contains cooperative systems.
  - **Mixed**: more realistic. Various behaviors.

$\rightarrow$ Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/rioting activity $u$
  - Social tension/susceptibility $v$
- $u$ is explicit (intensity of the riot) ; $v$ is implicit
- **Triggering event** perturbs the system at equilibrium at $t = 0$.
- **Dynamics of $u$**: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- **Retroaction on $v$**: 3 cases:
  - **Inhibiting**: rioting consumes social tension.
    - Formally contains SIR (infection consumes susceptibles).
  - **Enhancing**: rioting increases social tension.
    - Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    - Formally contains cooperative systems.
  - **Mixed**: more realistic. Various behaviors.

$\Rightarrow$ Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- **Two fields**:  
  - **Level of unrest/rioting activity** $u$  
  - **Social tension/susceptibility** $v$

- $u$ is explicit (intensity of the riot) ; $v$ is implicit

- **Triggering event** perturbs the system at equilibrium at $t = 0$.

- **Dynamics of $u$**: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.

- **Retroaction on $v$**. 3 cases:
  - **Inhibiting**: rioting consumes social tension.  
    $\implies$ Describes ephemeral movements (Paris 2005, London 2011)  
    Formally contains $SIR$ (infection consumes susceptibles).
  - **Enhancing**: rioting increases social tension.  
    $\implies$ Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)  
    Formally contains cooperative systems.
  - **Mixed**: more realistic. Various behaviors.

$\implies$ Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- **Two fields:**
  - **Level of unrest/rioting activity** \( u \)
  - **Social tension/susceptibility** \( v \)

- \( u \) is explicit (intensity of the riot) ; \( v \) is implicit

- **Triggering event** perturbs the system at equilibrium at \( t = 0 \).

- **Dynamics of** \( u \): Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.

- **Retroaction** on \( v \). 3 cases:
  - **Inhibiting**: rioting consumes social tension.
    \( \leadsto \) Describes ephemeral movements (Paris 2005, London 2011)
    Formally contains \( SIR \) (infection consumes susceptibles).
  - **Enhancing**: rioting increases social tension.
    \( \leadsto \) Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    Formally contains cooperative systems.
  - **Mixed**: more realistic. Various behaviors.

\( \leadsto \) Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/rioting activity $u$
  - Social tension/susceptibility $v$

- $u$ is explicit (intensity of the riot); $v$ is implicit.

- Triggering event perturbs the system at equilibrium at $t = 0$.

- Dynamics of $u$:
  - Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.

- Retroaction on $v$. 3 cases:
  - Inhibiting: rioting consumes social tension.
    $\Rightarrow$ Describes ephemeral movements (Paris 2005, London 2011)
    Formally contains $SIR$ (infection consumes susceptibles).
  - Enhancing: rioting increases social tension.
    $\Rightarrow$ Describes time-persisting movements or lasting upheaval (Arab
    Spring, Yellow Vest movement)
    Formally contains cooperative systems.

$\Rightarrow$ Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - **Level of unrest/riot activity** $u$
  - **Social tension/susceptibility** $v$
- $u$ is explicit (intensity of the riot); $v$ is implicit.
- **Triggering event** perturbs the system at equilibrium $t = 0$.
- **Dynamics of $u$**: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- **Retroaction** on $v$. 3 cases:
  - **Inhibiting**: rioting consumes social tension.
    - Formally contains SIR (infection consumes susceptibles).
  - **Enhancing**: rioting increases social tension.
    - Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    - Formally contains cooperative systems.
  - **Mixed**: more realistic. Various behaviors.

  $\implies$ Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/rioting activity $u$
  - Social tension/susceptibility $v$

- $u$ is explicit (intensity of the riot) ; $v$ is implicit

- Triggering event perturbs the system at equilibrium at $t = 0$.

- Dynamics of $u$:
  - Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.

- Retroaction on $v$. 3 cases:
  - Inhibiting: rioting consumes social tension.
    $\Rightarrow$ Describes ephemeral movements (Paris 2005, London 2011)
    Formally contains $SIR$ (infection consumes susceptibles).
  - Enhancing: rioting increases social tension.
    $\Rightarrow$ Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    Formally contains cooperative systems.

$\Rightarrow$ Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- **Two fields:**
  - Level of unrest/riot activity $u$
  - Social tension/susceptibility $v$
- $u$ is explicit (intensity of the riot) ; $v$ is implicit
- **Triggering event** perturbs the system at equilibrium at $t = 0$.
- **Dynamics of $u$**: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- **Retroaction** on $v$. 3 cases:
  - **Inhibiting**: rioting consumes social tension.
    - Formally contains $SIR$ (infection consumes susceptibles).
  - **Enhancing**: rioting increases social tension.
    - Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    - Formally contains cooperative systems.
  - **Mixed**: more realistic. Various behaviors.

$\Rightarrow$ Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/rioting activity $u$
  - Social tension/susceptibility $v$
- $u$ is explicit (intensity of the riot) ; $v$ is implicit
- **Triggering event** perturbs the system at equilibrium at $t = 0$.
- **Dynamics of** $u$: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- **Retroaction** on $v$. 3 cases:
  - Inhibiting: rioting consumes social tension.
    $\Rightarrow$ Describes ephemeral movements (Paris 2005, London 2011)
    Formally contains SIR (infection consumes susceptibles).
  - Enhancing: rioting increases social tension.
    $\Rightarrow$ Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    Formally contains cooperative systems.

$\Rightarrow$ Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/riot activity \( u \)
  - Social tension/susceptibility \( v \)
- \( u \) is explicit (intensity of the riot); \( v \) is implicit
- Triggering event perturbs the system at equilibrium at \( t = 0 \).
- Dynamics of \( u \): Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- Retroaction on \( v \). 3 cases:
  - Inhibiting: rioting consumes social tension.
    - Formally contains SIR (infection consumes susceptibles).
  - Enhancing: rioting increases social tension.
    - Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement)
    - Formally contains cooperative systems.

\( \rightsquigarrow \) Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/riot activity \( u \)
  - Social tension/susceptibility \( v \)
- \( u \) is explicit (intensity of the riot); \( v \) is implicit.
- Triggering event perturbs the system at equilibrium at \( t = 0 \).
- Dynamics of \( u \): Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- Retroaction on \( v \). 3 cases:
  - Inhibiting: rioting consumes social tension.
    Formally contains SIR (infection consumes susceptibles).
  - Enhancing: rioting increases social tension.
    Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movement).
    Formally contains cooperative systems.
- Fit the epidemiology paradigm.
Epidemiological modeling of riots: the approach

- Two fields:
  - Level of unrest/rioting activity $u$
  - Social tension/susceptibility $v$
- $u$ is explicit (intensity of the riot) ; $v$ is implicit
- Triggering event perturbs the system at equilibrium at $t = 0$.
- Dynamics of $u$: Endogeneous growth (social imitation), saturation, relaxation (fatigue), diffusion, etc.
- Retroaction on $v$. 3 cases:
  - Inhibiting: rioting consumes social tension.  
    $\Rightarrow$ Describes ephemeral movements (Paris 2005, London 2011) 
    Formally contains SIR (infection consumes susceptibles).
  - Enhancing: rioting increases social tension.  
    $\Rightarrow$ Describes time-persisting movements or lasting upheaval (Arab Spring, Yellow Vest movment) 
    Formally contains cooperative systems.

$\Rightarrow$ Fit the epidemiology paradigm.
Mathematical framework &
main results
General Framework:

\[ u(t, x) \]: level of activity \hspace{1cm} \rightarrow \hspace{1cm} \nabla v(t, x) \]: level of susceptibility

\[
\begin{align*}
\frac{\partial_t u}{d_1} - \Delta_x u &= u F(u, v), \\
\frac{\partial_t v}{d_2} - \Delta_x v &= u G(u, v) + (v_b - v) H(u, v).
\end{align*}
\]

\[ (0, v_b) \] is a steady state.

