Landscape epidemiology of emerging infectious diseases in natural and human-altered ecosystems

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UNC CHARLOTTE

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BACKGROUND

- Natural ecosystems are complex:
  - Non-linear dynamics
  - Feedbacks & interactions
  - Difficult to measure & almost never a single "right" scale of observation

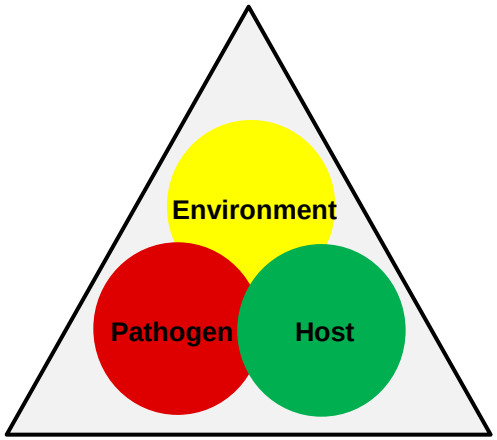
BACKGROUND

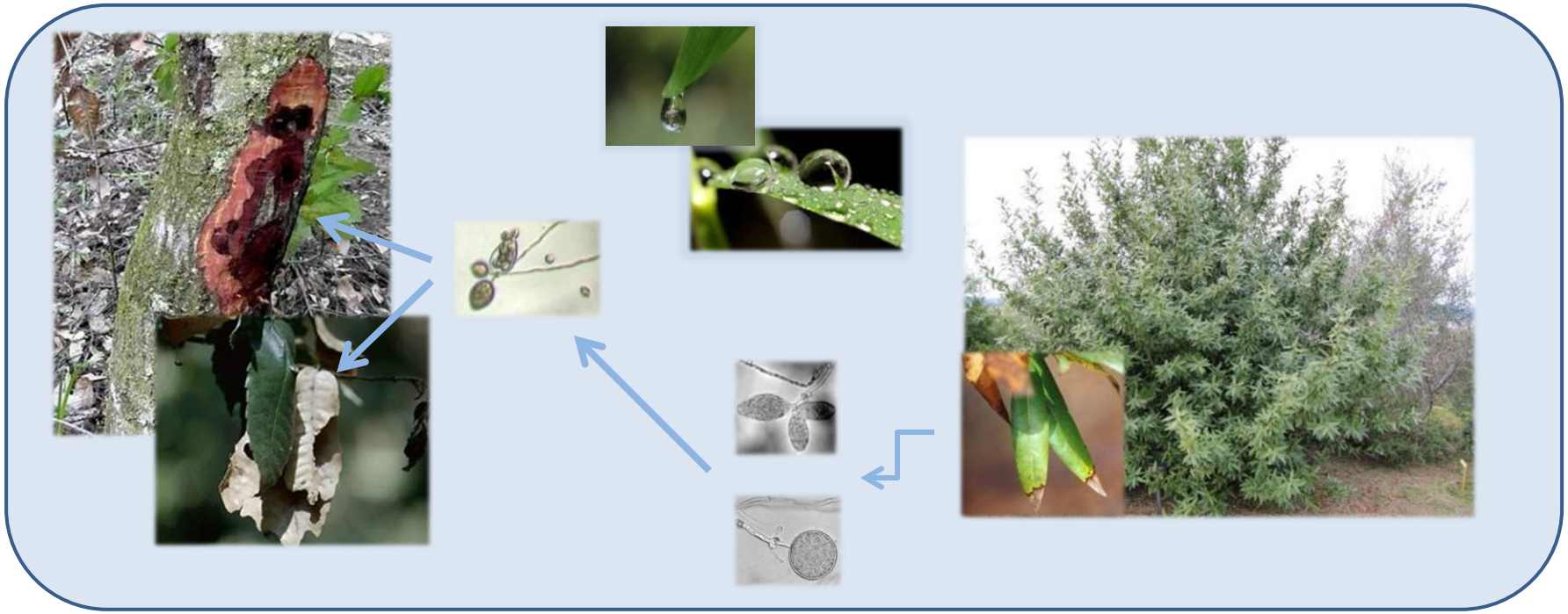
- Natural ecosystems are complex:
  - Non-linear dynamics
  - Feedbacks & interactions
  - Difficult to measure all relevant variables (including unknown effects)

- In **human-altered ecosystems** additional uncertainties further complicate our understanding

DISEASE ECOLOGY

- Pathogens interact locally with plant and animal populations

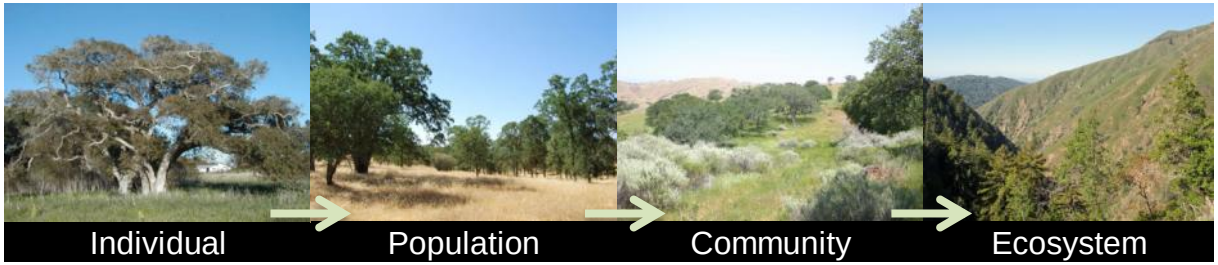




DISEASE ECOLOGY

- Local interactions of pathogens with plant and animal populations
- Limited understanding of larger-scale effects of spatio-temporal heterogeneity on pathogen dispersal

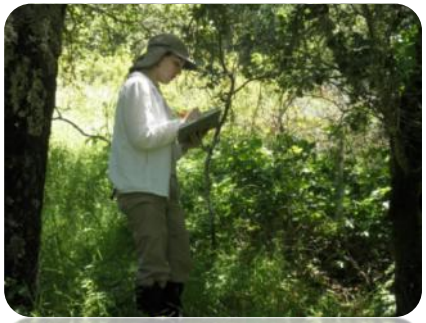
Jarrah dieback
(*Phythopthora cinnamomi*)



LANDSCAPE EPIDEMIOLOGY

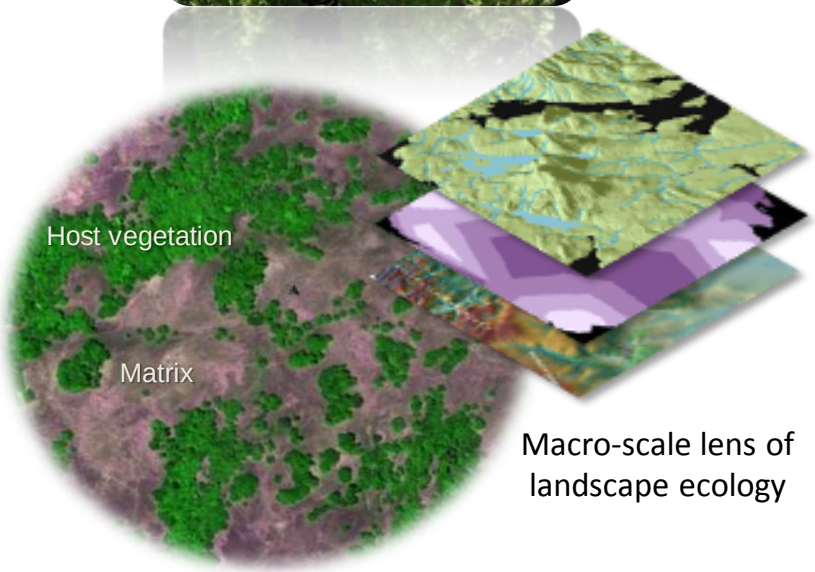
landscape heterogeneity ⇄ ecological processes

- More than simply establishing plots in the field and measuring environmental conditions.



Disease ecology

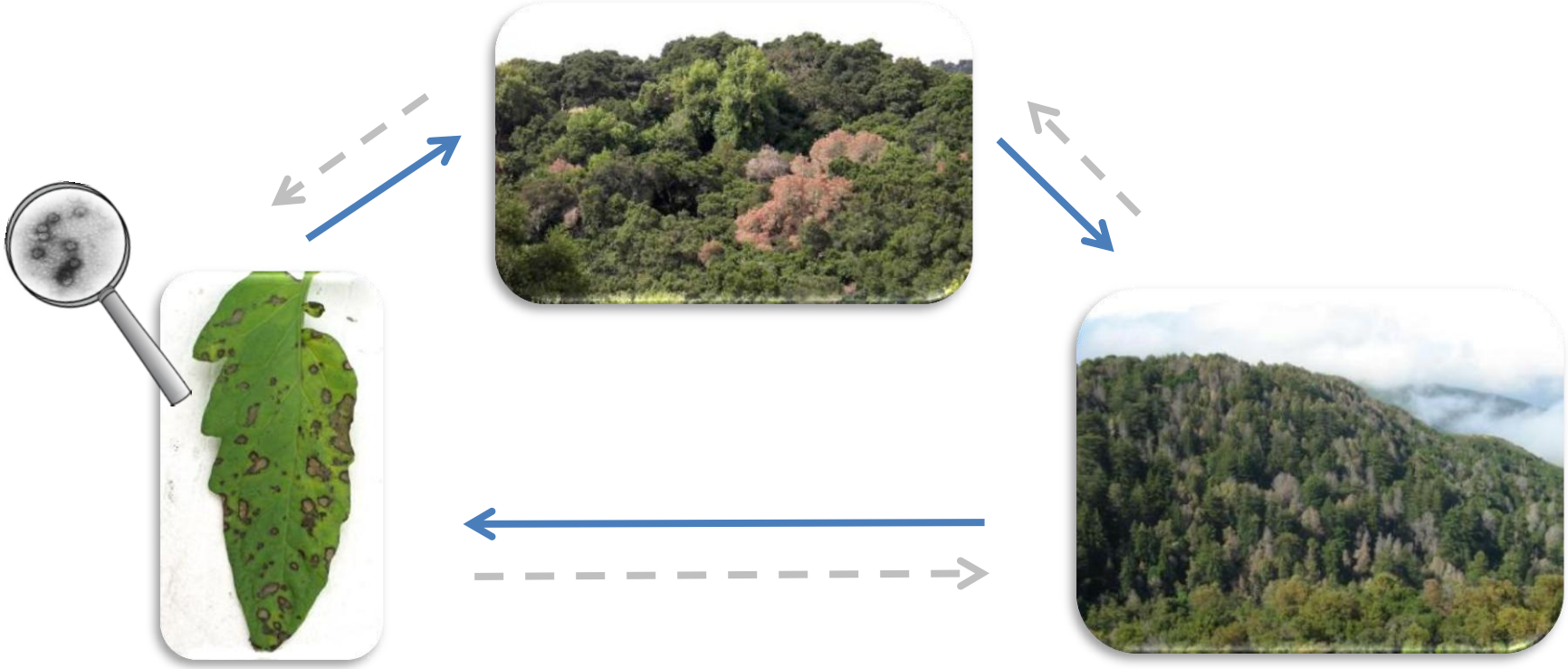
- Key components:
  - Geographical distribution of disease
  - Landscape connectivity



Macro-scale lens of landscape ecology

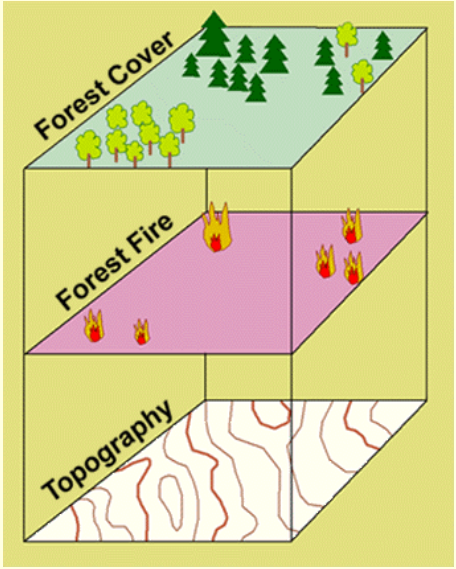
LANDSCAPE EPIDEMIOLOGY

- Spatial linking of molecular & microbial observations of disease distribution with measurements of surrounding landscape



LANDSCAPE EPIDEMIOLOGY

- GIS can assimilate large spatial datasets, but limited to static snapshots of spatial variables.



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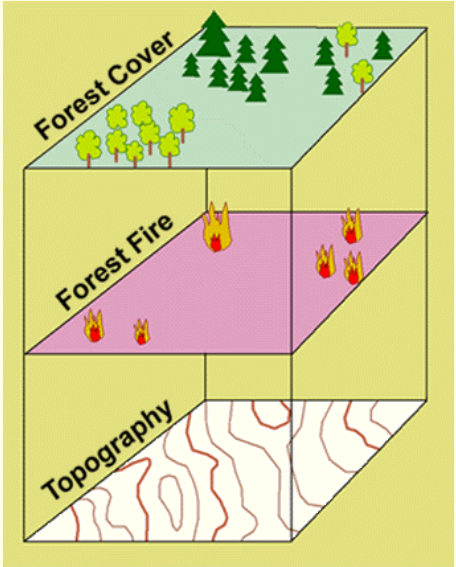
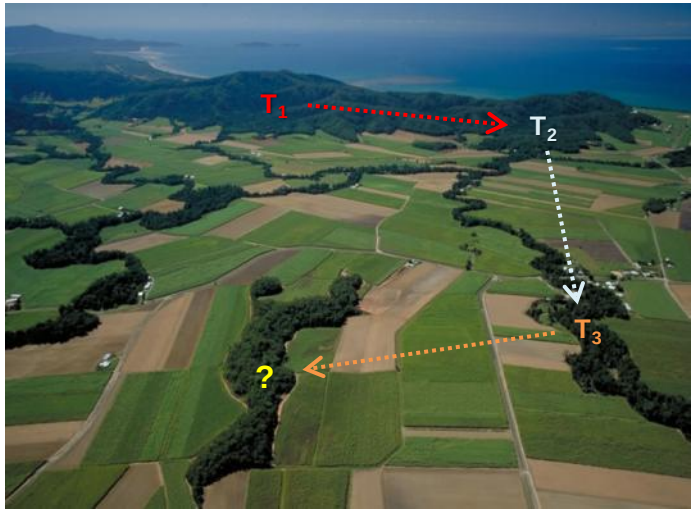


- We must embrace ways to integrate spatial and *temporal* dimensions in landscape epidemiology.
  - process-based understanding of epidemic behavior over time
  - e.g., dispersal and infection



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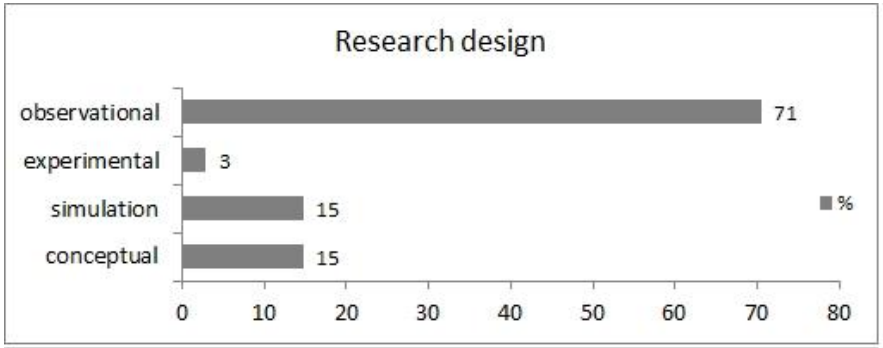

