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Linking novel resources & infectious disease dynamics in wildlife

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Summary

Although land-use changes send many wildlife populations into decline, some species benefit from novel food resources available in human-altered habitats. This resource provisioning can affect infectious disease dynamics, although this has rarely been quantified. Our goal is to provide a conceptual framework to examine provisioning and identify key mechanisms. We also characterize the range of disease outcomes through meta-analysis and outline a mechanistic model that can capture the net effect of provisioning on disease transmission.

Background

Humans can accidentally or intentionally supplement wildlife with novel food sources. **By providing abundant and accessible resources, provisioning can result in sedentary, aggregated, and well-fed populations**. Shifts in dietary composition can have direct effects on health and might also alter infectious disease dynamics, as demonstrated through several high-profile examples.

In the case of Hendra virus in flying foxes in Australia (L), accidental provisioning in the form of gardens and fruit trees led to sedentary bat colonies in urban centers. Modeling simulations revealed that **increased recruitment of susceptibles through births and loss of connectivity between colonies** resulted in explosive outbreaks upon reintroduction of virus due to loss of immunity over time.

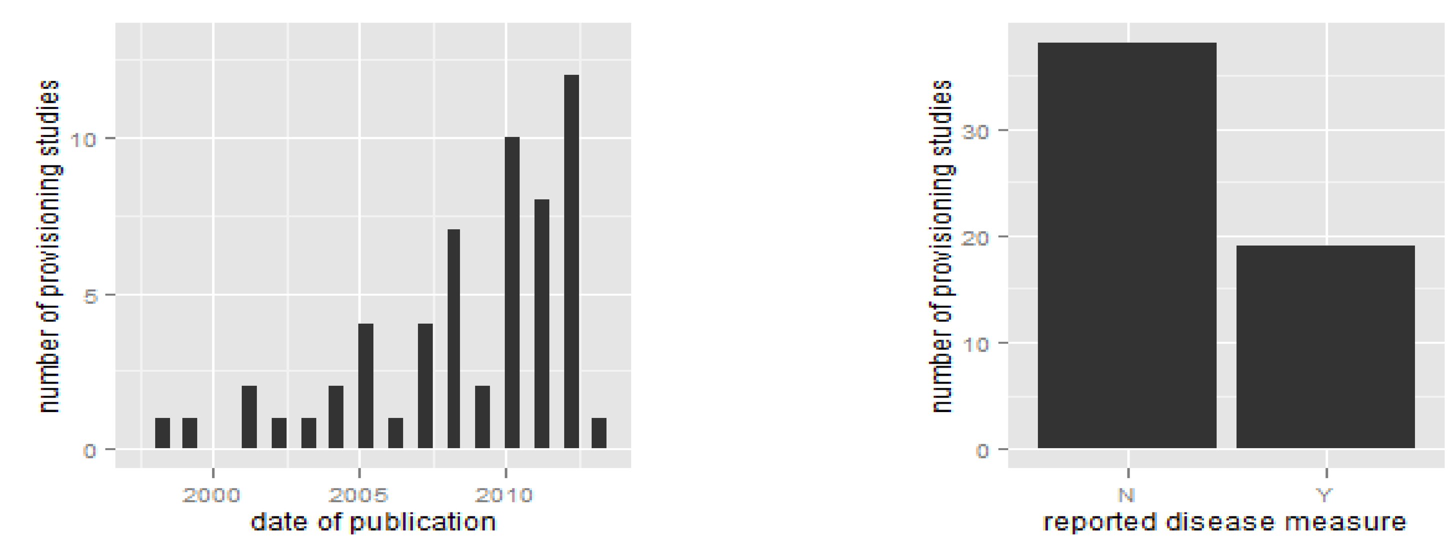
In the case of gastrointestinal parasites in long-tailed macaques in Indonesia (R), intentional provisioning in the form of handouts from tourists instead was associated with decreased pathogen prevalence and intensity. **Decreased foraging range and increased nutrition** in provisioned populations likely led to reduced exposure to and improved recovery and clearance of environmental-borne pathogens.

Further synthesis and analysis is needed to uncover the pervasiveness of this phenomenon and identify ways that provisioning affects infectious disease.

Patterns of Provisioning

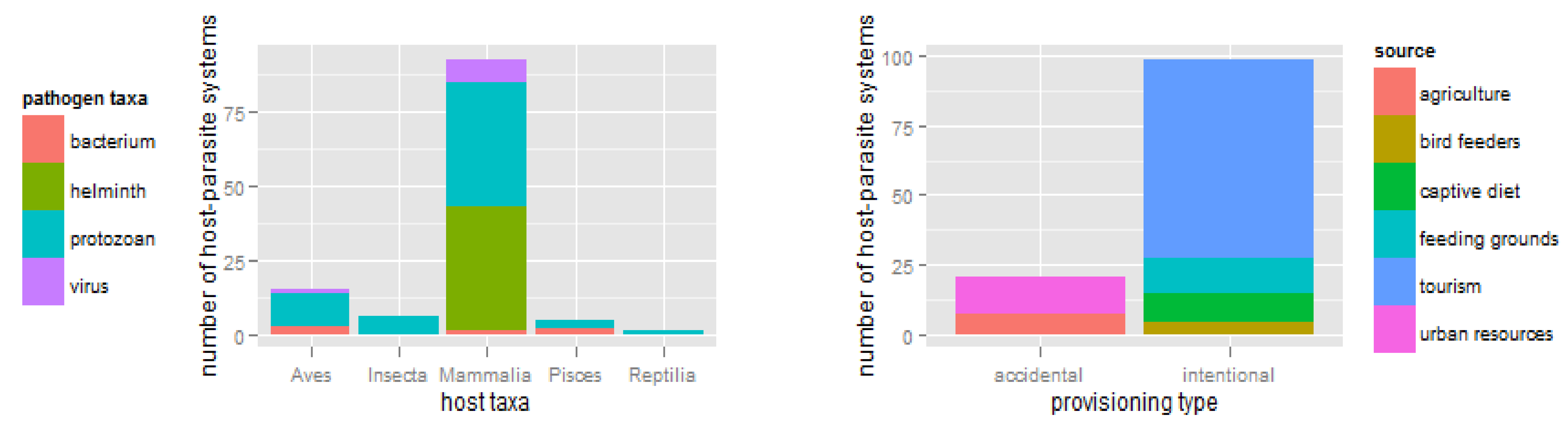
To determine patterns of provisioning from published studies, we conducted a **meta-analysis** using articles identified via **Web of Science** using search terms related to resource provisioning, pathogen taxa, foraging ecology, and disease processes. We only selected studies that explicitly focused on resource provisioning and held to a criterion of comparisons between provisioned or unprovisioned groups, resulting in **57 studies since 1998**. Although provisioning studies have increased over time, only **30% reported a measure of disease** (Figure 1).