Setting:

- \( (0, v_b) \) is stable when \( u \equiv 0 \Rightarrow \) we assume \( H \geq 0 \).
- Triggering event at \( t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b) \) comp. supp.
- Dynamics of \( u \): \( uF(u, v) \)
- Retroaction on \( v \): \( uG(u, v) + (v_b - v) H(u, v) \)

Assumptions:

- \( v \mapsto F(u, v) \) \( \uparrow \) ("\( v \) fuels \( u \)"") (for simplicity)
- \( d_1 > 0, d_2 \geq 0 \)
- \( u, v \) remain bounded: saturation

Questions:
**General Framework:**

\[ u(t, x): \text{level of activity}; \quad v(t, x): \text{level of susceptibility} \]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*}
\]

\( (0, v_b) \) is a **steady state**.

**Setting:**

- \((0, v_b)\) is stable when \(u \equiv 0 \Rightarrow \) we assume \(H \geq 0\).
- **Triggering event** at \(t = 0 \Rightarrow \) \((u_0, v_0) \approx (0, v_b)\) comp. supp.
- **Dynamics of** \(u\): \(uF(u, v)\)
- **Retroaction on** \(v\): \(uG(u, v) + (v_b - v)H(u, v)\)

**Assumptions:**

- \(v \mapsto F(u, v) \uparrow \) (“\(v\) fuels \(u\)”) (for simplicity)
- \(d_1 > 0, \ d_2 \geq 0\)
- \(u, v\) remain bounded: **saturation**

**Questions:**
General Framework:

\[ u(t, x) : \text{level of activity} \quad ; \quad v(t, x) : \text{level of susceptibility} \]

\[
\begin{aligned}
\begin{cases}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{cases}
\end{aligned}
\]

\( (0, v_b) \) is a steady state.

Setting:

- \( (0, v_b) \) is stable when \( u \equiv 0 \Rightarrow \) we assume \( H \geq 0 \).
- Triggering event at \( t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b) \) comp. supp.
- Dynamics of \( u \): \( uF(u, v) \)
- Retroaction on \( v \): \( uG(u, v) + (v_b - v)H(u, v) \)

Assumptions:

- \( v \mapsto F(u, v) \mapsto \) ("\( v \) fuels \( u \)"") (for simplicity)
- \( d_1 > 0, d_2 \geq 0 \)
- \( u, v \) remain bounded: saturation

Questions:
General Framework:

\[ u(t, x) : \text{level of activity} \quad ; \quad v(t, x) : \text{level of susceptibility} \]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*}
\]

\( (0, v_b) \) is a steady state.

Setting:

- \((0, v_b)\) is stable when \(u \equiv 0 \Rightarrow \) we assume \(H \geq 0\).
- Triggering event at \(t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b)\) comp. supp.
- Dynamics of \(u\): \(uF(u, v)\)
- Retroaction on \(v\): \(uG(u, v) + (v_b - v)H(u, v)\)

Assumptions:

- \(v \mapsto F(u, v) \uparrow \) (“\(v\) fuels \(u\)”) (for simplicity)
- \(d_1 > 0, d_2 \geq 0\)
- \(u, v\) remain bounded: saturation

Questions:
General Framework:

\[ u(t, x) : \text{level of activity} \quad ; \quad v(t, x) : \text{level of susceptibility} \]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*}
\]

\( (0, v_b) \) is a steady state.

Setting:

- \( (0, v_b) \) is stable when \( u \equiv 0 \Rightarrow \) we assume \( H \geq 0 \).
- Triggering event at \( t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b) \) comp. supp.
- Dynamics of \( u \): \( uF(u, v) \)
- Retroaction on \( v \): \( uG(u, v) + (v_b - v)H(u, v) \)

Assumptions:

- \( v \mapsto F(u, v) \uparrow \) ("\( v \) fuels \( u \")) (for simplicity)
- \( d_1 > 0, \ d_2 \geq 0 \)
- \( u, v \) remain bounded: saturation

Questions:
General Framework:

$u(t, x)$: level of activity; $v(t, x)$: level of susceptibility

\[
\begin{aligned}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{aligned}
\]

$(0, v_b)$ is a steady state.

Setting:

- $(0, v_b)$ is stable when $u \equiv 0 \Rightarrow$ we assume $H \geq 0$.
- Triggering event at $t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b)$ comp. supp.
- Dynamics of $u$: $uF(u, v)$
  - Retroaction on $v$: $uG(u, v) + (v_b - v)H(u, v)$

Assumptions:

- $v \mapsto F(u, v)$ ("$v$ fuels $u$") (for simplicity)
- $d_1 > 0, d_2 \geq 0$
- $u, v$ remain bounded: saturation

Questions:
General Framework:

\[ u(t, x): \text{level of } \text{activity} \quad ; \quad v(t, x): \text{level of } \text{susceptibility} \]

\[ \begin{cases} \partial_t u - d_1 \Delta_x u = uF(u, v), \\ \partial_t v - d_2 \Delta_x v = uG(u, v) + (v_b - v)H(u, v). \end{cases} \]

\( (0, v_b) \) is a steady state.

Setting:

- \((0, v_b)\) is stable when \(u \equiv 0 \Rightarrow \) we assume \(H \geq 0\).
- Triggering event at \(t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b)\) comp. supp.
- Dynamics of \(u\): \(uF(u, v)\)
- Retroaction on \(v\): \(uG(u, v) + (v_b - v)H(u, v)\)

Assumptions:

- \(v \mapsto F(u, v) \uparrow (\text{"}v \text{ fuels } u\text{"})\) (for simplicity)
- \(d_1 > 0, \ d_2 \geq 0\)
- \(u, v\) remain bounded: saturation

Questions:
**General Framework:**

\[ u(t, x) : \text{level of activity} \quad ; \quad v(t, x) : \text{level of susceptibility} \]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*}
\]

\( (0, v_b) \) is a steady state.

**Setting:**

- \((0, v_b)\) is stable when \( u \equiv 0 \) \( \Rightarrow \) we assume \( H \geq 0 \).
- Triggering event at \( t = 0 \) \( \Rightarrow \) \((u_0, v_0) \approx (0, v_b)\) comp. supp.
- Dynamics of \( u \): \( uF(u, v) \)
- Retroaction on \( v \): \( uG(u, v) + (v_b - v)H(u, v) \)

**Assumptions:**

- \( v \mapsto F(u, v) \uparrow ("v fuels u") \) (for simplicity)
- \( d_1 > 0, d_2 \geq 0 \)
- \( u, v \) remain bounded: saturation

**Questions:**
General Framework:

\[ \begin{align*}
\dot{u}(t, x) & : \text{level of activity} ; \\
\dot{v}(t, x) & : \text{level of susceptibility}
\end{align*} \]

\[ \begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*} \]

\( (0, v_b) \) is a steady state.

Setting:

- \( (0, v_b) \) is stable when \( u \equiv 0 \Rightarrow \text{we assume } H \geq 0. \)
- Triggering event at \( t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b) \) comp. supp.
- Dynamics of \( u: uF(u, v) \)
- Retroaction on \( v: uG(u, v) + (v_b - v)H(u, v) \)

Assumptions:

- \( v \mapsto F(u, v) \uparrow \) ("\( v \) fuels \( u \"\)) (for simplicity)
- \( d_1 > 0, d_2 \geq 0 \)
- \( u, v \) remain bounded: saturation

Questions:
General Framework:

\[ u(t, x): \text{level of activity}; \quad v(t, x): \text{level of susceptibility} \]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*}
\]

\((0, v_b)\) is a steady state.