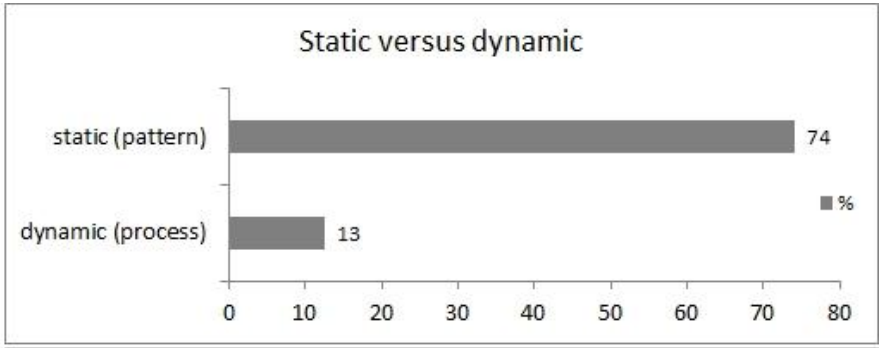

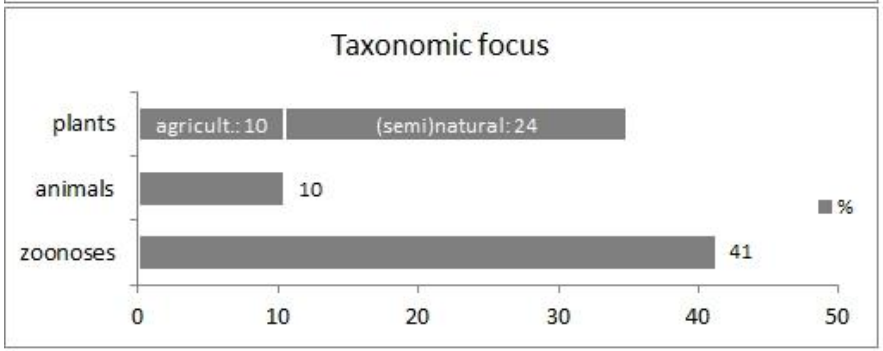

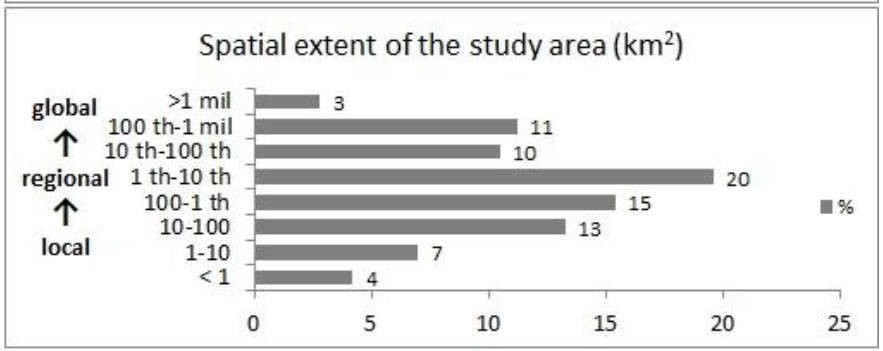

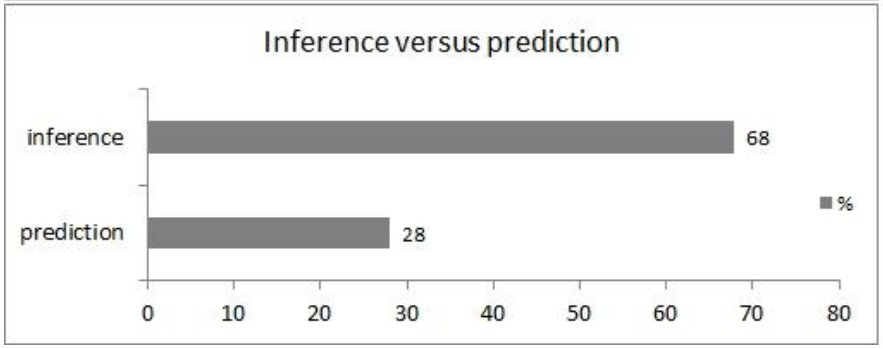

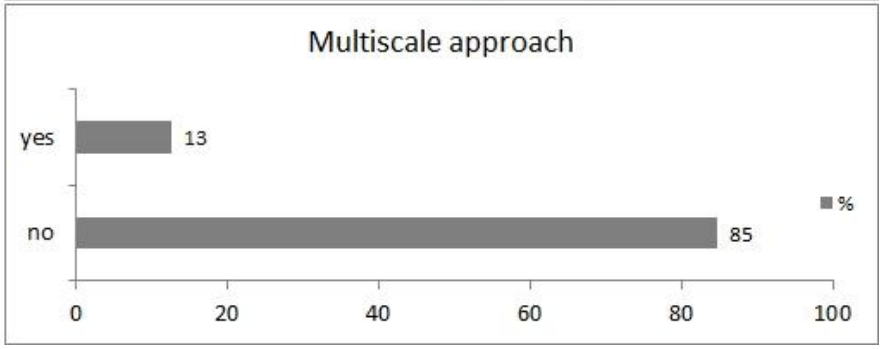

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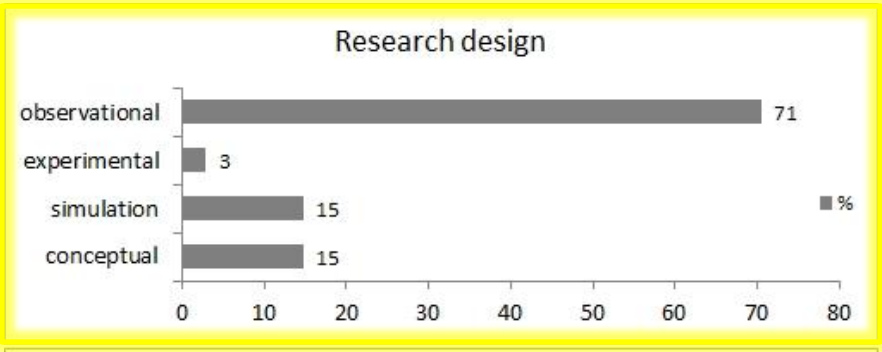

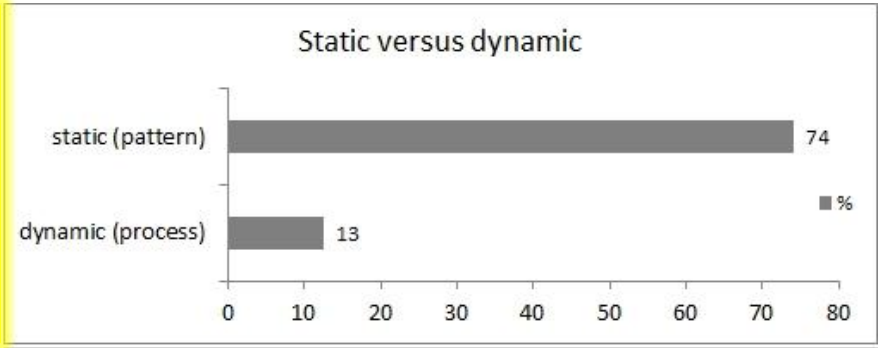

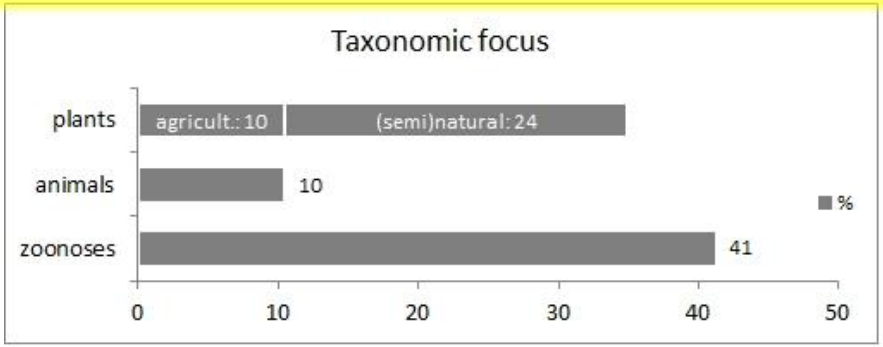

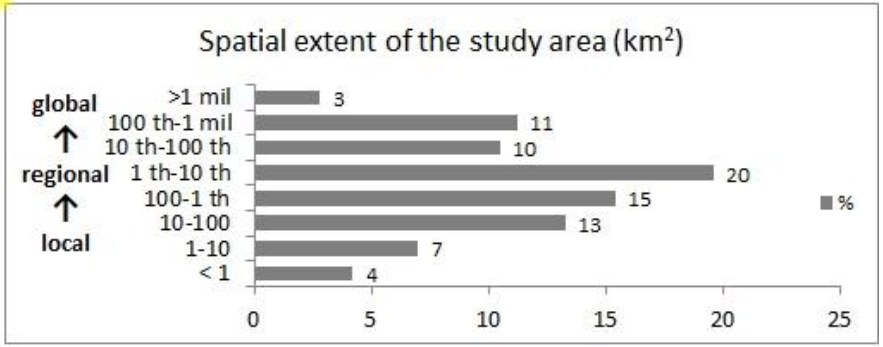

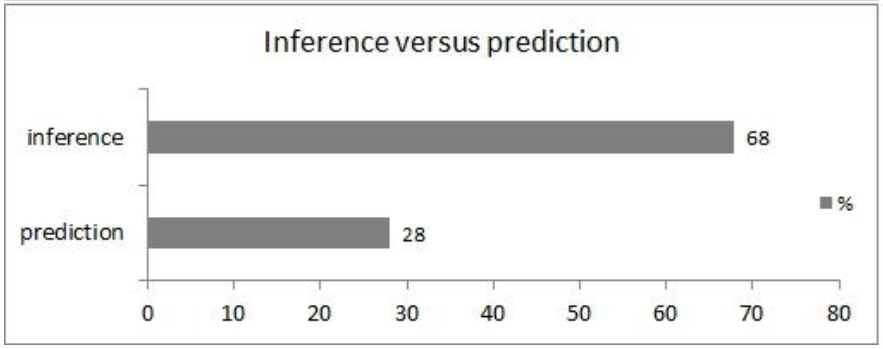

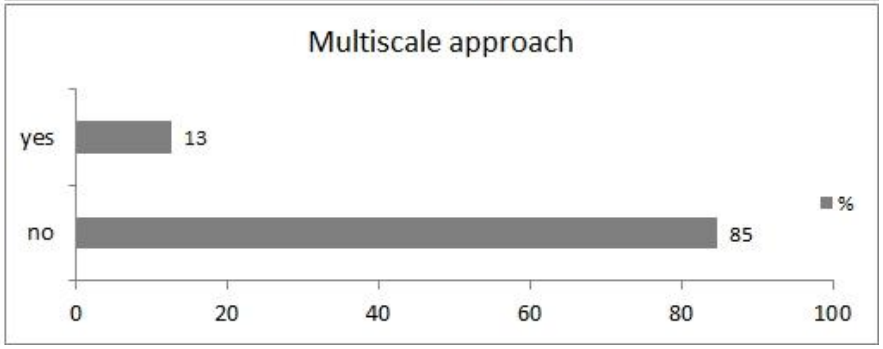

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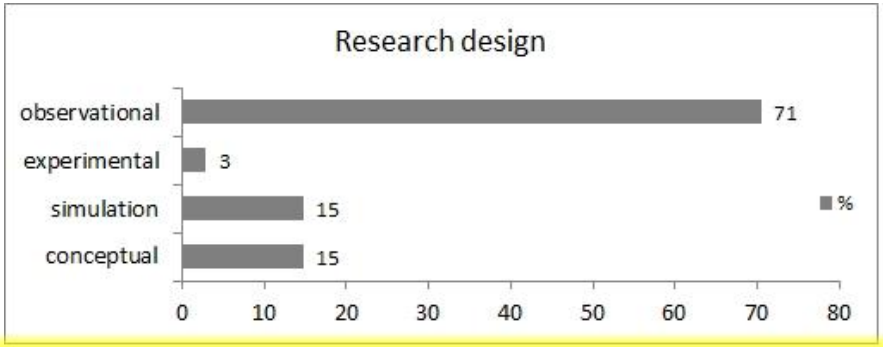