Figure 1: publication trends



We extracted data for **each host-pathogen combination and disease measure**, resulting in 120 lines of data. Variables included provisioning type and source, host taxonomic class and dietary niche, pathogen type and disease measure, study design, and location. **Preliminary results** indicate that provisioning is primarily studied in **mammals**, with **protozoa** being the main pathogen group sampled. **Intentional provisioning** is studied more than accidental forms, and the best-studied sources include **tourism, urban resources, and feeding grounds** (Figure 2).

Figure 2: provisioning patterns



Key Mechanisms

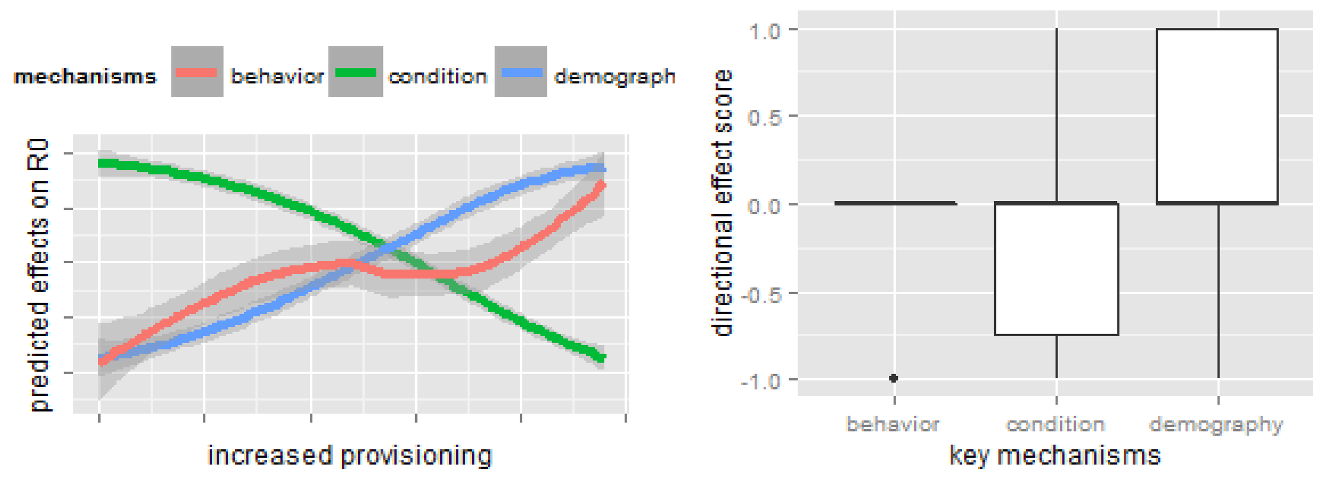
We identified **three primary mechanisms** by which provisioning influences infectious disease dynamics, all of which together could **interact to produce non-linear patterns in overall transmission** (Figure 3, L).

Condition. Provisioning could decrease foraging effort and provide nutritional content, increasing host resources for immune defenses and thus boosting host resistance, recovery, or tolerance to infection.

Demography. Steady resources could increase births or lower death rates, leading to a buildup of susceptibles and increases in transmission (unless lifelong immunity protects against later infections).

Behavior. Aggregated or sedentary groups might form around novel resources, increasing contact rates and favor pathogen transmission. Aggregation could also lower host exposure to pathogens through reduced movement.

Figure 3: provisioning mechanisms



By assigning a **directional effect on disease measures from -1 to 1**, our preliminary analysis shows a **range of outcomes** for each mechanism (Figure 3, R). Current work involves extracting effect sizes from each study to **determine the magnitude** of each mechanism on disease and **analyze the major predictors** of provisioning.

Modeling Approaches to Provisioning

Because provisioning will **simultaneously alter multiple processes that can have opposing effects on infectious disease**, mechanistic models are critical for teasing apart mechanisms and predicting the net outcome on transmission. We here outline a **SEIR model** where parameters driving demography, susceptibility, and contact are coupled to a **functional form of resource availability**. Parameters in **blue** are those expected to increase with provisioning; those in **red** will decrease.

$\boldsymbol{\beta}$ = transmission rate, $\boldsymbol{\sigma}$ = probability of infection given exposure
$\boldsymbol{\gamma}$ = recovery rate, $\boldsymbol{\omega}$ = rate of immunity loss, $\boldsymbol{\alpha}$ = disease mortality
$\boldsymbol{b}$ = birth rate, $\boldsymbol{d}$ = death rate