Setting:

- \((0, v_b)\) is stable when \(u \equiv 0 \Rightarrow \) we assume \(H \geq 0\).
- Triggering event at \(t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b)\) comp. supp.
- Dynamics of \(u\): \(uF(u, v)\)
- Retroaction on \(v\): \(uG(u, v) + (v_b - v)H(u, v)\)

Assumptions:

- \(v \mapsto F(u, v) \uparrow\) ("\(v\) fuels \(u\)"") (for simplicity)
- \(d_1 > 0, \ d_2 \geq 0\)
- \(u, v\) remain bounded: saturation

Questions:
General Framework:

$u(t, x)$: level of activity; $v(t, x)$: level of susceptibility

\[
\begin{cases}
\partial_t u - d_1 \Delta_x u = uF(u, v), \\
\partial_t v - d_2 \Delta_x v = uG(u, v) + (v_b - v)H(u, v).
\end{cases}
\]

$(0, v_b)$ is a steady state.

Setting:

- $(0, v_b)$ is stable when $u \equiv 0 \Rightarrow$ we assume $H \geq 0$.
- Triggering event at $t = 0 \Rightarrow (u_0, v_0) \approx (0, v_b)$ comp. supp.
- Dynamics of $u$: $uF(u, v)$
- Retroaction on $v$: $uG(u, v) + (v_b - v)H(u, v)$

Assumptions:

- $v \mapsto F(u, v) \uparrow$ ("$v$ fuels $u")$ (for simplicity)
- $d_1 > 0, d_2 \geq 0$
- $u, v$ remain bounded: saturation

Questions:
General Framework:

\[ u(t, x) : \text{level of activity}; \quad v(t, x) : \text{level of susceptibility} \]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*}
\]

\((0, v_b)\) is a steady state.

Setting:

Assumptions:

Questions:

- Which behavior can be generated with this framework?
- **Threshold** phenomenon on initial susceptibility \(v_0 \equiv v_b\)?
- Spatial propagation?
- Limit in long time?
General Framework:

\[ u(t, x) \]: level of activity ; \[ v(t, x) \]: level of susceptibility

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*}
\]

\( (0, v_b) \) is a steady state.

Setting:

Assumptions:

Questions:

- Which behavior can be generated with this framework?
- Threshold phenomenon on initial susceptibility \( v_0 \equiv v_b \)?
- Spatial propagation?
- Limit in long time?
General Framework:

\[ u(t, x) : \text{level of activity}; \quad v(t, x) : \text{level of susceptibility} \]

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v) , \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_b - v) H(u, v) .
\end{align*}
\]

\( (0, v_b) \) is a steady state.

Setting:

Assumptions:

Questions:

- Which behavior can be generated with this framework?
- **Threshold** phenomenon on initial susceptibility \( v_0 \equiv v_b \)?
- Spatial propagation?
- Limit in long time?
General Framework:

\[ u(t, x) \]: level of activity  
\[ v(t, x) \]: level of susceptibility

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_b - v) H(u, v).
\end{align*}
\]

\( (0, v_b) \) is a steady state.

Setting:

Assumptions:

Questions:

- Which behavior can be generated with this framework?
- **Threshold** phenomenon on initial susceptibility \( v_0 \equiv v_b \)?
- Spatial propagation?
- Limit in long time?
▷ General Framework:

\( u(t, x) \): level of activity; \( v(t, x) \): level of susceptibility

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v).
\end{align*}
\]

\( (0, v_b) \) is a steady state.

▷ Setting:

▷ Assumptions:

▷ Questions:

- Which behavior can be generated with this framework?
- **Threshold** phenomenon on initial susceptibility \( v_0 \equiv v_b \)?
- Spatial propagation?
- Limit in long time?
\[
\begin{aligned}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v),
\end{aligned}
\]

▷ **Threshold phenomenon**: on the sign of \( K_0 := F(0, v_0) \)

**Theorem (Threshold)**

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): return to calm.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): outburst of activity (Hair-trigger effect).

\[ \Rightarrow \text{Generalize the Threshold phenomenon to a very large class of models} \]

**Math. definition:**

- return to calm \( \iff \limsup_{t \to +\infty} |(u, v - v_0)| = 0 \)
- \( x \in \mathbb{R}^n \)
- outburst of activity \( \iff \limsup_{t \to +\infty} |(u, v - v_0)| > 0, \quad \forall U \subset \mathbb{R}^n. \)
\[
\begin{cases}
    \partial_t u - d_1 \Delta_x u = uF(u, v), \\
    \partial_t v - d_2 \Delta_x v = uG(u, v) + (v_0 - v)H(u, v),
\end{cases}
\]

\[\text{Threshold phenomenon: on the sign of } K_0 := F(0, v_0)\]

**Theorem (Threshold)**

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): return to calm.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): outburst of activity (Hair-trigger effect).

\[\text{Generalize the Threshold phenomenon to a very large class of models}\]

**Math. definition:**

- return to calm \(\iff\) \(\limsup_{t \to +\infty \atop x \in \mathbb{R}^n} |(u, v - v_0)| = 0\)
- outburst of activity \(\iff\) \(\limsup_{t \to +\infty \atop x \in U} |(u, v - v_0)| > 0, \quad \forall U \subset \mathbb{R}^n\).
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_0 - v) H(u, v),
\end{align*}
\]

\textbf{Threshold phenomenon}: on the sign of \( K_0 := F(0, v_0) \)

\textbf{Theorem (Threshold)}

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): \textit{return to calm}.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): \textit{outburst of activity} \((Hair-trigger effect)\).

\(\Rightarrow\) Generalize the \textit{Threshold phenomenon} to a very large class of models

\textbf{Math. definition:}

\[
\begin{align*}
\text{return to calm} \iff \limsup_{t \to +\infty, x \in \mathbb{R}^n} |(u, v - v_0)| = 0 \\
\text{outburst of activity} \iff \limsup_{t \to +\infty, x \in U} |(u, v - v_0)| > 0, \quad \forall U \subset \mathbb{R}^n.
\end{align*}
\]
\[
\begin{cases}
\partial_t u - d_1 \Delta_x u = u F(u, v), \\
\partial_t v - d_2 \Delta_x v = u G(u, v) + (v_0 - v) H(u, v),
\end{cases}
\]

**Threshold phenomenon**: on the sign of \( K_0 := F(0, v_0) \)

**Theorem (Threshold)**

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): return to calm.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): outburst of activity (Hair-trigger effect).

\( \Rightarrow \) Generalize the **Threshold phenomenon** to a very large class of models

**Math. definition:**

- \( \text{return to calm } \Leftrightarrow \limsup_{t \to +\infty, x \in \mathbb{R}^n} |(u, v - v_0)| = 0 \)
- \( \text{outburst of activity } \Leftrightarrow \limsup_{t \to +\infty, x \in U} |(u, v - v_0)| > 0, \quad \forall U \subset \mathbb{R}^n. \)
\[
\begin{aligned}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_0 - v) H(u, v),
\end{aligned}
\]

\textbf{Threshold phenomenon}: on the sign of \( K_0 := F(0, v_0) \)

\textbf{Theorem (Threshold)}

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): return to calm.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): outburst of activity (Hair-trigger effect).

\( \rightsquigarrow \) Generalize the \textbf{Threshold phenomenon} to a very large class of models

\textbf{Math. definition}:

- Return to calm \( \Leftrightarrow \limsup_{t \to +\infty} \limsup_{x \in \mathbb{R}^n} |(u, v - v_0)| = 0 \)
- Outburst of activity \( \Leftrightarrow \limsup_{t \to +\infty} \limsup_{x \in U} |(u, v - v_0)| > 0, \quad \forall U \subset \mathbb{R}^n \).
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v), 
\end{align*}
\]

Threshold phenomenon: on the sign of \( K_0 := F(0, v_0) \)

**Theorem (Threshold)**

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): return to calm.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): outburst of activity (Hair-trigger effect).