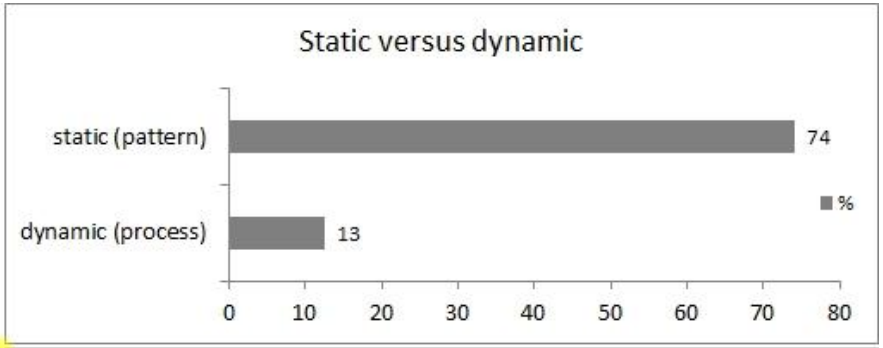

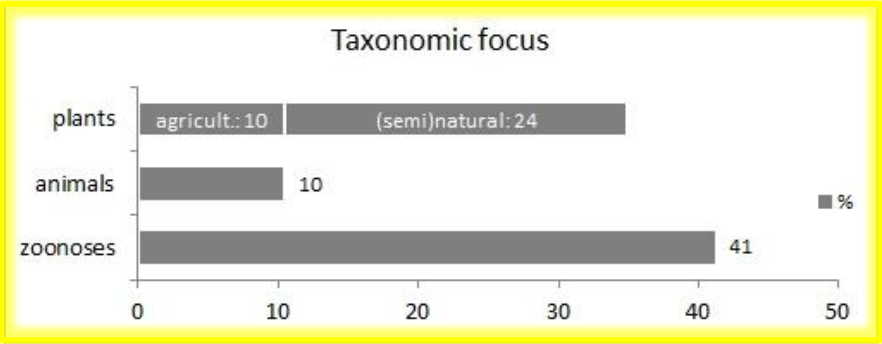

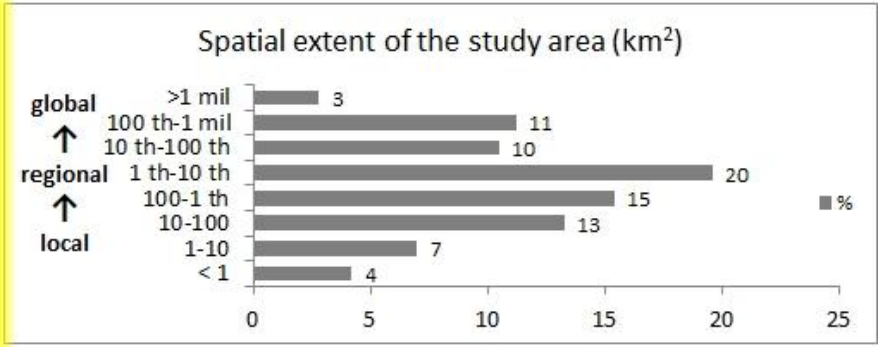

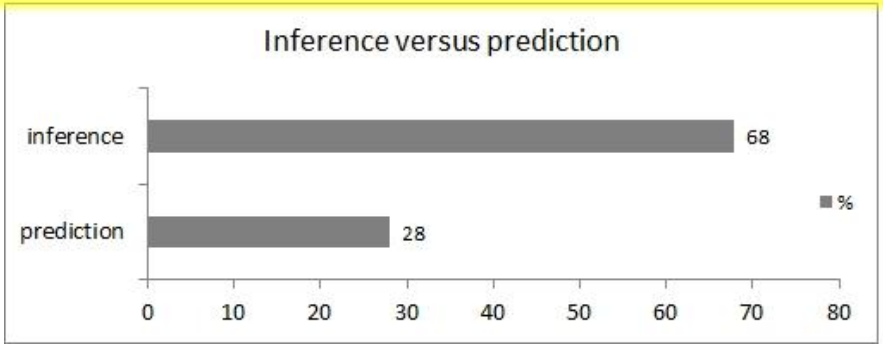

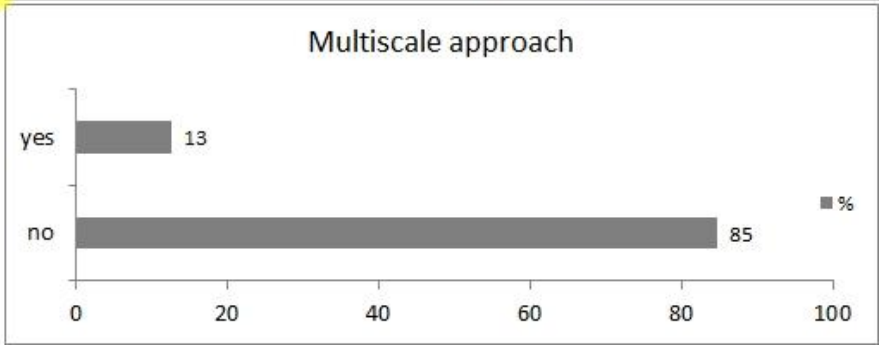

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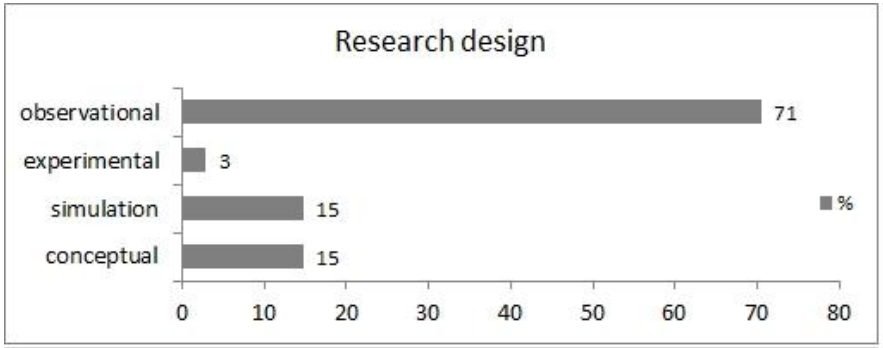

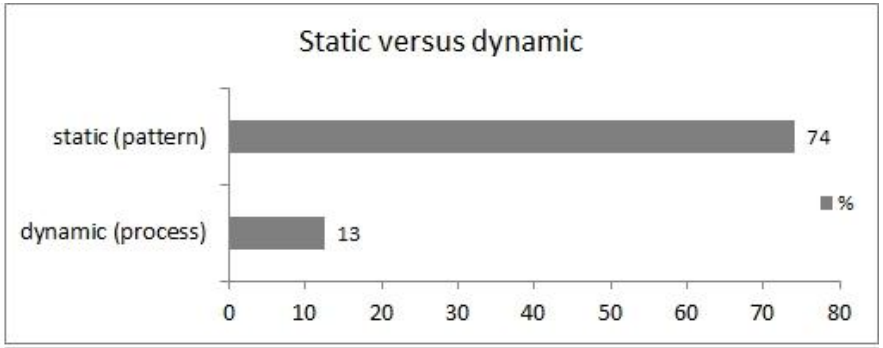

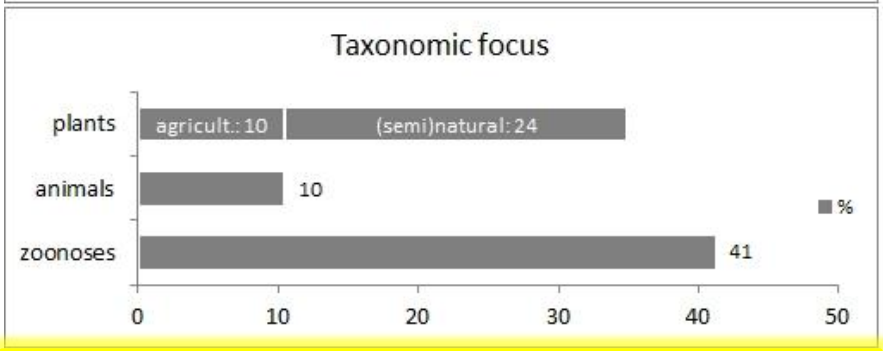

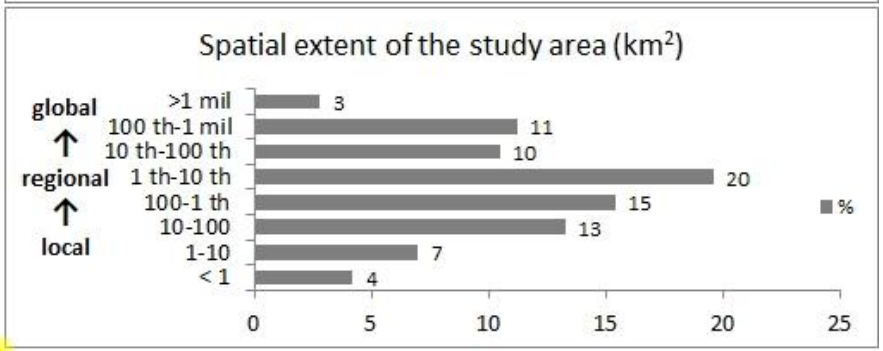

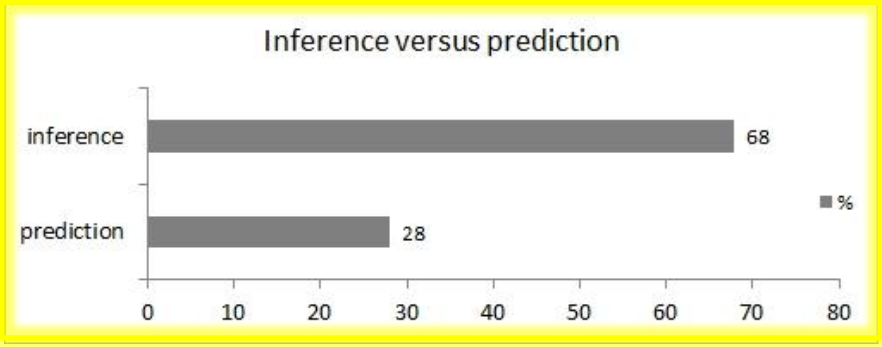

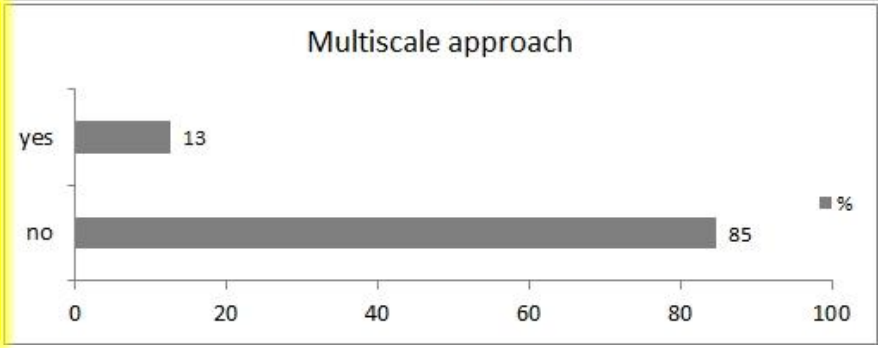

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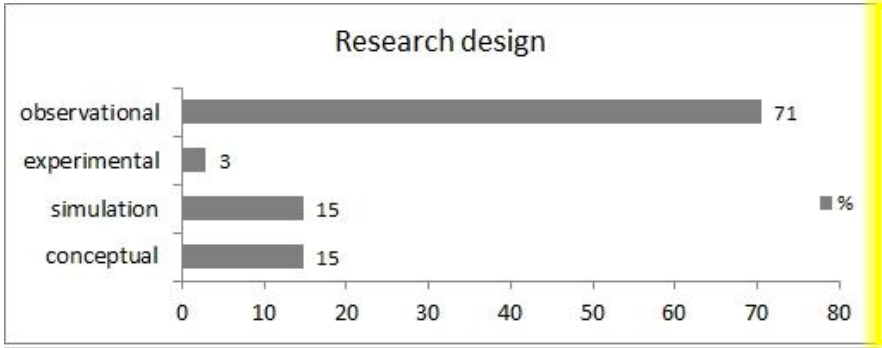

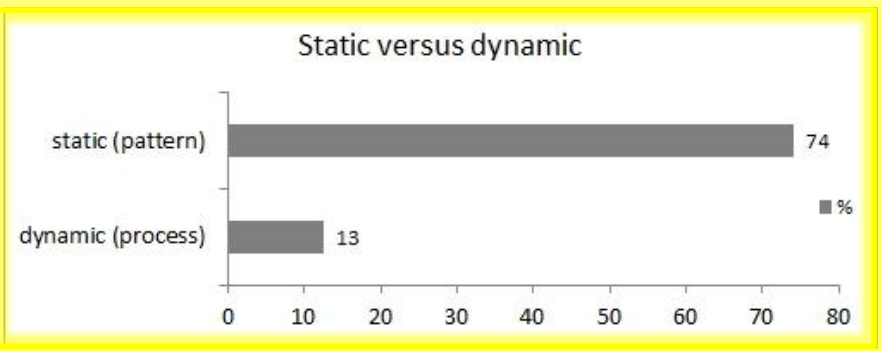

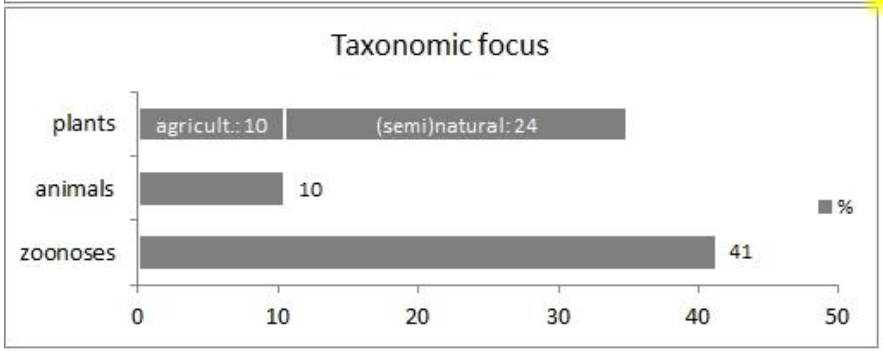

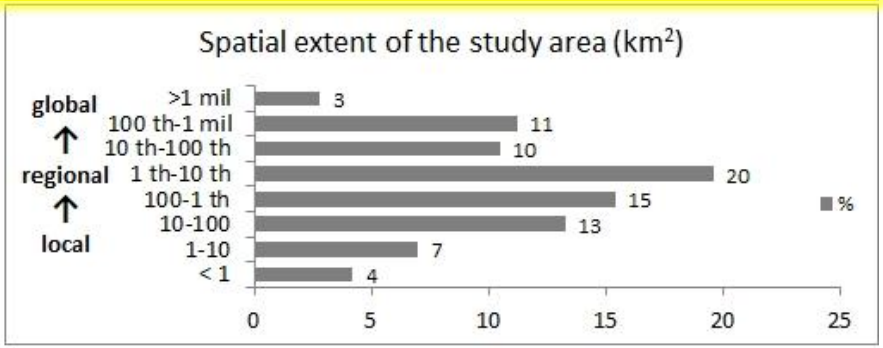

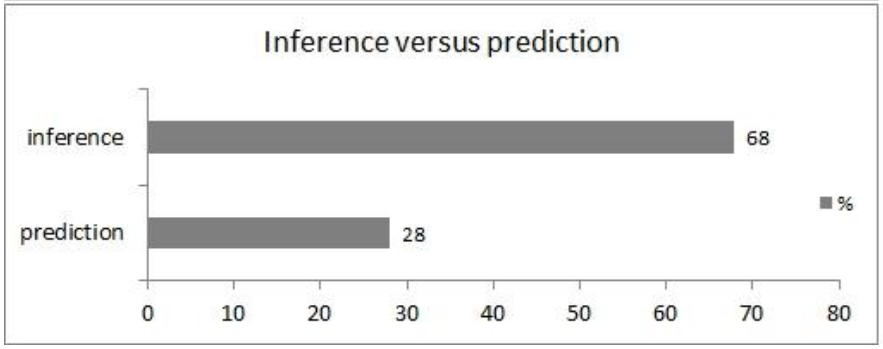

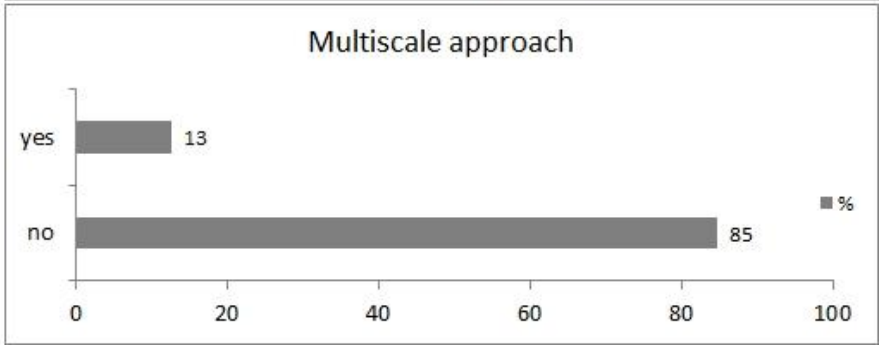

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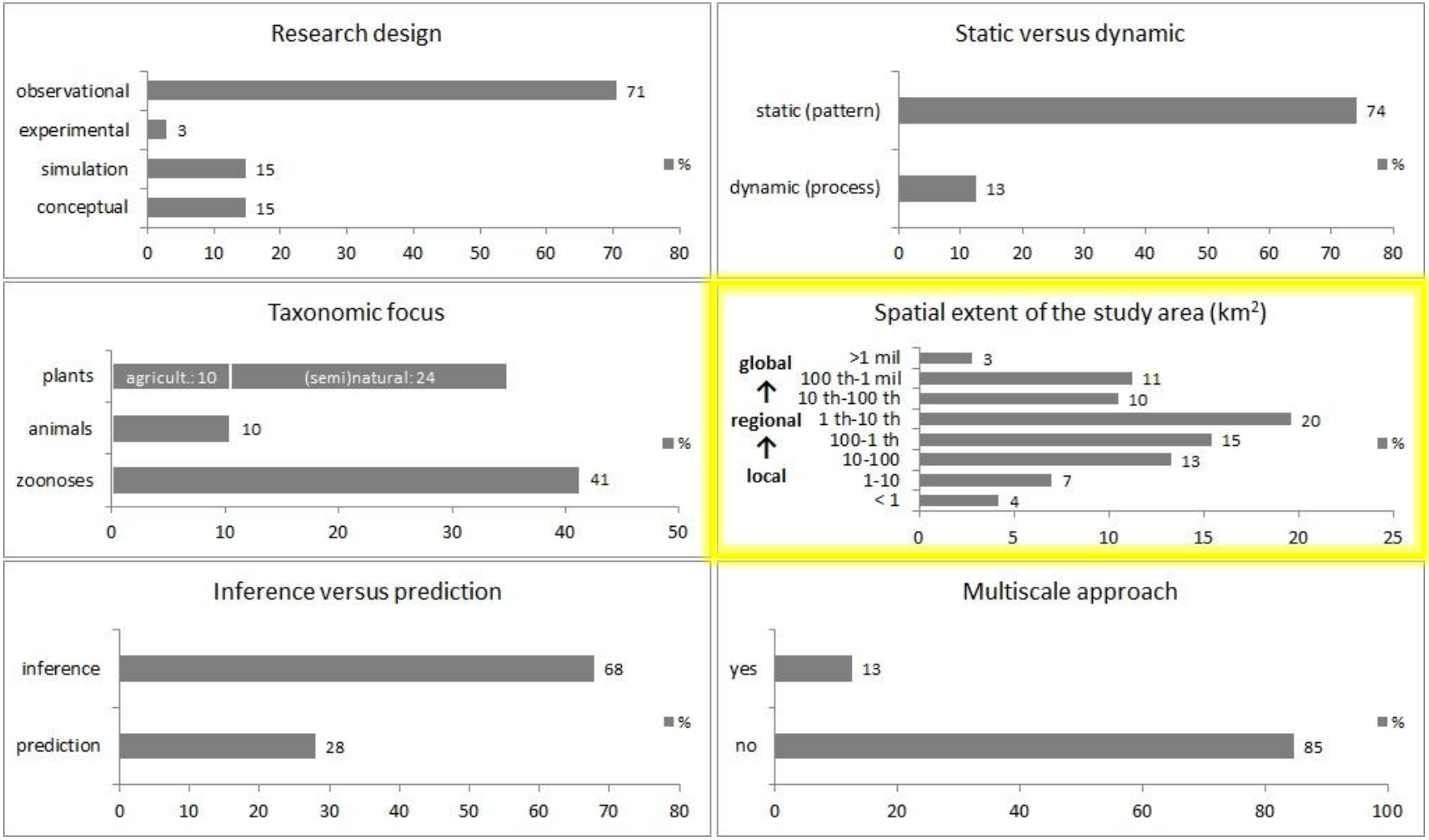