\( \Rightarrow \) Generalize the Threshold phenomenon to a very large class of models

**Remark:**

- sign of \( K_0 \): determines the stability of \((0, v_0)\).
- Generalize the threshold on \( R_0 \) in the SIR model.
- \( K_0 \) does not depend on the retroaction on \( v \)
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v),
\end{align*}
\]

\textbf{Threshold phenomenon :} on the sign of \( K_0 := F(0, v_0) \)

\textbf{Theorem (Threshold)}

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): return to calm.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): outburst of activity \((Hair-trigger effect)\).

\[\rightsquigarrow \text{Generalize the Threshold phenomenon to a very large class of models} \]

\textbf{Remark:}

- sign of \( K_0 \): determines the \textit{stability} of \((0, v_0)\).
- Generalize the threshold on \( R_0 \) in the \textit{SIR} model.
- \( K_0 \) does not depend on the \textit{retroaction} on \( v \)
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_0 - v) H(u, v),
\end{align*}
\]

\textbf{Threshold phenomenon}: on the sign of \( K_0 := F(0, v_0) \)

\textbf{Theorem (Threshold)}

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): return to calm.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): outburst of activity \((Hair-trigger effect)\).

\textbf{Remark}: 
- sign of \( K_0 \): determines the stability of \((0, v_0)\).
- Generalize the threshold on \( R_0 \) in the \textit{SIR} model.
- \( K_0 \) does not depend on the retroaction on \( v \).
\[
\begin{align*}
\frac{\partial}{\partial t} u - d_1 \Delta_x u &= uF(u, v), \\
\frac{\partial}{\partial t} v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v),
\end{align*}
\]

\textbf{Threshold phenomenon:} on the sign of \( K_0 := F(0, v_0) \)

\textbf{Theorem (Threshold)}

- If \( K_0 < 0 \) (low initial susceptibility \( v_0 \)): return to calm.
- If \( K_0 > 0 \) (high initial susceptibility \( v_0 \)): outburst of activity \((\text{Hair-trigger effect})\).

\( \Rightarrow \) Generalize the \textbf{Threshold phenomenon} to a very large class of models.

\textbf{Remark:}

- sign of \( K_0 \): determines the \textit{stability} of \((0, v_0)\).
- Generalize the threshold on \( R_0 \) in the \textit{SIR} model.
- \( K_0 \) does not depend on the \textit{retroaction} on \( v \).
\[
\begin{aligned}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v).
\end{aligned}
\]

Questions:

- Spatial propagation?
- Final state?

Depends on the parameters. Two sub-classes:

- **Inhibiting**: \( uG(u, v) + (v_b - v)H(u, v) \leq 0 \)
  
  \( \text{"activity consumes susceptibility"} \)

  (Formally contains the SIR model.)

- **Enhancing**: \( uG(u, v) + (v_b - v)H(u, v) \geq 0 \)
  
  \( \text{"activity increases susceptibility"} \)

  (Formally contains cooperative systems.)

- **General case**
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v).
\end{align*}
\]

**Questions:**

- **Spatial propagation?**
- **Final state?**

Depends on the parameters. Two sub-classes:

- **Inhibiting:** \( uG(u, v) + (v_b - v)H(u, v) \leq 0 \)
  
  "activity consumes susceptibility"
  
  (Formally contains the SIR model.)

- **Enhancing:** \( uG(u, v) + (v_b - v)H(u, v) \geq 0 \)
  
  "activity increases susceptibility"
  
  (Formally contains cooperative systems.)

- **General case**
\[
\begin{cases}
\frac{\partial_t u}{\partial t} - d_1 \Delta_x u = uF(u, v), \\
\frac{\partial_t v}{\partial t} - d_2 \Delta_x v = uG(u, v) + (v_0 - v)H(u, v).
\end{cases}
\]

**Questions:**

- **Spatial propagation?**
- **Final state?**

\[\Rightarrow\text{Depends on the parameters. Two sub-classes:}\]

- **Inhibiting:** \( uG(u, v) + (v_b - v)H(u, v) \leq 0 \)
  
  "activity consumes susceptibility"
  
  (Formally contains the *SIR* model.)

- **Enhancing:** \( uG(u, v) + (v_b - v)H(u, v) \geq 0 \)
  
  "activity increases susceptibility"
  
  (Formally contains cooperative systems.)

- **General case**
\[ \begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_0 - v) H(u, v). 
\end{align*} \]

Questions:

- Spatial propagation?
- Final state?

 Depends on the parameters. Two sub-classes:

- Inhibiting: \( u G(u, v) + (v_b - v) H(u, v) \leq 0 \)
  
  \( \text{“activity consumes susceptibility”} \)
  
  (Formally contains the \textit{SIR} model.)

- Enhancing: \( u G(u, v) + (v_b - v) H(u, v) \geq 0 \)
  
  \( \text{“activity increases susceptibility”} \)
  
  (Formally contains cooperative systems.)

- General case
\[ \begin{aligned} \partial_t u - d_1 \Delta_x u &= uF(u, v), \\ \partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v). \end{aligned} \]

Questions:

- **Spatial propagation?**
- **Final state?**

** Depends on the parameters. Two sub-classes:

- **Inhibiting:** \( uG(u, v) + (v_b - v)H(u, v) \leq 0 \)  
  "activity consumes susceptibility"  
  (Formally contains the SIR model.)

- **Enhancing:** \( uG(u, v) + (v_b - v)H(u, v) \geq 0 \)  
  "activity increases susceptibility"  
  (Formally contains cooperative systems.)

- **General case**
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_0 - v) H(u, v).
\end{align*}
\]

**Questions:**

- Spatial propagation?
- Final state?

\(\Rightarrow\) Depends on the parameters. Two sub-classes:

- **Inhibiting:** \(u G(u, v) + (v_0 - v) H(u, v) \leq 0\)
  
  "activity consumes susceptibility"

  (Formally contains the SIR model.)

- **Enhancing:** \(u G(u, v) + (v_0 - v) H(u, v) \geq 0\)
  
  "activity increases susceptibility"

  (Formally contains cooperative systems.)

- **General case**
\[
\begin{align*}
\frac{\partial}{\partial t} u - d_1 \Delta_x u &= u F(u, v), \\
\frac{\partial}{\partial t} v - d_2 \Delta_x v &= u G(u, v) + (v_0 - v) H(u, v).
\end{align*}
\]

Questions:

- Spatial propagation?
- Final state?

\(\implies\) Depends on the parameters. Two sub-classes:

- **Inhibiting**: \(u G(u, v) + (v_b - v) H(u, v) \leq 0\)
  
  "activity consumes susceptibility"

  (Formally contains the SIR model.)

- **Enhancing**: \(u G(u, v) + (v_b - v) H(u, v) \geq 0\)
  
  "activity increases susceptibility"

  (Formally contains cooperative systems.)

- **General case**
\[
\begin{aligned}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v).
\end{aligned}
\]

**Questions:**

- **Spatial propagation?**
- **Final state?**

→ Depends on the parameters. Two sub-classes:

- **Inhibiting:** \( uG(u, v) + (v_b - v)H(u, v) \leq 0 \)
  
  "activity consumes susceptibility"

  (Formally contains the SIR model.)

- **Enhancing:** \( uG(u, v) + (v_b - v)H(u, v) \geq 0 \)

  "activity increases susceptibility"

  (Formally contains cooperative systems.)

- **General case**
\[ \begin{aligned} \partial_t u - d_1 \Delta_x u &= uF(u, v), \\ \partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v). \end{aligned} \]

Questions:

- Spatial propagation?
- Final state?

> Depends on the parameters. Two sub-classes:

- **Inhibiting**: \( uG(u, v) + (v_b - v)H(u, v) \leq 0 \)
  
  \( \text{“activity consumes susceptibility”} \)
  
  (Formally contains the SIR model.)

- **Enhancing**: \( uG(u, v) + (v_b - v)H(u, v) \geq 0 \)
  
  \( \text{“activity increases susceptibility”} \)
  
  (Formally contains cooperative systems.)

- **General case**
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_0 - v)H(u, v).
\end{align*}
\]

**Questions:**

- Spatial propagation?
- Final state?

→ Depends on the parameters. Two sub-classes:

- **Inhibiting**: \(uG(u, v) + (v_b - v)H(u, v) \leq 0\)
  
  "activity consumes susceptibility"
  
  (Formally contains the SIR model.)

- **Enhancing**: \(uG(u, v) + (v_b - v)H(u, v) \geq 0\)
  
  "activity increases susceptibility"
  
  (Formally contains cooperative systems.)

- **General case**
\[
\begin{cases}
\partial_t u - d_1 \Delta_x u = u F(u, v), \\
\partial_t v - d_2 \Delta_x v = u G(u, v) + (v_0 - v) H(u, v).
\end{cases}
\]

**Questions:**

- **Spatial propagation?**
- **Final state?**

\[\Rightarrow\text{Depends on the parameters. Two sub-classes:}\]

- **Inhibiting:** \[u G(u, v) + (v_b - v) H(u, v) \leq 0\]
  
  "activity consumes susceptibility"

  (Formally contains the SIR model.)

- **Enhancing:** \[u G(u, v) + (v_b - v) H(u, v) \geq 0\]
  
  "activity increases susceptibility"

  (Formally contains cooperative systems.)

- **General case**
**Inhibiting case:**

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_b - v) H(u, v) \leq 0
\end{align*}
\]

**Theorem - Inhibiting case (spatial propagation)**

Assume \( K_0 = F(0, v_0) > 0 \) (i.e. \( v_0 \) large enough).

- Spatial propagation at speed \( c_0 := 2 \sqrt{d_1 K_0} \).
- Final state: \( u(t = +\infty) = 0, v(t = +\infty) = v_\infty \) (implicit).
- Existence of bumps/wave traveling at speed \( c > c_0 \); non-existence if \( 0 < c < c_0 \) or \( K_0 < 0 \).

\[\rightsquigarrow\] Generalize the classical results on the SI model

Rmk: \( c_0 \) does not depend on the retroaction on \( v \).
\[ \begin{align*}
&\partial_t u - d_1 \Delta x u = u F(u, v), \\
&\partial_t v - d_2 \Delta x v = u G(u, v) + (v_b - v) H(u, v) \leq 0
\end{align*} \]

**Theorem - Inhibiting case (spatial propagation)**

Assume \( K_0 = F(0, v_0) > 0 \) (i.e. \( v_0 \) large enough).

- Spatial propagation at speed \( c_0 := 2 \sqrt{d_1 K_0} \).
- Final state: \( u(t = +\infty) = 0, \ v(t = +\infty) = v_\infty \) (implicit).
- Existence of bumps/wave traveling at speed \( c > c_0 \); non-existence if \( 0 < c < c_0 \) or \( K_0 < 0 \).

\[ \Rightarrow \] Generalize the classical results on the SI model

Rmk: \( c_0 \) does not depend on the *retroaction* on \( v \).
**Inhibiting case:**

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v) \leq 0
\end{align*}
\]

**Theorem - Inhibiting case (spatial propagation)**

Assume \( K_0 = F(0, v_0) > 0 \) (i.e. \( v_0 \) large enough).

- Spatial propagation at speed \( c_0 := 2\sqrt{d_1 K_0} \).
- Final state: \( u(t = +\infty) = 0, \ v(t = +\infty) = v_\infty \) (implicit).
- Existence of bumps/wave traveling at speed \( c > c_0 \); non-existence if \( 0 < c < c_0 \) or \( K_0 < 0 \).

\[\rightsquigarrow\] Generalize the classical results on the *SI* model.

Rmk: \( c_0 \) does not depend on the *retroaction* on \( v \).
Inhibiting case:

\[
\begin{aligned}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v) \leq 0
\end{aligned}
\]

**Theorem - Inhibiting case (spatial propagation)**

Assume \( K_0 = F(0, v_0) > 0 \) (i.e. \( v_0 \) large enough).

- Spatial propagation at speed \( c_0 := 2\sqrt{d_1 K_0} \).
- Final state: \( u(t = +\infty) = 0, \ v(t = +\infty) = v_{\infty} \) (implicit).
- Existence of bumps/wave traveling at speed \( c > c_0 \); non-existence if \( 0 < c < c_0 \) or \( K_0 < 0 \).

\[\rightsquigarrow\] Generalize the classical results on the SI model

Rmk: \( c_0 \) does not depend on the retroaction on \( v \).
Inhibiting case:

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_b - v) H(u, v) \leq 0
\end{align*}
\]

Theorem - Inhibiting case (spatial propagation)

Assume \( K_0 = F(0, v_0) > 0 \) (i.e. \( v_0 \) large enough).

- Spatial propagation at speed \( c_0 := 2 \sqrt{d_1 K_0} \).
- Final state: \( u(t = +\infty) = 0, \, v(t = +\infty) = v_\infty \) (implicit).
- Existence of bumps/wave traveling at speed \( c > c_0 \); non-existence if \( 0 < c < c_0 \) or \( K_0 < 0 \).

\( \Rightarrow \) Generalize the classical results on the SI model

Rmk: \( c_0 \) does not depend on the retroaction on \( v \).
**Inhibiting case:**

\[
\begin{aligned}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v) \leq 0
\end{aligned}
\]

**Theorem - Inhibiting case (spatial propagation)**

Assume \( K_0 = F(0, v_0) > 0 \) (i.e. \( v_0 \) large enough).

- Spatial propagation at speed \( c_0 := 2\sqrt{d_1 K_0} \).
- Final state: \( u(t = +\infty) = 0, v(t = +\infty) = v_\infty \) (implicit).
- Existence of bumps/wave traveling at speed \( c > c_0 \); non-existence if \( 0 < c < c_0 \) or \( K_0 < 0 \).

\[\leftrightarrow \text{Generalize the classical results on the } SI \text{ model} \]

Rmk: \( c_0 \) does not depend on the retroaction on \( v \).
\[ \begin{cases} \partial_t u - d_1 \Delta_x u = uF(u, v), \\ \partial_t v - d_2 \Delta_x v = uG(u, v) + (v_b - v)H(u, v) \leq 0 \end{cases} \]

**Theorem - Inhibiting case (spatial propagation)**

Assume \( K_0 = F(0, v_0) > 0 \) (i.e. \( v_0 \) large enough).