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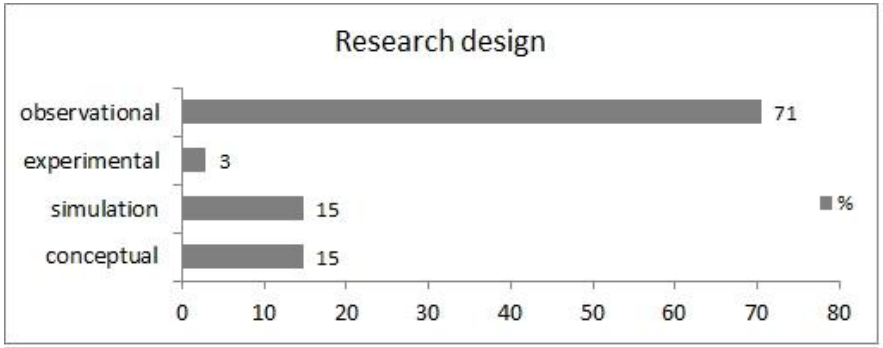

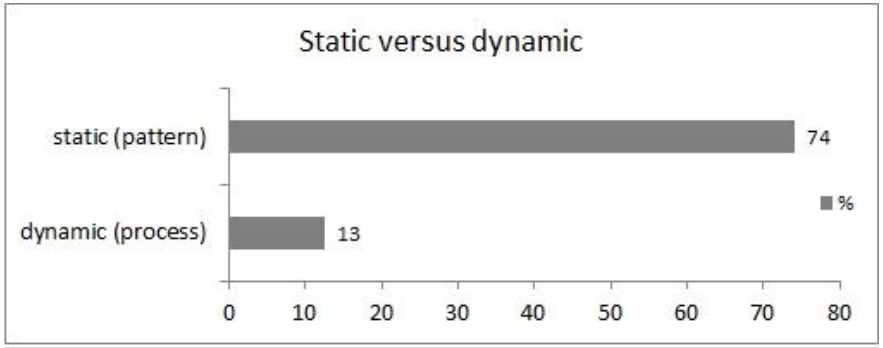

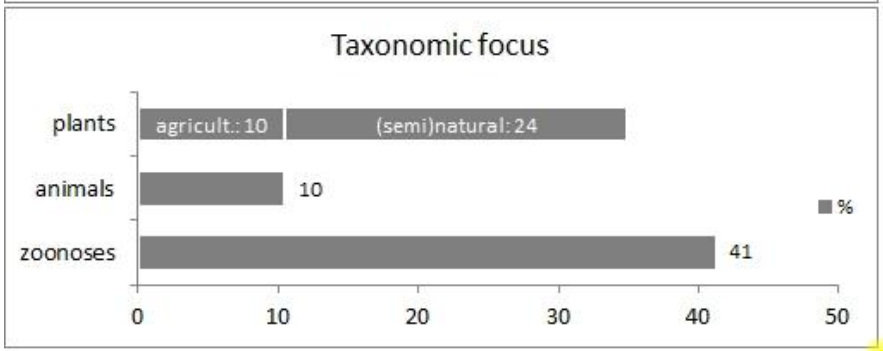

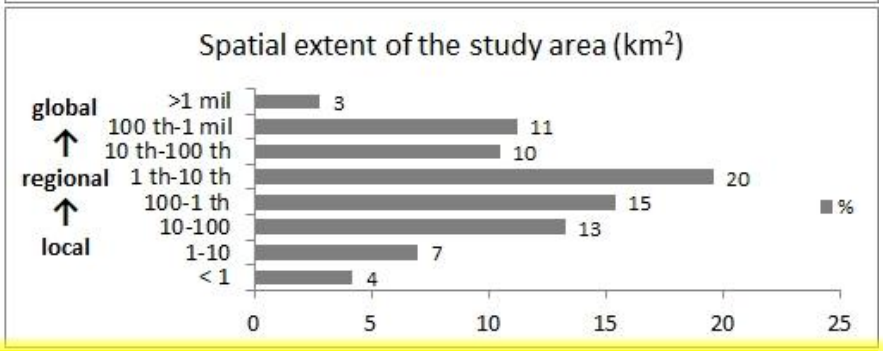

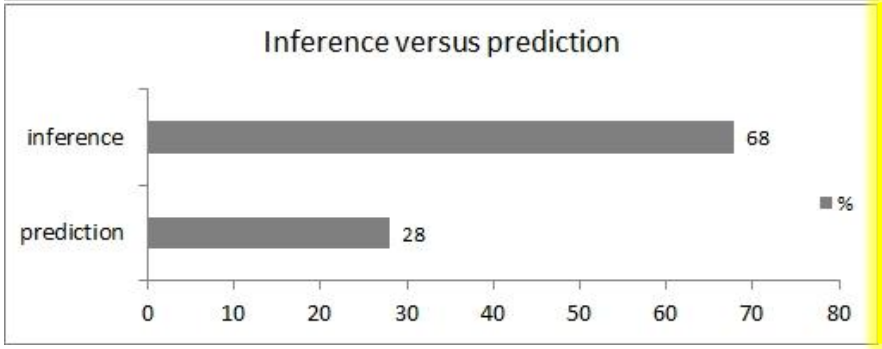

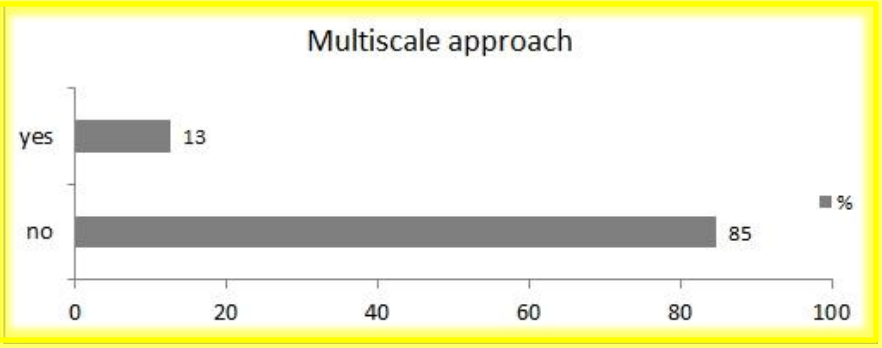

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Plant Diseases Analyzed Using LE Approach

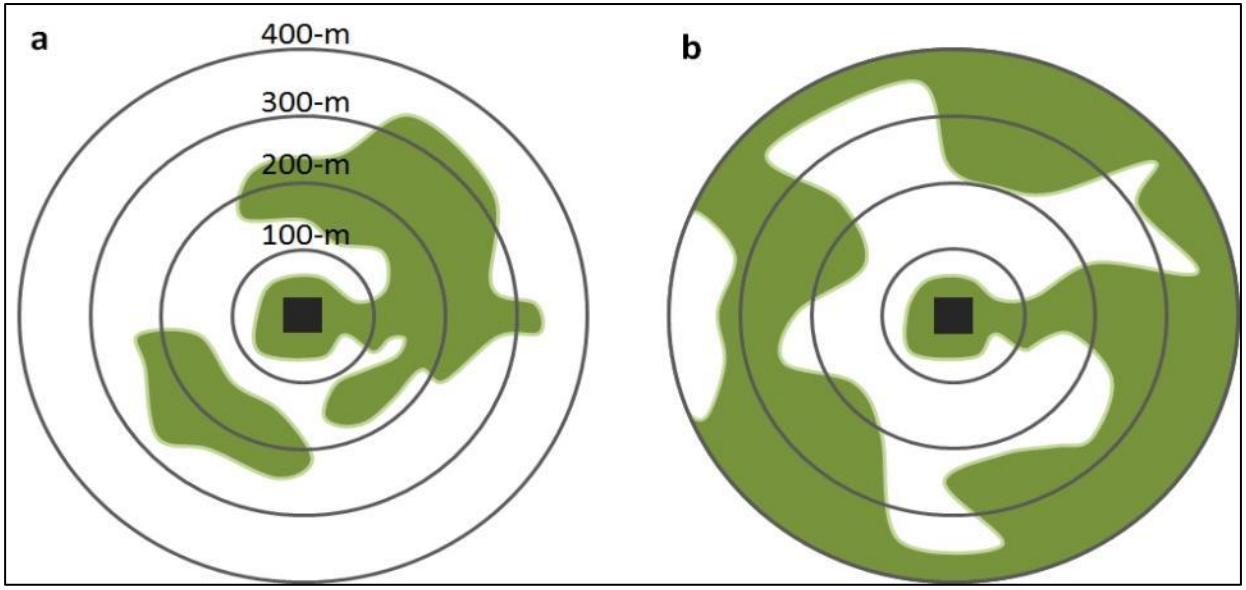
Disease (n=11)	Pathogen (taxonomic class)	EID (Yes/No)	Predominant Dispersal Mode (biotic, abiotic, human)	Ecosystem Type (natural, human-altered, agricultural)	Number of publications
cedar root disease	*Phytophthora lateralis* (Oomycetes)	Yes	abiotic (water), human	natural	2
eastern spruce dwarf mistletoe	*Arceuthobium pusillum* (macroparasite)	No	abiotic (seed dispersal)	natural	1
foot rot/head blight	*Fusarium culmorum/ F. Graminearum* (Sordariomycetes)	Yes	abiotic (rain & wind)	agricultural	1
jarrah dieback	*Phytophthora cinnamomi* (Oomycetes)	Yes	human, abiotic (water)	natural	1
mal del cipŕes	*Phytophthora austrocedrae* (Oomycetes)	Yes	abiotic (air & water)	natural	1
(no name at this time)	*Phytophthora kernoviae* (Oomycetes)	Yes	abiotic (rain & wind)	human-altered	1
oak wilt	*Ceratocystis fagacearum* (Sordariomycetes)	Yes	biotic (root grafting & insects)	human-altered	1
powdery mildew	*Podosphaera plantaginis* (Leotiomycetes)	No	abiotic (wind)	agricultural	1
southern corn leaf blight	*Cochliobolus heterostrophus* (Dothiodeomycetes)	No	abiotic (wind)	human-altered	1
sudden oak death	*Phytophthora ramorum* (Oomycetes)	Yes	abiotic (rain & wind), human	natural, human-altered	14
white pine blister rust	*Cronartium ribicola* (Pucciniomycetes)	Yes	abiotic (wind)	natural	2

Practicing Landscape Epidemiology

1) What is the spatial scale?
2) Is the model static or dynamic?
3) Is the model spatially implicit or explicit?
4) How ecologically meaningful are the variables?
5) Is the goal inference or prediction?

What is the spatial scale?

- Scale of observation drives results & conclusions
- No single 'best' scale; each species uniquely perceives its environment.



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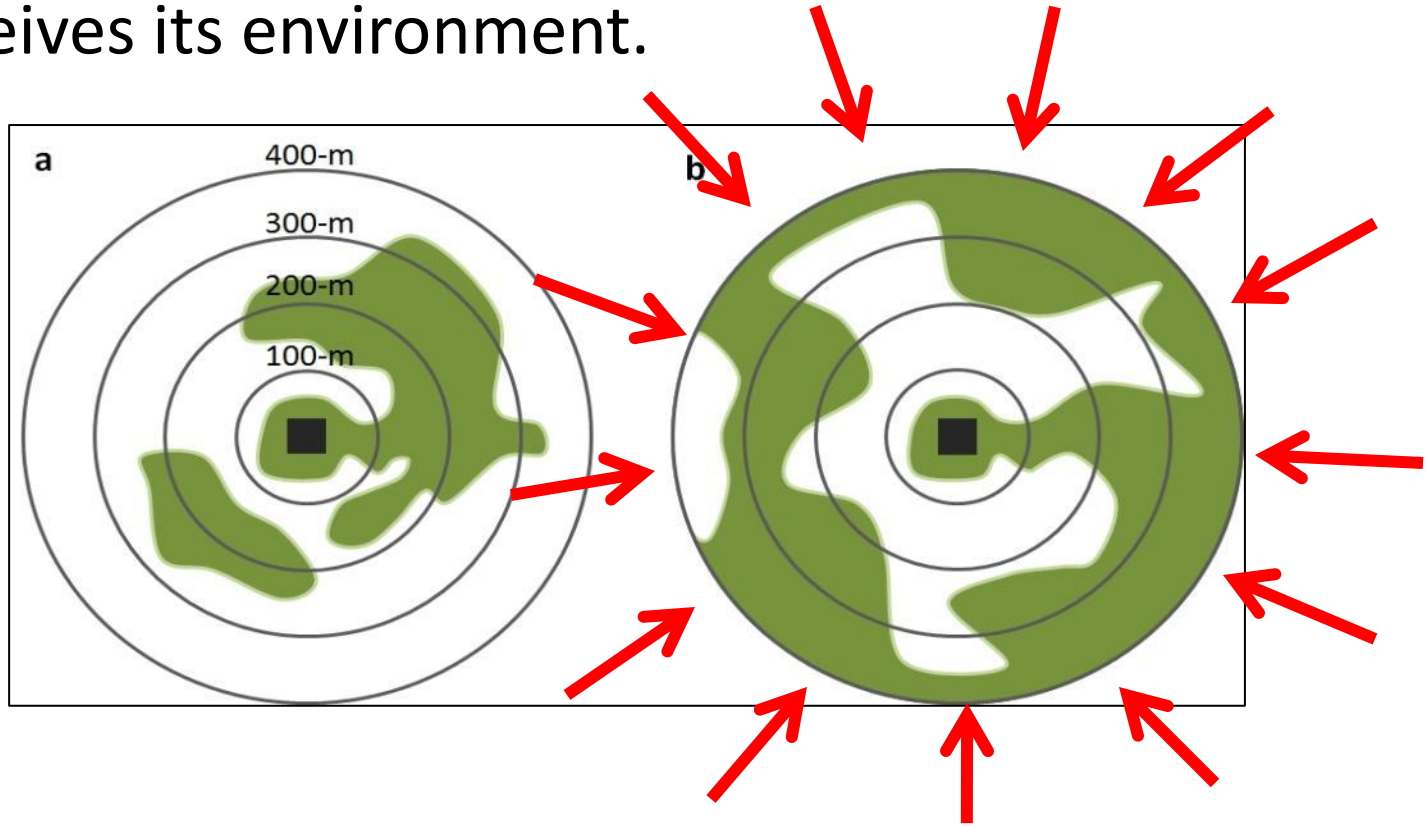
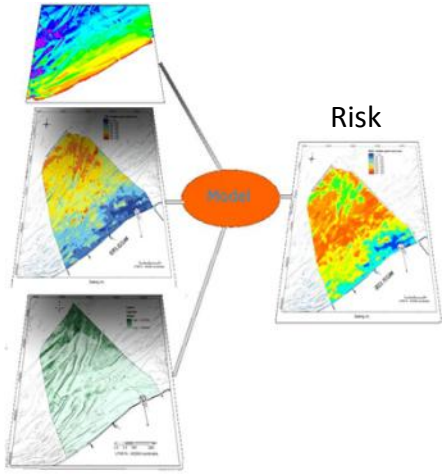


Is the model static or dynamic?

- Static approaches have yielded novel insights into how real-world heterogeneity affects disease risk.
- Often use correlative species distribution models.

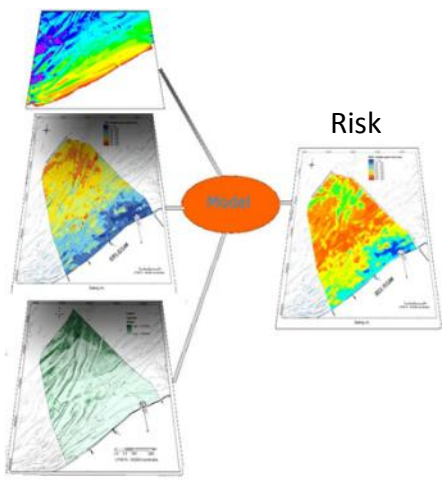
Static Models	Dynamic Models
$\text{logit}(p_i) = \ln\left(\frac{p_i}{1-p_i}\right) = \beta_0 + \beta_1 x_{1,i} + \ldots + \beta_k x_{k,i}$ 	

Is the model static or dynamic?

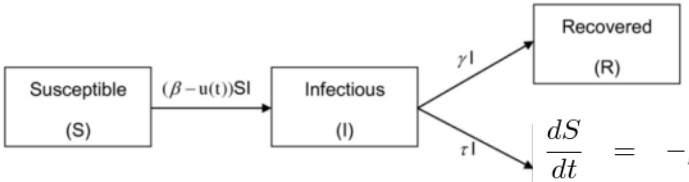
- Dynamic approaches allow phenomenon to change through time (e.g., seasonality, biocontrol agents).
- Applications to realistic landscapes is rare.

Static Models

$$\text{logit}\,(p_i) = \ln\left(\frac{p_i}{1-p_i}\right) = \beta_0 + \beta_1 x_{1,i} + \ \ldots\ + \beta_k x_{k,i}$$



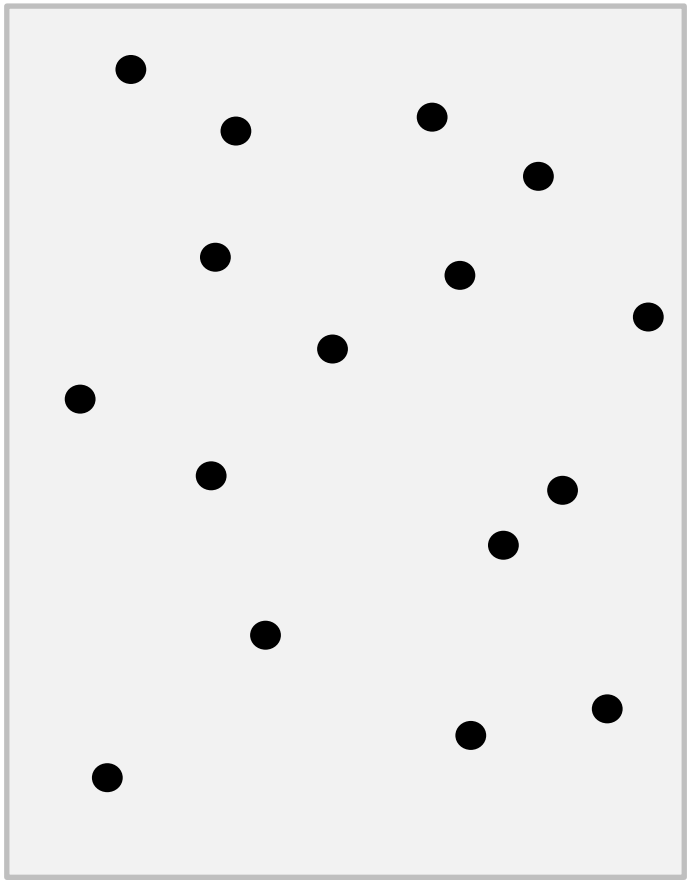
Dynamic Models



$$\begin{aligned}
\frac{dS}{dt} &= -\beta\frac{SI}{N} - \nu S \\
\frac{dI}{dt} &= \beta\frac{SI}{N} - \gamma I \\
\frac{dR}{dt} &= \gamma I + \nu S \\
N &= S + I + R
\end{aligned}$$

Is the model spatially implicit or explicit?

- Nonspatial $z = f(x) + \varepsilon$
  - Disregard spatial location



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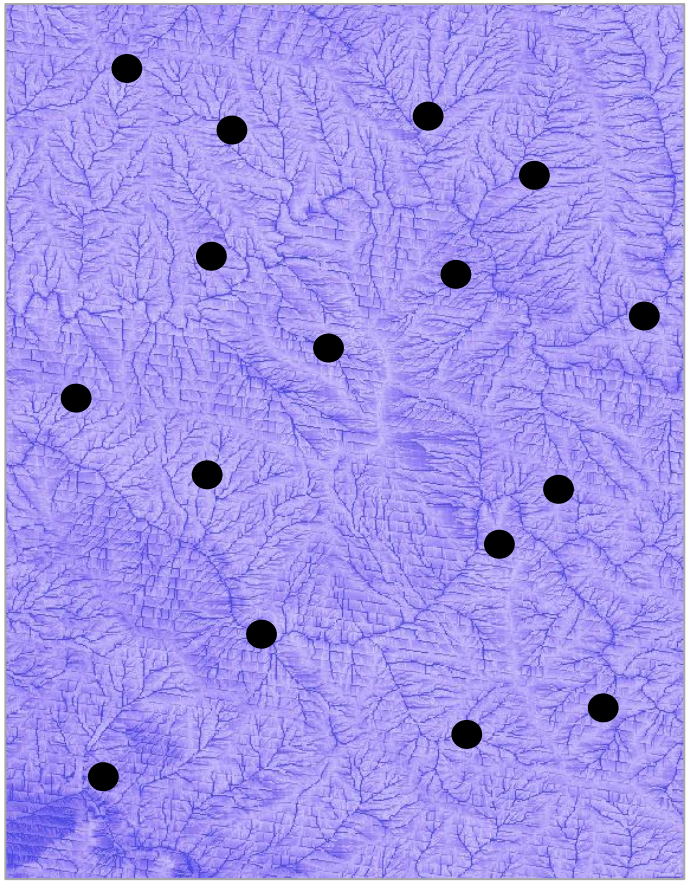
  - References spatial location of explanatory variables



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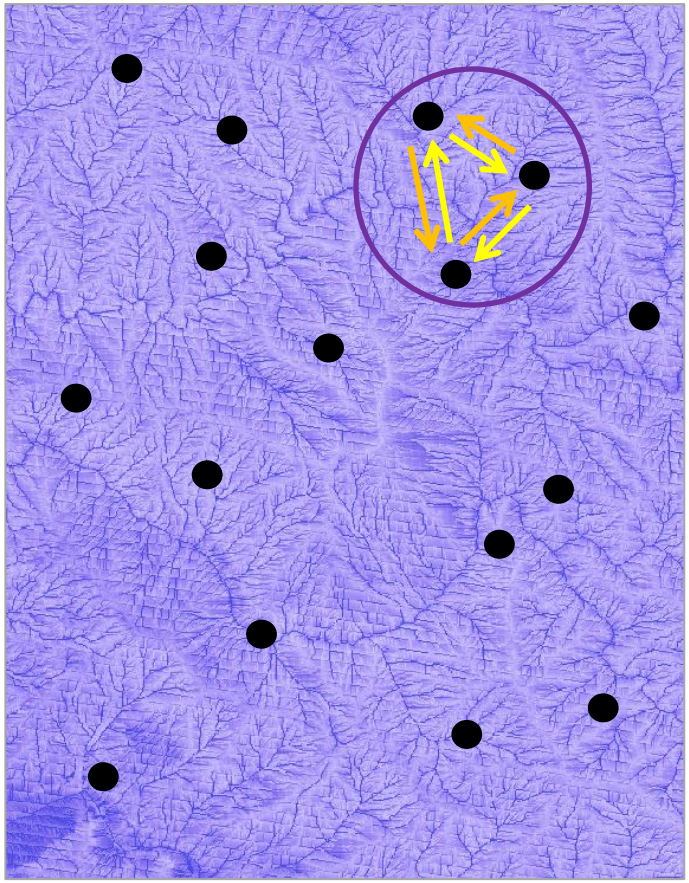
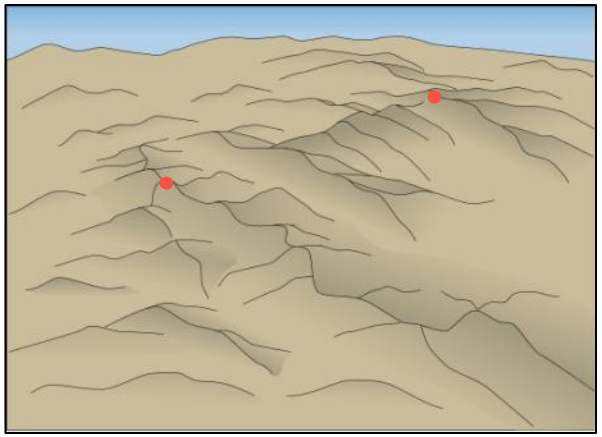
- Nonspatial $z = f(x) + \varepsilon$
  - Disregard spatial location
- Spatially implicit $z_i = f(x_i) + \varepsilon$
  - References spatial location of explanatory variables
- Spatially explicit $z_i = f(x_i) + s_i + \varepsilon$
  - Also accounts for spatial dependency among neighboring sites

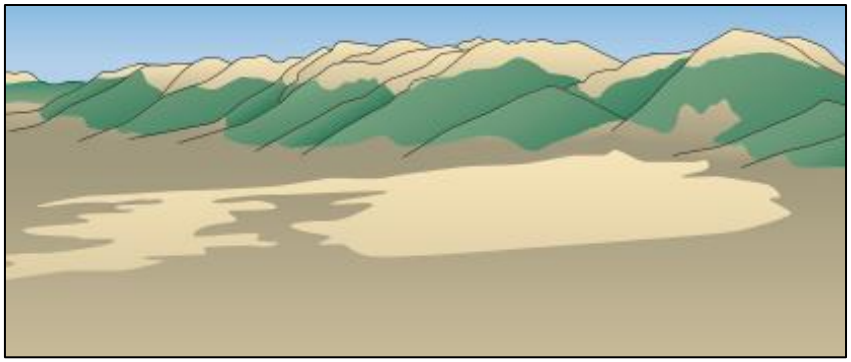


How ecologically meaningful are the variables?

- Indirect variables have no direct physiological effect on an organism's survival.
- They serve as surrogates for more functionally direct factors.

Indirect Variables	Direct Variables
• elevation • slope steepness • distance to drainage	• temperature • moisture availability • solar irradiation





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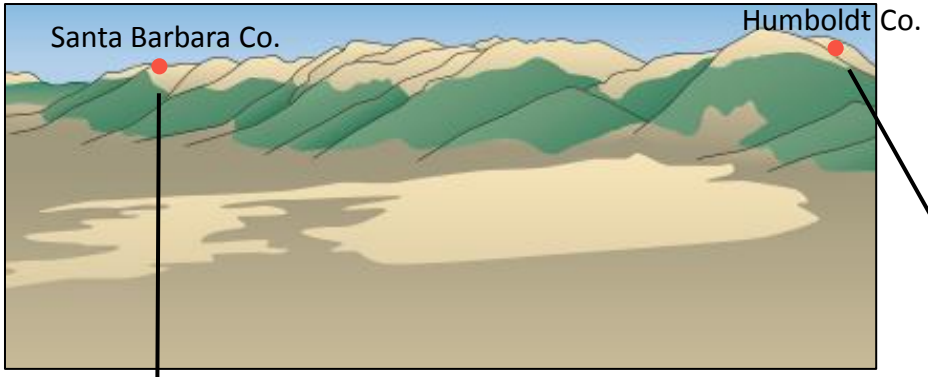
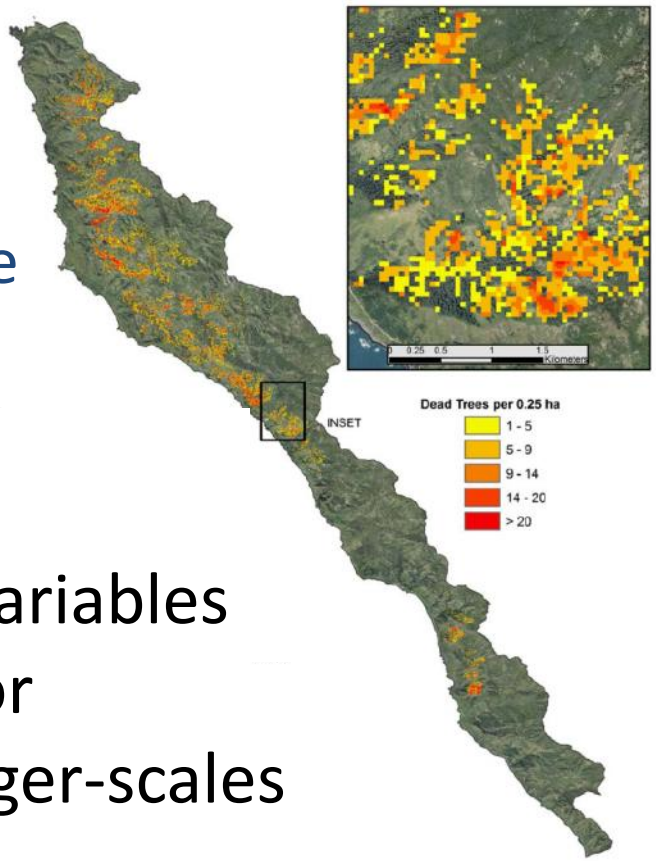


Direct Variables

- temperature
- moisture availability
- solar irradiation

Is the goal inference or prediction?

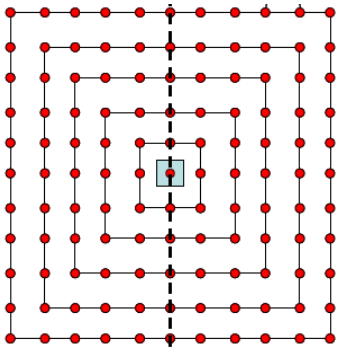
- Research objective: → obtain a mechanistic understanding / → forecast epidemic outcomes
  - ***Inference***: invoke parsimony to communicate generality of results
  - ***Prediction***: complex models may be preferred so long as they predict disease patterns.