- Spatial propagation at speed \( c_0 := 2\sqrt{d_1 K_0} \).
- Final state: \( u(t = +\infty) = 0, v(t = +\infty) = v_\infty \) (implicit).
- Existence of bumps/wave traveling at speed \( c > c_0 \); non-existence if \( 0 < c < c_0 \) or \( K_0 < 0 \).

\[ \sim \rightarrow \text{Generalize the classical results on the } SI \text{ model} \]

Rmk: \( c_0 \) does not depend on the retroaction on \( v \).
Inhibiting case: (example) : modified $SI(R)$

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v \left( 1 - u \right) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= -uv \leq 0.
\end{align*}
\]
Enhancing case:
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v) H(u, v) \geq 0.
\end{align*}
\]

Theorem - Enhancing case

Assume \( K_0 := F(0, v_0) > 0 \). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \bar{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \bar{c}] \).
- Explicit final states with \( u_\infty > 0 \).
- Existence of wave traveling at speed \( c > \bar{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

Rmk: \( c \) depends on the retroaction on \( v \) (differs from the inhib. case).
Enhancing case:

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v) \geq 0.
\end{align*}
\]

Theorem - Enhancing case

Assume \( K_0 := F(0, v_0) > 0 \). Set \( c_0 := 2\sqrt{d_1 K_0} \), \( \overline{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \overline{c}] \).
- Explicit final states with \( u_\infty > 0 \).
- Existence of wave traveling at speed \( c > \overline{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

Rmk: \( c \) depends on the retroaction on \( v \) (differs from the inhib. case).
**Enhancing case:**

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v) \geq 0.
\end{align*}
\]

**Theorem - Enhancing case**

Assume \( K_0 := F(0, v_0) > 0 \). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \bar{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \bar{c}] \).
- Explicit final states with \( u_\infty > 0 \).
- Existence of wave traveling at speed \( c > \bar{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

Rmk: \( c \) depends on the retroaction on \( v \) (differs from the inhib. case).
Enhancing case:

\[ \begin{cases} 
\partial_t u - d_1 \Delta_x u = uF(u, v), \\
\partial_t v - d_2 \Delta_x v = uG(u, v) + (v_b - v)H(u, v) \geq 0.
\end{cases} \]

**Theorem - Enhancing case**

Assume \( K_0 := F(0, v_0) > 0 \). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \bar{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \bar{c}] \).
- Explicit final states with \( u_\infty > 0 \).
- Existence of wave traveling at speed \( c > \bar{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

Rmk: \( c \) depends on the retroaction on \( v \) (differs from the inhib. case).
Enhancing case:

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v) \geq 0.
\end{align*}
\]

Theorem - Enhancing case

Assume \( K_0 := F(0, v_0) > 0 \). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \bar{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \bar{c}] \).
- Explicit final states with \( u_\infty > 0 \).
- Existence of wave traveling at speed \( c > \bar{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

Rmk: \( c \) depends on the retroaction on \( v \) (differs from the inhib. case).
**Enhancing case:**

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v) \geq 0.
\end{align*}
\]

**Theorem - Enhancing case**

Assume \( K_0 := F(0, v_0) > 0 \). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \bar{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \bar{c}] \).
- Explicit final states with \( u_\infty > 0 \).
- Existence of wave traveling at speed \( c > \bar{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

Rmk: \( c \) depends on the **retroaction** on \( v \) (differs from the inhib. case).
Enhancing case (example) : host/parasite cooperative system

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v (1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= uv(1 - v) \geq 0.
\end{align*}
\]

Final state : \( u_\infty = \frac{2}{3}, \ v_\infty = 1. \)

\[\Rightarrow\] reaction and diffusion of susceptibility \( v \) speeds up the propagation. Differs from the inhib. case (and so from SIR).
Enhancing case (example) : host/parasite cooperative system

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( \nu (1 - u) - \frac{1}{3} \right), \\
\partial_t \nu - d_2 \Delta_x \nu &= u \nu (1 - \nu) \geq 0.
\end{align*}
\]

Final state : \( u_\infty = 2/3, \ \nu_\infty = 1. \)

反應和擴散的Susceptibility ν 加快了傳播的速度。與抑制情況不同（和SIR差別）。
Enhancing case (example) : host/parasite cooperative system

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v(1-u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= uv(1-v) \geq 0.
\end{align*}
\]

Final state : \( u_\infty = 2/3, \ v_\infty = 1. \)
Enhancing case (example) : host/parasite cooperative system

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v \left( 1 - u \right) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= kuv \left( 1 - v \right) \geq 0, \quad k > 0.
\end{align*}
\]

Final state : \( u_\infty = \frac{2}{3}, \ v_\infty = 1. \)

→ The speed of propagation depends on the retroaction on \( v \):

\( d_2 \) reaction and diffusion of susceptibility \( v \) speeds up the propagation. Differs from the inhib. case (and so from SIR).
**Enhancing case (example) : host/parasite cooperative system**

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v (1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= u v (1 - v) \geq 0.
\end{align*}
\]

Final state : \( u_\infty = 2/3, \ v_\infty = 1. \)

\[\rightarrow\] The speed of propagation depends on the retroaction on \( v \):

\( d_2 \) \( \Rightarrow \) reaction and diffusion of susceptibility \( v \) speeds up the propagation. Differs from the inhib. case (and so from SIR).
## Recap. Comparison inhibiting/enhancing

<table>
<thead>
<tr>
<th>Example</th>
<th>Inhibiting</th>
<th>Enhancing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Example</td>
<td>SI; prey/predators; flame propagation</td>
<td>cooperative systems (host parasite, etc)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Shape of T.W. for $u$</th>
<th><img src="image1.png" alt="Graph" /></th>
<th><img src="image2.png" alt="Graph" /></th>
</tr>
</thead>
<tbody>
<tr>
<td>Final state</td>
<td>calm</td>
<td>excited</td>
</tr>
<tr>
<td>$K_0 &gt; 0$</td>
<td>$\text{ephemeral activity}$</td>
<td>$\text{lasting activity}$</td>
</tr>
<tr>
<td>Asymptotic speed of propagation $c$</td>
<td>$= c_0$ indep. on the retroaction on $v$</td>
<td>$\in [c_0, \bar{c}]$ depends on the retroaction on $v$</td>
</tr>
</tbody>
</table>
**General case:**

\[
\begin{align*}
\partial_t u - d_1 \Delta x u &= uF(u, v), \\
\partial_t v - d_2 \Delta x v &= uG(u, v) + (v_b - v)H(u, v)
\end{align*}
\]

**Theorem - General case**

Assume $K_0 > 0$ (i.e. $v_0$ large). Set $c_0 := 2 \sqrt{d_1 K_0}$; $\bar{c} := 2 \sqrt{d_1 \sup F}$.

- Spatial propagation at speed $c \in [c_0, \bar{c}]$.
- Existence of wave traveling at speed $c > \bar{c}$ if $d_2 \ll 1$; non-existence if $0 < c < c_0$.

Generalize *propagation results* to a very large class of models.

This class of systems can generate various behaviors.
General case:

\[
\begin{cases}
    \partial_t u - d_1 \Delta_x u = uF(u, v), \\
    \partial_t v - d_2 \Delta_x v = uG(u, v) + (v_b - v)H(u, v)
\end{cases}
\]

**Theorem - General case**

Assume \( K_0 > 0 \) (i.e. \( v_0 \) large). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \bar{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \bar{c}] \).
- Existence of wave traveling at speed \( c > \bar{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

~ Generalize propagation results to a very large class of models

\( \rightarrow \) This class of systems can generate various behaviors.
General case:

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u,v), \\
\partial_t v - d_2 \Delta_x v &= uG(u,v) + (v_b - v)H(u,v)
\end{align*}
\]

Theorem - General case

Assume \( K_0 > 0 \) (i.e. \( v_0 \) large). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \overline{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \overline{c}] \).
- Existence of wave traveling at speed \( c > \overline{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

Generalize propagation results to a very large class of models.