- **Analytical trade-offs**: fine-scale variables collected in the field so important for inference not always available at larger-scales

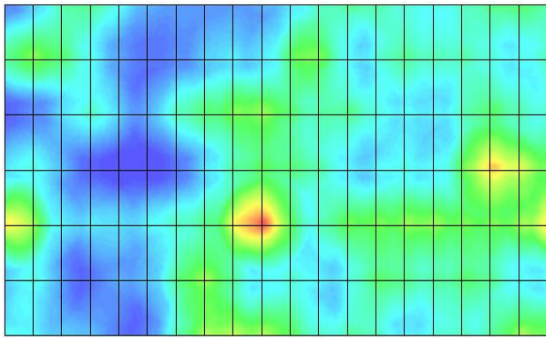
ANALYTICAL FRONTIERS

1) Using a multi-scale lens



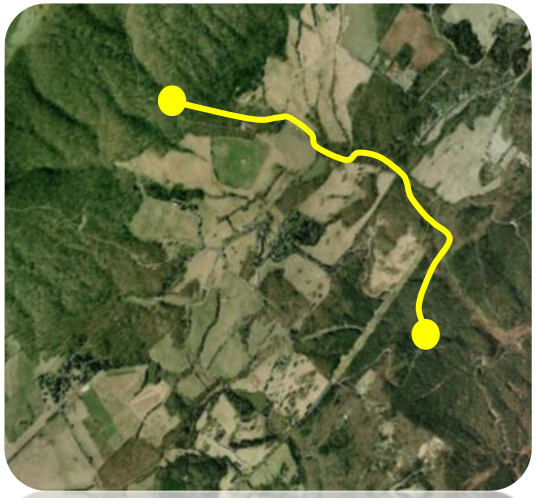
2) Incorporating landscape connectivity

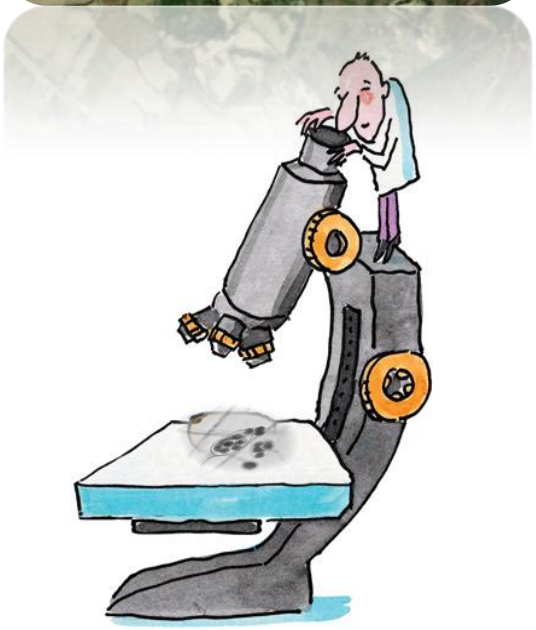
3) Optimizing disease control with dynamic landscape modeling



Using a multiscale lens

- Glancing upward versus peering downward… *'Which scale is best?'*

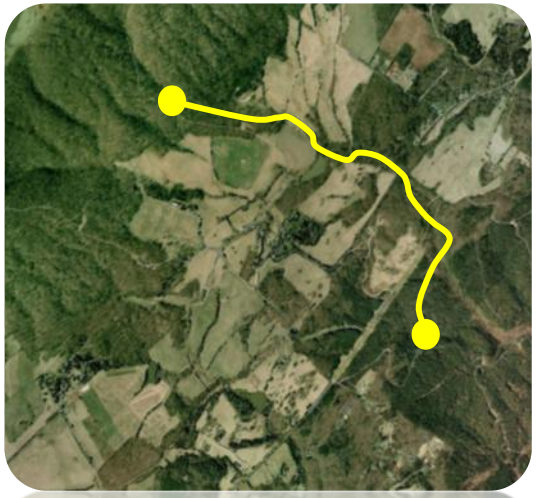


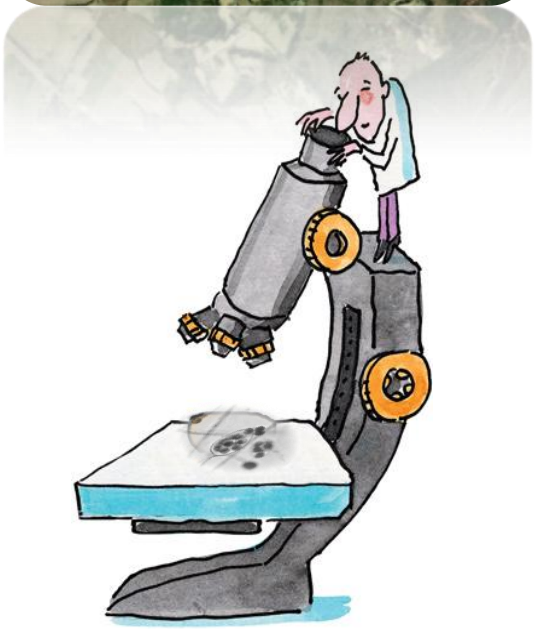


Using a multiscale lens

- Glancing upward versus peering downward… *'Which scale is best?'*

- Importance of multiscale approach:
  - Processes observed at one scale may operate differently at another scale
  - Scaling up - when relationships between patterns and processes are consistent across scales



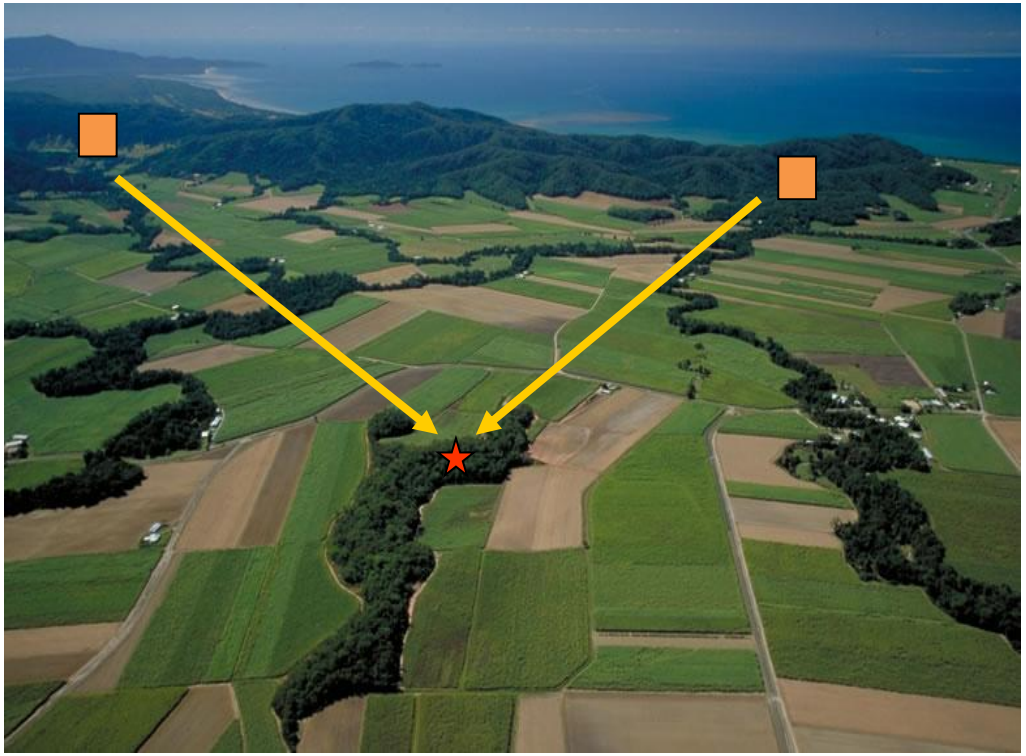


Incorporating landscape connectivity

- Dispersal of inoculum is the 1st precondition for spatial spread of emerging plant diseases.

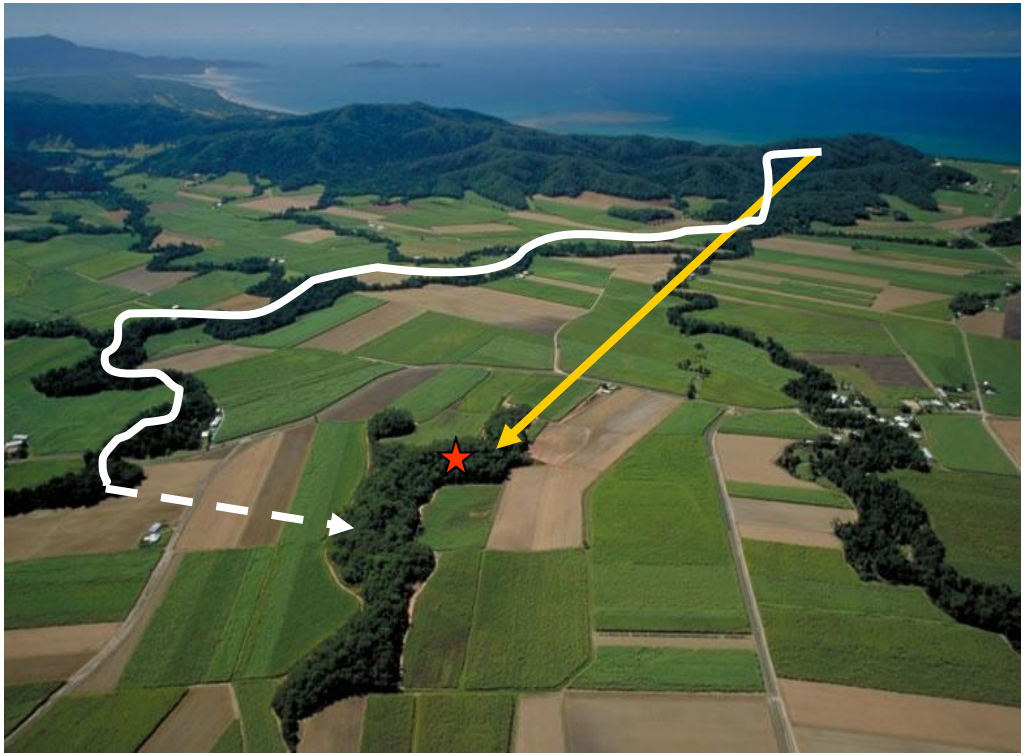
Incorporating landscape connectivity

- Human-altered corridors may facilitate disease spread by increasing landscape connectivity.



Incorporating landscape connectivity

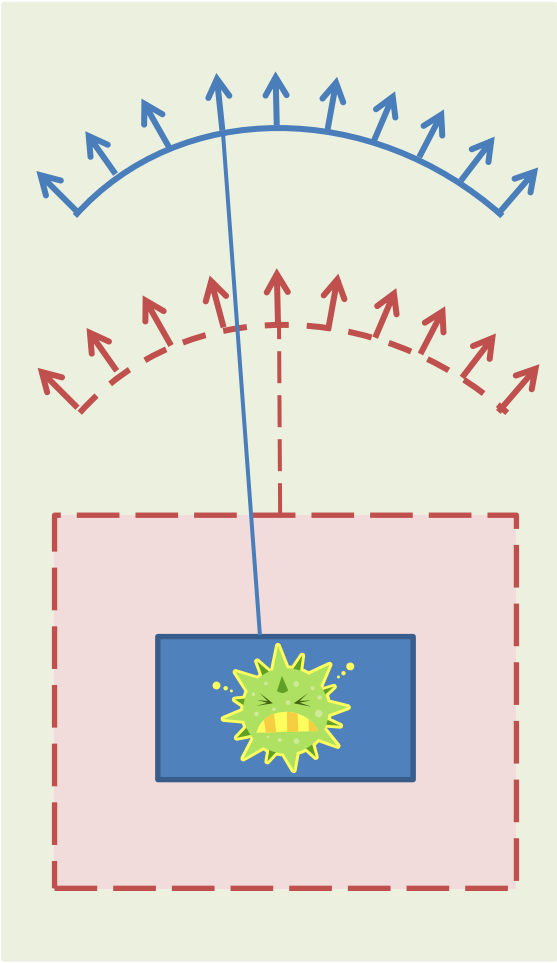
- Least cost path analyses may needed to model transmission pathways between fragmented patches of host and non-host habitat.



Optimizing disease control with dynamic landscape modeling

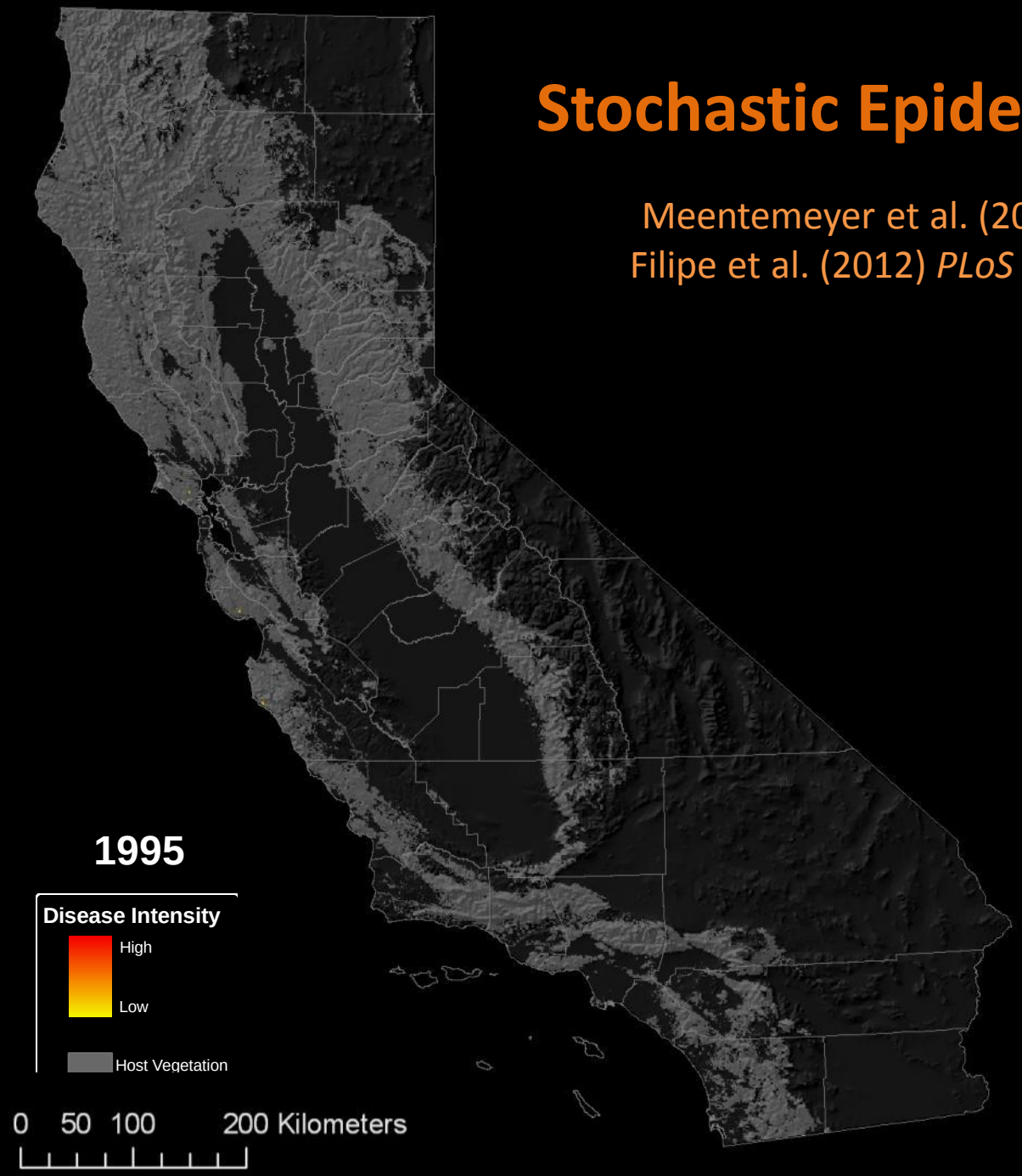
- Successful control depends on management strategies that match spatiotemporal scale of epidemic spread.