This class of systems can generate various behaviors.
General case:

\[
\begin{cases}
\partial_t u - d_1 \Delta_x u = uF(u, v), \\
\partial_t v - d_2 \Delta_x v = uG(u, v) + (v_b - v)H(u, v)
\end{cases}
\]

**Theorem - General case**

Assume \( K_0 > 0 \) (i.e. \( v_0 \) large). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \bar{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \bar{c}] \).
- Existence of wave traveling at speed \( c > \bar{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

\[\leadsto\] Generalize propagation results to a very large class of models

\[\rightarrow\] This class of systems can generate various behaviors.
General case:

\[
\begin{cases}
\partial_t u - d_1 \Delta_x u = u F(u, v), \\
\partial_t v - d_2 \Delta_x v = u G(u, v) + (v_b - v) H(u, v)
\end{cases}
\]

**Theorem - General case**

Assume \( K_0 > 0 \) (i.e. \( v_0 \) large). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \overline{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \overline{c}] \).
- Existence of wave traveling at speed \( c > \overline{c} \) if \( d_2 \ll 1 \); non-existence if \( 0 < c < c_0 \).

\[\rightsquigarrow\] Generalize propagation results to a very large class of models.

\[\rightarrow\] This class of systems can generate various behaviors.
General case:

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v)
\end{align*}
\]

Theorem - General case

Assume \( K_0 > 0 \) (i.e. \( v_0 \) large). Set \( c_0 := 2\sqrt{d_1 K_0} \); \( \bar{c} := 2\sqrt{d_1 \sup F} \).

- Spatial propagation at speed \( c \in [c_0, \bar{c}] \).
- Existence of wave traveling at speed \( c > \bar{c} \) if \( d_2 \ll 1 \) ; non-existence if \( 0 < c < c_0 \).

\( \rightsquigarrow \) Generalize propagation results to a very large class of models

\( \rightarrow \) This class of systems can generate various behaviors.
General case (ex.1): **Double threshold** on $v_0$ (initial susceptibility).

\[
\begin{aligned}
\partial_t u - d_1 \Delta_x u &= u \left( v(1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= uv(1 - v)(v - \frac{1}{2})
\end{aligned}
\]

- Low initial susceptibility ($v_0 < 1/3$): calm
- Medium init. susceptibility ($1/3 < v_0 < 1/2$): ephemeral activity
- High initial susceptibility ($v_0 > 1/2$): lasting activity
General case (ex.1): Double threshold on $v_0$ (initial susceptibility).

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v(1-u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= uv(1-v)(v - \frac{1}{2})
\end{align*}
\]

- Low initial susceptibility ($v_0 < \frac{1}{3}$): calm
- Medium init. susceptibility ($\frac{1}{3} < v_0 < \frac{1}{2}$): ephemeral activity
- High initial susceptibility ($v_0 > \frac{1}{2}$): lasting activity
General case (ex.1): Double threshold on $\nu_0$ (initial susceptibility).

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( \nu (1 - u) - \frac{1}{3} \right), \\
\partial_t \nu - d_2 \Delta_x \nu &= \nu \nu (1 - \nu) (\nu - \frac{1}{2})
\end{align*}
\]

- Low initial susceptibility ($\nu_0 < 1/3$): calm
- Medium init. susceptibility ($1/3 < \nu_0 < 1/2$): ephemeral activity
- High initial susceptibility ($\nu_0 > 1/2$): lasting activity
General case (ex.1): Double threshold on $v_0$ (initial susceptibility).

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v(1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= uv(1 - v)(v - \frac{1}{2})
\end{align*}
\]

- Low initial susceptibility ($v_0 < 1/3$): calm
- Medium init. susceptibility ($1/3 < v_0 < 1/2$): ephemeral activity
- High initial susceptibility ($v_0 > 1/2$): lasting activity
**General case (ex.1): Double threshold on** $v_0$ *(initial susceptibility).*

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v(1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= u v (1 - v) (v - \frac{1}{2})
\end{align*}
\]

- Low initial susceptibility ($v_0 < \frac{1}{3}$): calm
- Medium init. susceptibility ($\frac{1}{3} < v_0 < \frac{1}{2}$): ephemeral activity
- High initial susceptibility ($v_0 > \frac{1}{2}$): lasting activity
**General case (ex.2):** Threshold on $u_0$ (triggering event).

\[
\begin{align*}
\partial_t u - d_1 \Delta x u &= u \left( v(1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta x v &= uv \left( 1 - v \right) \left( v + u - \frac{1}{2} \right); \quad v_0 = 0.4.
\end{align*}
\]

- Small triggering event $u_0$: ephemeral activity
- Large triggering event $u_0$: lasting activity
General case (ex.2): Threshold on $u_0$ (triggering event).

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v(1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= uv(1 - v)(v + u - \frac{1}{2}); \quad v_0 = 0.4.
\end{align*}
\]

- Small triggering event $u_0$: ephemeral activity
- Large triggering event $u_0$: lasting activity
General case (ex.2): Threshold on $u_0$ (triggering event).

\[
\begin{cases}
\partial_t u - d_1 \Delta_x u = u \left( v(1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v = uv(1 - v)(v + u - \frac{1}{2}) \quad ; \quad v_0 = 0.4.
\end{cases}
\]

- Small triggering event $u_0$: ephemeral activity
- Large triggering event $u_0$: lasting activity
General case (ex.2): Threshold on $u_0$ (triggering event).

\[
\begin{align*}
\partial_t u - d_1 \Delta x u &= u \left( v(1 - u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta x v &= uv(1 - v)(v + u - \frac{1}{2}); \quad v_0 = 0.4.
\end{align*}
\]

- Small triggering event $u_0$: ephemeral activity
- Large triggering event $u_0$: lasting activity
**General case (ex.3): terrace.**

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( v(1-u) - \frac{1}{3} \right), \\
\partial_t v - d_2 \Delta_x v &= u(u - \frac{1}{4})v(1-v).
\end{align*}
\]

\[\rightarrow\text{ fast ephemeral movement followed by slow lasting upheaval}\]
General case (ex.4): oscillations.

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u \left( \nu (1 - u) - \frac{1}{3} \right), \\
\partial_t \nu - d_2 \Delta_x \nu &= u \nu (1 - \nu)(\nu - 10u)
\end{align*}
\]
Possible extensions: spatial heterogeneity
**Spatial heterogeneity 1:** non-constant initial susceptibility. Suppose that $v_0$ is periodic (favorable/unfavorable zones) → city/suburbs, poor/rich neighborhood, etc.

- Large favorable zone: propagation
- Small favorable zone: blockage
Spatial heterogeneity 1: non-constant initial susceptibility. Suppose that $v_0$ is periodic (favorable/unfavorable zones) $\rightarrow$ city/suburbs, poor/rich neighborhood, etc.

- Large favorable zone: propagation
- Small favorable zone: blockage
Spatial heterogeneity 1: non-constant initial susceptibility. Suppose that $v_0$ is periodic (favorable/unfavorable zones) → city/suburbs, poor/rich neighborhood, etc.

- Large favorable zone: propagation
- Small favorable zone: blockage
Spatial heterogeneity 2: gap problem.

Suppose that $u$ has no endogeneous growth in some area.

- Small gap: propagation
- Large gap: blockage
Spatial heterogeneity 2: gap problem.
Suppose that $u$ has no endogeneous growth in some area.

- Small gap: propagation
- Large gap: blockage
Spatial heterogeneity 2: gap problem.
Suppose that $u$ has no endogeneous growth in some area.

- Small gap: propagation
- Large gap: blockage
Spatial heterogeneity 3: include geometry.

For a domain $\Omega \subset \mathbb{R}^n$,

$$
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), & x \in \Omega \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_b - v) H(u, v) & x \in \Omega
\end{align*}
$$

with a no-flux condition

$$
\partial_\nu u = \partial_\nu v = 0, \quad \partial \Omega,
$$

where $\partial_\nu$ is the outer normal derivative.

Spatial obstructions (river, highways, borders, street, fences, etc.)

Spatial heterogeneity 4: line of fast diffusion [Berestycki, Roquejoffre, Rossi]

Other extensions:

- More compartments:
  - Susceptible $\rightarrow$ Exposed $\rightarrow$ Infected $\rightarrow$ Recovered
  $\Rightarrow$ multidimensional system on $(u, v_1, \ldots, v_m)$.
- Individual variabilities (age, viral load, phenotype, etc.)
  $\Rightarrow$ Extra variable $y \in \mathbb{R}^p$.
- Time-delay (incubation period), etc...
Spatial heterogeneity 3: include geometry.