- Mathematical models of population dynamics rarely applied to realistic geographies



Stochastic Epidemiological Model

Meentemeyer et al. (2011) *Ecosphere* 2(2):art17
Filipe et al. (2012) *PLoS Comput. Biol.* 8:e1002328



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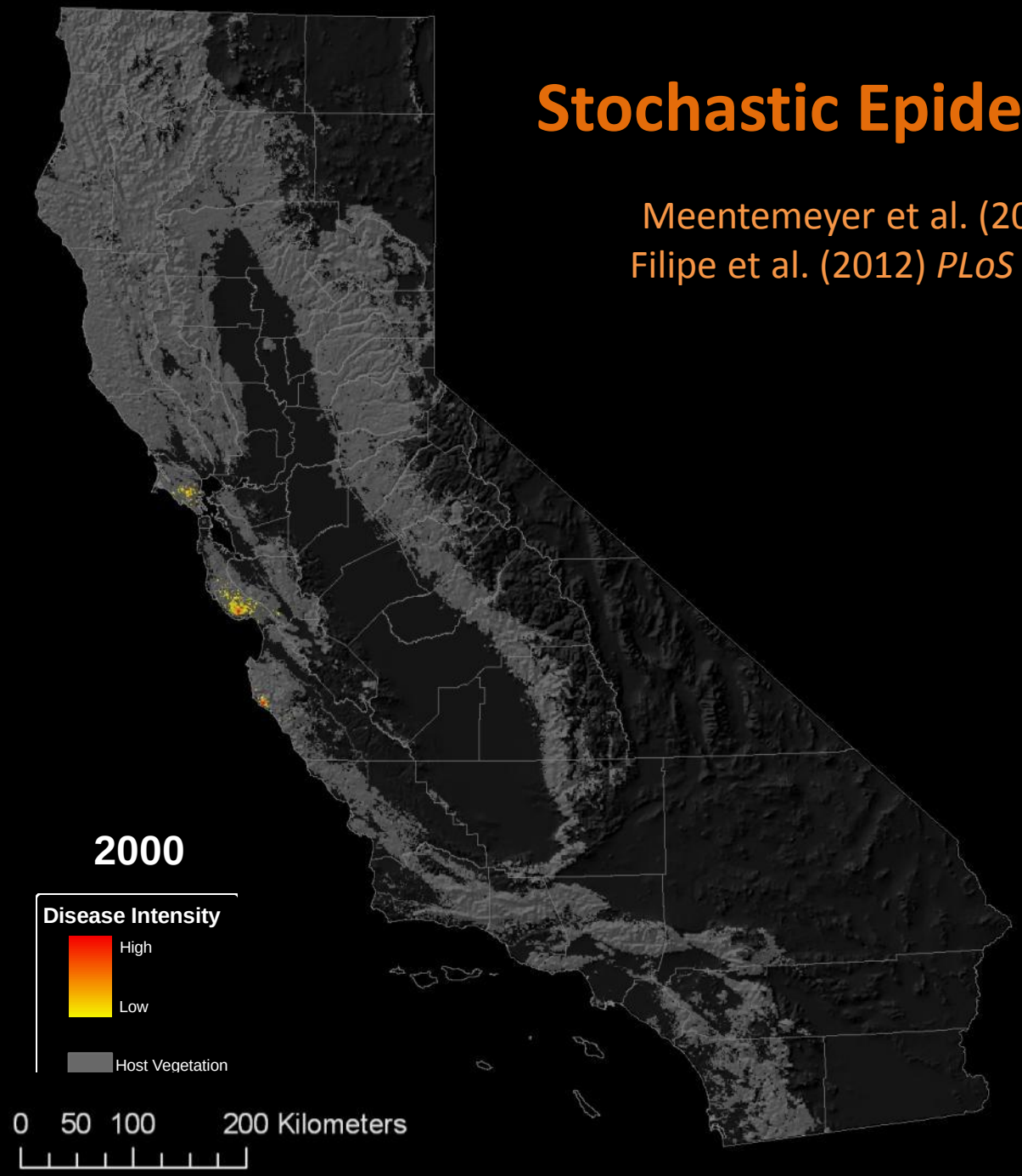

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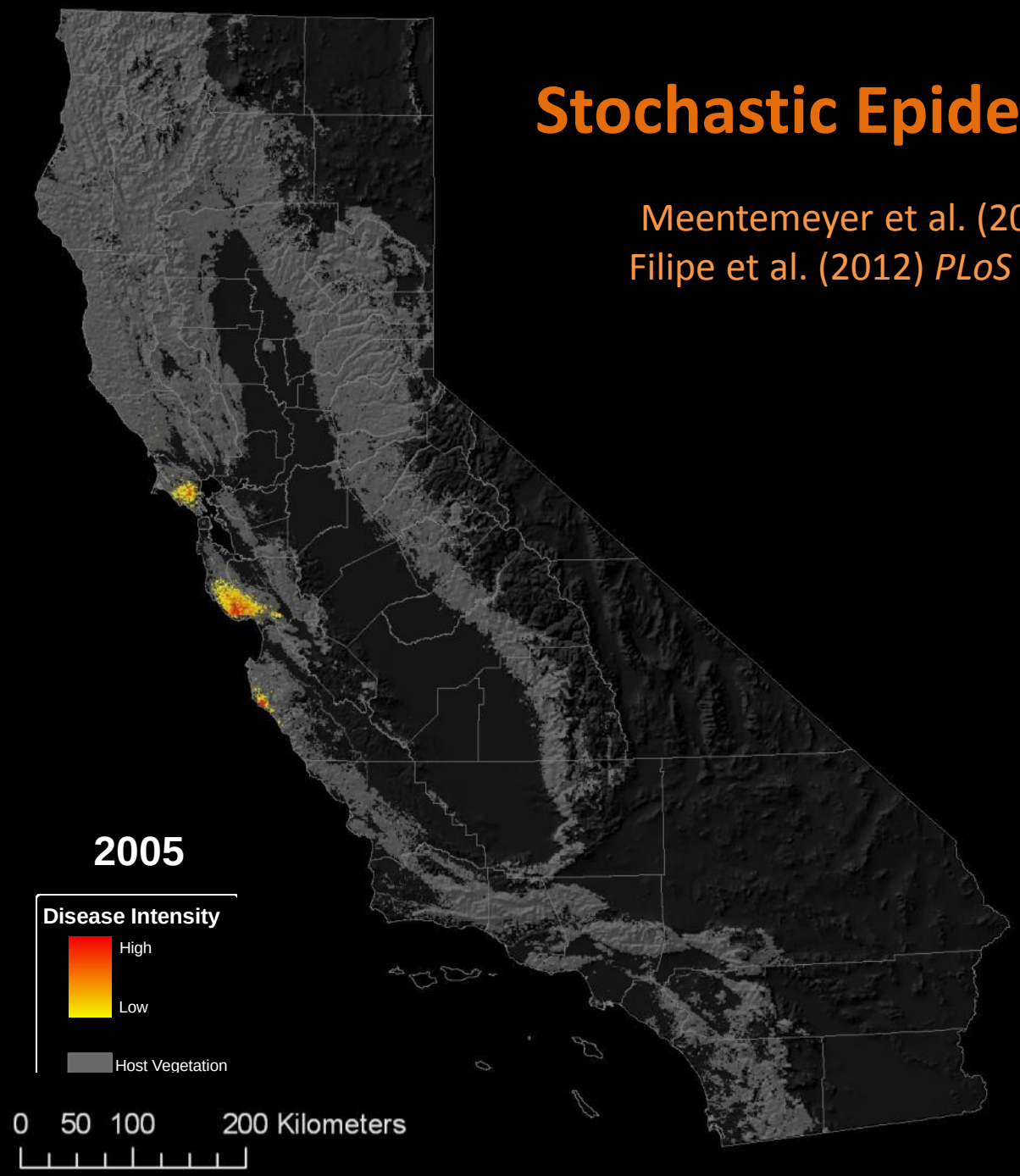

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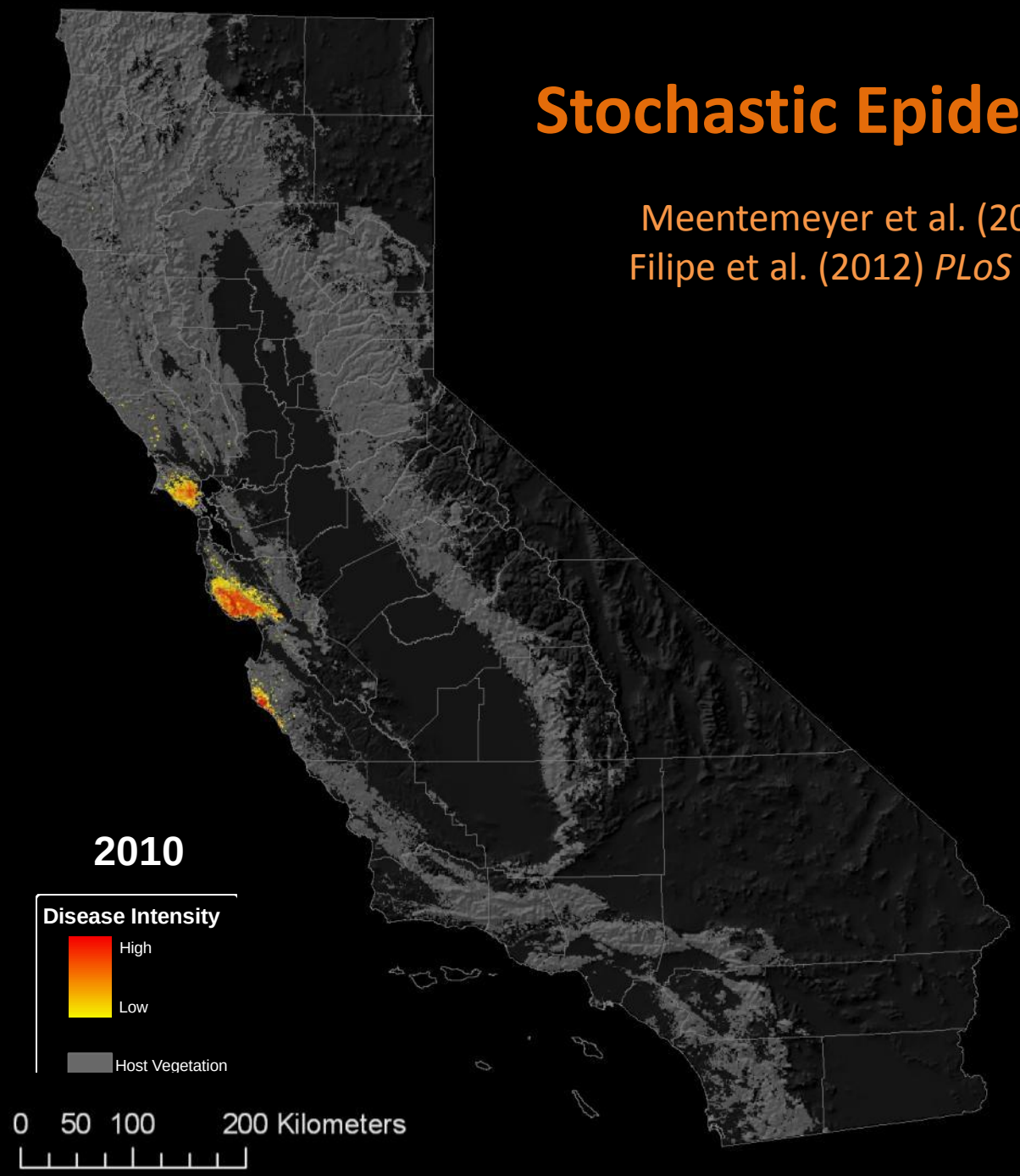

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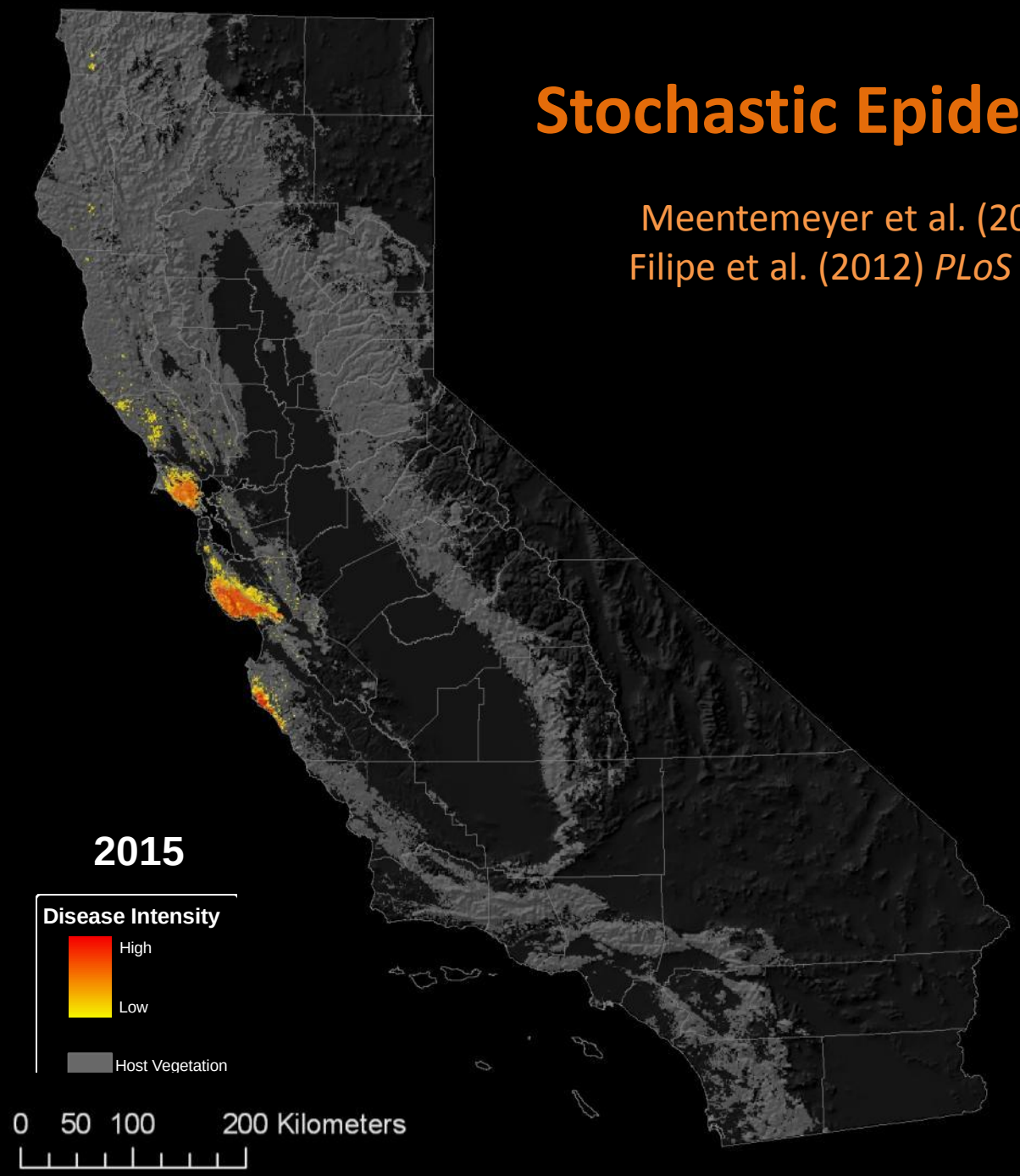

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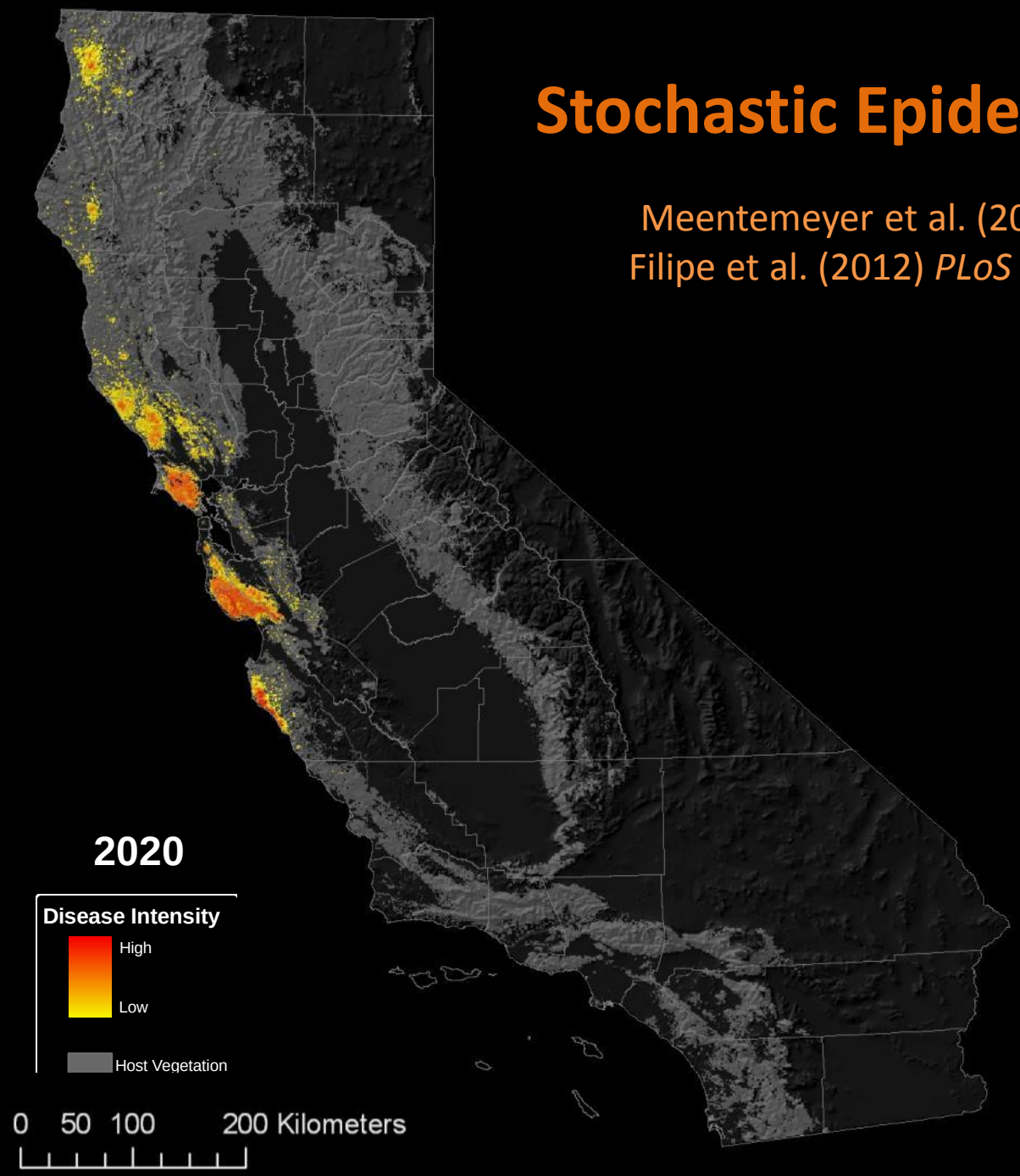

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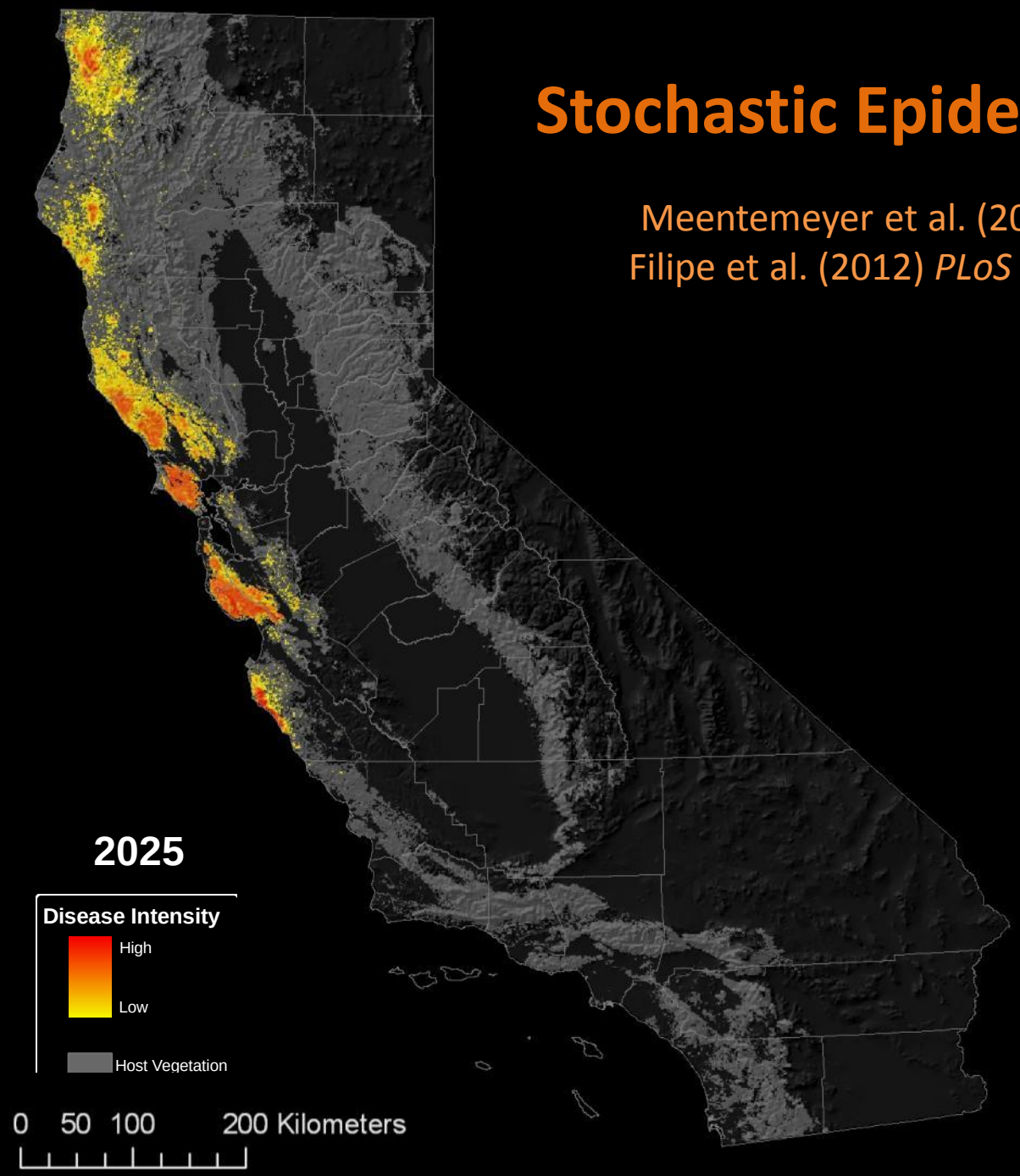

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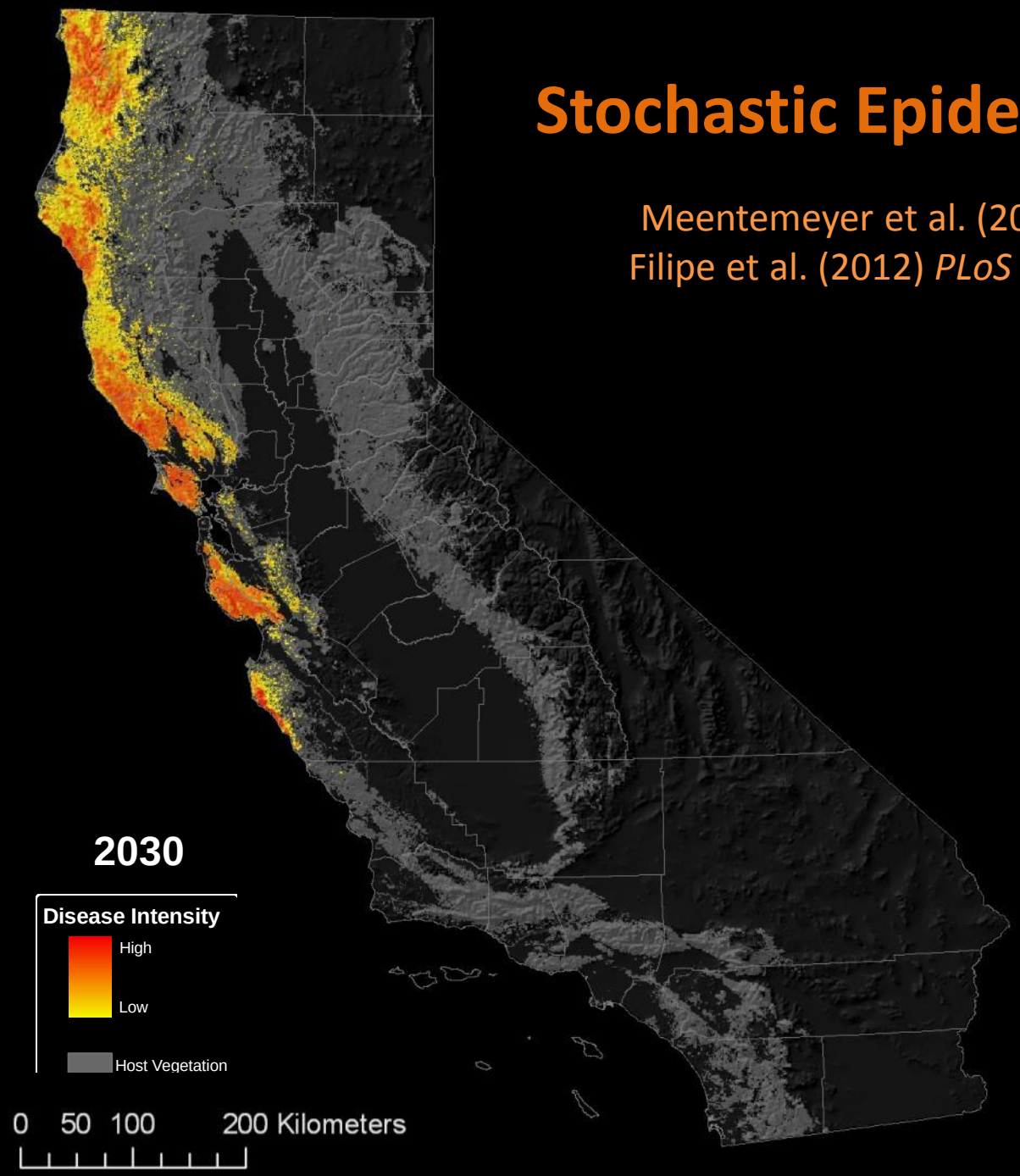
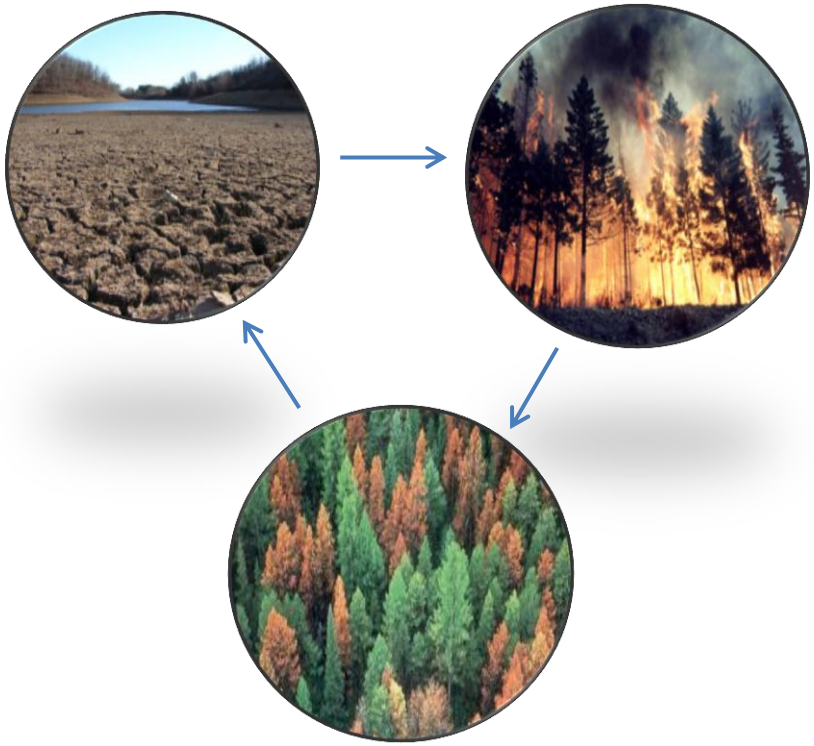


WHAT'S NEXT…?

- Understanding disease as a component of interacting ecological disturbances.



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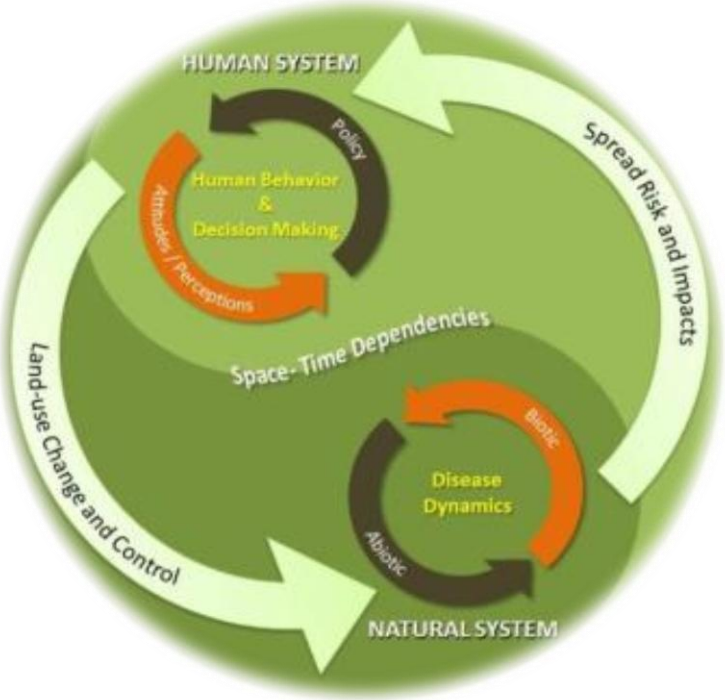
- Scaling up the ecological and economic impacts of disease.

WHAT'S NEXT…?

- Understanding disease as a component of interacting ecological disturbances.
- Scaling up the ecological and economic impacts of disease.
- Examining disease dynamics as a coupled human-natural system.



Thank you for your attention!

Funding Agencies

- NSF-NIH Ecology of Infectious Diseases program
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