For a domain $\Omega \subset \mathbb{R}^n$,

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v)
\end{align*}
\]

for $x \in \Omega$,

with a no-flux condition

\[
\partial_\nu u = \partial_\nu v = 0, \quad \partial \Omega,
\]

where $\partial_\nu$ is the outer normal derivative.

Spatial obstructions (river, highways, borders, street, fences, etc.)

Spatial heterogeneity 4: line of fast diffusion [Berestycki, Roquejoffre, Rossi]

Other extensions:

- More compartments:
  - Susceptible $\rightarrow$ Exposed $\rightarrow$ Infected $\rightarrow$ Recovered
  - Multidimensional system on $(u, v_1, \ldots, v_m)$.

- Individual variabilities (age, viral load, phenotype, etc.)
  - Extra variable $y \in \mathbb{R}^p$.

- Time-delay (incubation period), etc...
Spatial heterogeneity 3: include geometry.

For a domain $\Omega \subset \mathbb{R}^n$,

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= u F(u, v), \\
\partial_t v - d_2 \Delta_x v &= u G(u, v) + (v_b - v) H(u, v)
\end{align*}
\]

$x \in \Omega$

with a no-flux condition

\[
\partial_{\nu} u = \partial_{\nu} v = 0, \quad \partial \Omega,
\]

where $\partial_{\nu}$ is the outer normal derivative.

$\rightarrow$ spatial obstructions (river, highways, borders, street, fences, etc.)

Spatial heterogeneity 4: line of fast diffusion [Berestycki, Roquejoffre, Rossi]

Other extensions:

- More compartments:

\[
\text{Susceptible} \rightarrow \text{Exposed} \rightarrow \text{Infected} \rightarrow \text{Recovered}
\]

$\rightsquigarrow$ multidimensional system on $(u, v_1, \ldots, v_m)$.

- Individual variabilities (age, viral load, phenotype, etc.)

$\Rightarrow$ Extra variable $y \in \mathbb{R}^p$.

- Time-delay (incubation period), etc...
Spatial heterogeneity 3: include geometry.

For a domain $\Omega \subset \mathbb{R}^n$,

\[
\begin{cases}
\partial_t u - d_1 \Delta_x u = uF(u, v), & x \in \Omega \\
\partial_t v - d_2 \Delta_x v = uG(u, v) + (v_b - v)H(u, v) & x \in \Omega
\end{cases}
\]

with a no-flux condition

\[\partial_{\nu} u = \partial_{\nu} v = 0, \quad \partial \Omega,\]

where $\partial_{\nu}$ is the outer normal derivative.

Spatial obstructions (river, highways, borders, street, fences, etc.)

Spatial heterogeneity 4: line of fast diffusion [Berestycki, Roquejoffre, Rossi]

Other extensions:

- More compartments:
  
  \[
  \begin{array}{cccccc}
  \text{Susceptible} & \rightarrow & \text{Exposed} & \rightarrow & \text{Infected} & \rightarrow & \text{Recovered} \\
  \end{array}
  \]

  \[\rightsquigarrow\text{multidimensional system on } (u, v_1, \ldots, v_m).\]

- Individual variabilities (age, viral load, phenotype, etc.)
  
  \[\Rightarrow\text{Extra variable } y \in \mathbb{R}^p.\]

- Time-delay (incubation period), etc...
Spatial heterogeneity 3: include geometry.
For a domain $\Omega \subset \mathbb{R}^n$,
\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), & x \in \Omega \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v) & x \in \Omega
\end{align*}
\]
with a no-flux condition
\[
\partial_\nu u = \partial_\nu v = 0, \quad \partial \Omega,
\]
where $\partial_\nu$ is the outer normal derivative.

Spatial obstructions (river, highways, borders, street, fences, etc.)

Spatial heterogeneity 4: line of fast diffusion [Berestycki, Roquejoffre, Rossi]

Other extensions:

- More compartments:
  
  \[
  \text{Susceptible} \rightarrow \text{Exposed} \rightarrow \text{Infected} \rightarrow \text{Recovered}
  \]

  \(\rightarrow\) multidimensional system on \((u, v_1, \ldots, v_m)\).

- Individual variabilities (age, viral load, phenotype, etc.)
  \(\Rightarrow\) Extra variable \(y \in \mathbb{R}^p\).

- Time-delay (incubation period), etc...
Spatial heterogeneity 3: include geometry.

For a domain \( \Omega \subset \mathbb{R}^n \),

\[
\begin{align*}
\partial_t u - d_1 \Delta_x u &= uF(u, v), \\ x &\in \Omega \\
\partial_t v - d_2 \Delta_x v &= uG(u, v) + (v_b - v)H(u, v), \\ x &\in \Omega 
\end{align*}
\]

with a no-flux condition

\[
\partial_\nu u = \partial_\nu v = 0, \quad \partial \Omega,
\]

where \( \partial_\nu \) is the outer normal derivative.

Spatial obstructions (river, highways, borders, street, fences, etc.)

Spatial heterogeneity 4: line of fast diffusion [Berestycki, Roquejoffre, Rossi]

Other extensions:

- More compartments:
  
  \[
  \text{Susceptible} \rightarrow \text{Exposed} \rightarrow \text{Infected} \rightarrow \text{Recovered}
  \]

  \( \rightarrow \) multidimensional system on \( (u, v_1, \ldots, v_m) \).

- Individual variabilities (age, viral load, phenotype, etc.)
  
  \( \Rightarrow \) Extra variable \( y \in \mathbb{R}^p \).

- Time-delay (incubation period), etc.
Conclusion
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include SI in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- **Threshold phenomenon** on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. SIR): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include SI in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- Threshold phenomenon on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. SIR): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the litterature.
- Include SI in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- **Threshold phenomenon** on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. SIR): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include $SI$ in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- Threshold phenomenon on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. $SIR$): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include **SI** in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- **Threshold phenomenon** on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. **SIR**): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the litterature.
- Include $SI$ in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- Threshold phenomenon on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. $SIR$): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include SIR in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- **Threshold phenomenon** on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
  - Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. SIR): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc.).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include $SI$ in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- **Threshold phenomenon** on the initial level of susceptibility
- **Asymptotic speed of propagation and traveling waves**
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. $SIR$): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include SI in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- **Threshold phenomenon** on the initial level of susceptibility
- **Asymptotic speed of propagation and traveling waves**
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. SIR): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include $SI$ in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- Threshold phenomenon on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. $SIR$): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include $SI$ in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- Threshold phenomenon on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. $SIR$): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the litterature.
- Include SI in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- Threshold phenomenon on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. SIR): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc.).
- Models with spatial heterogeneity.
Identify a general paradigm:

- General paradigm for epidemiology and propagation in excitable media (flame propagation, population dynamics, etc.) → unified view on topics studied separately in the literature.
- Include $SI$ in a large class of models → identify the core mechanisms and properties of epidemiology models.
- Epidemiology models for the propagation of collective behaviors. The example of riots.

Main mathematical results:

- Threshold phenomenon on the initial level of susceptibility
- Asymptotic speed of propagation and traveling waves
- Unified framework, general tools, robust methods

Classes of models useful in modeling:

- Inhibiting case (ex. $SIR$): ephemeral episodes of activity.
- Enhancing case: lasting episodes of activity.
- General case: various behaviors (double threshold, terrace, etc).
- Models with spatial heterogeneity.
Thank you!
Activity/Susceptibility systems:

Towards a paradigm for propagation of epidemics, collective behaviors, and propagation in excitable media

Samuel Nordmann (in collaboration with Henri Berestycki and Luca Rossi)

Tel-Aviv University. samueln@mail.tau.ac.il