

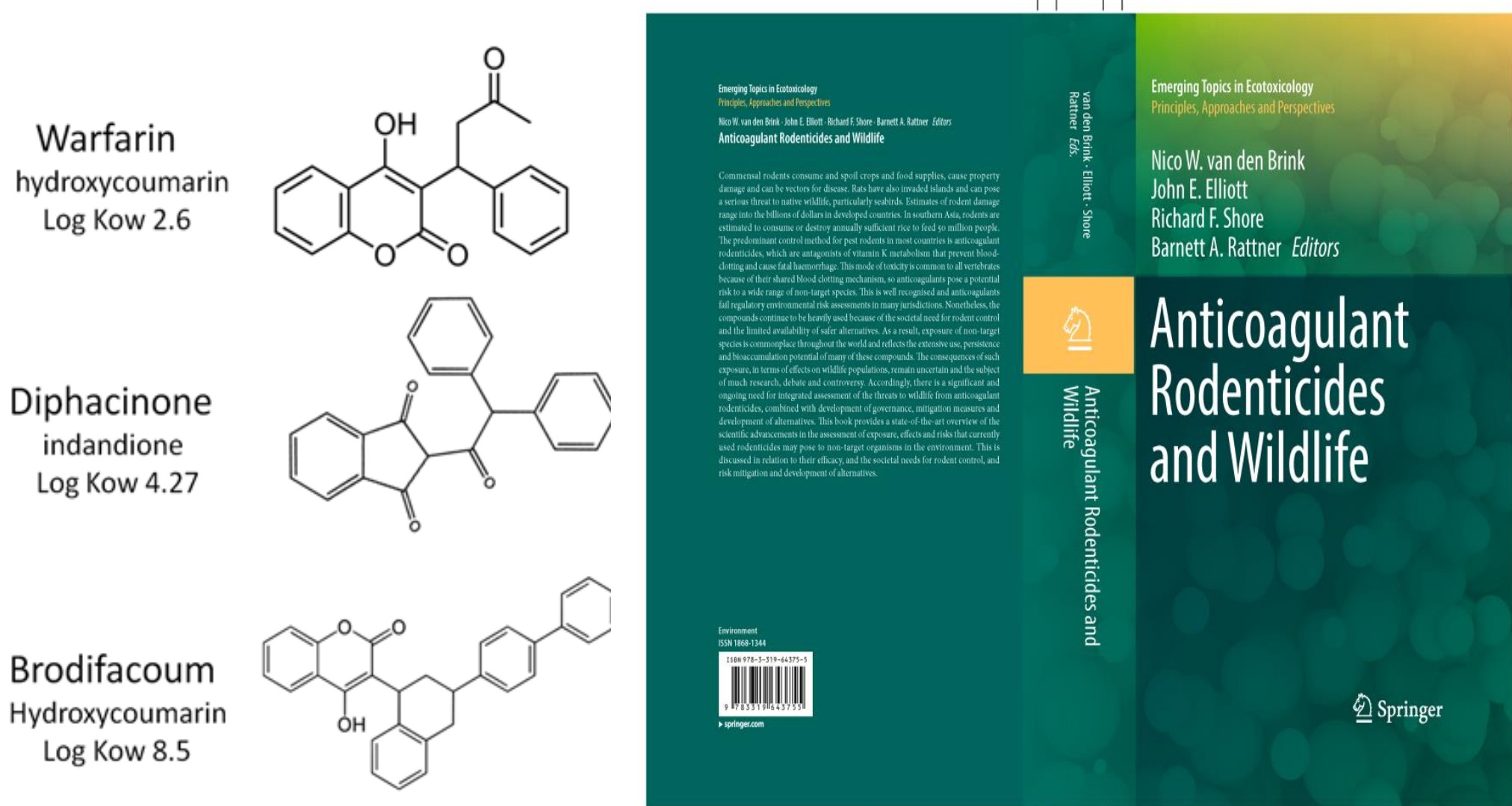
A synthesis of the interactions between anticoagulant rodenticides and wildlife

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Introduction

Rodents affect human food resources by consumption and fouling and vector disease – overall annual costs in the US alone exceed \$20 billion. Rodents inadvertently introduced to islands can also devastate endemic species. Various methods have been employed to control commensal rodents. Chemical control has been common practice for nearly a century. Currently, anticoagulant rodenticides (ARs) are the cornerstone of rodent control throughout the world.



A new reference text (ISSN 1868-1344), with 24 contributing scientists, addresses AR use, regulation, exposure pathways, toxicity, mechanism of action, pathology, pharmacokinetics, genetic resistance, non-target risk and its mitigation, pesticide alternatives and integrated pest management.

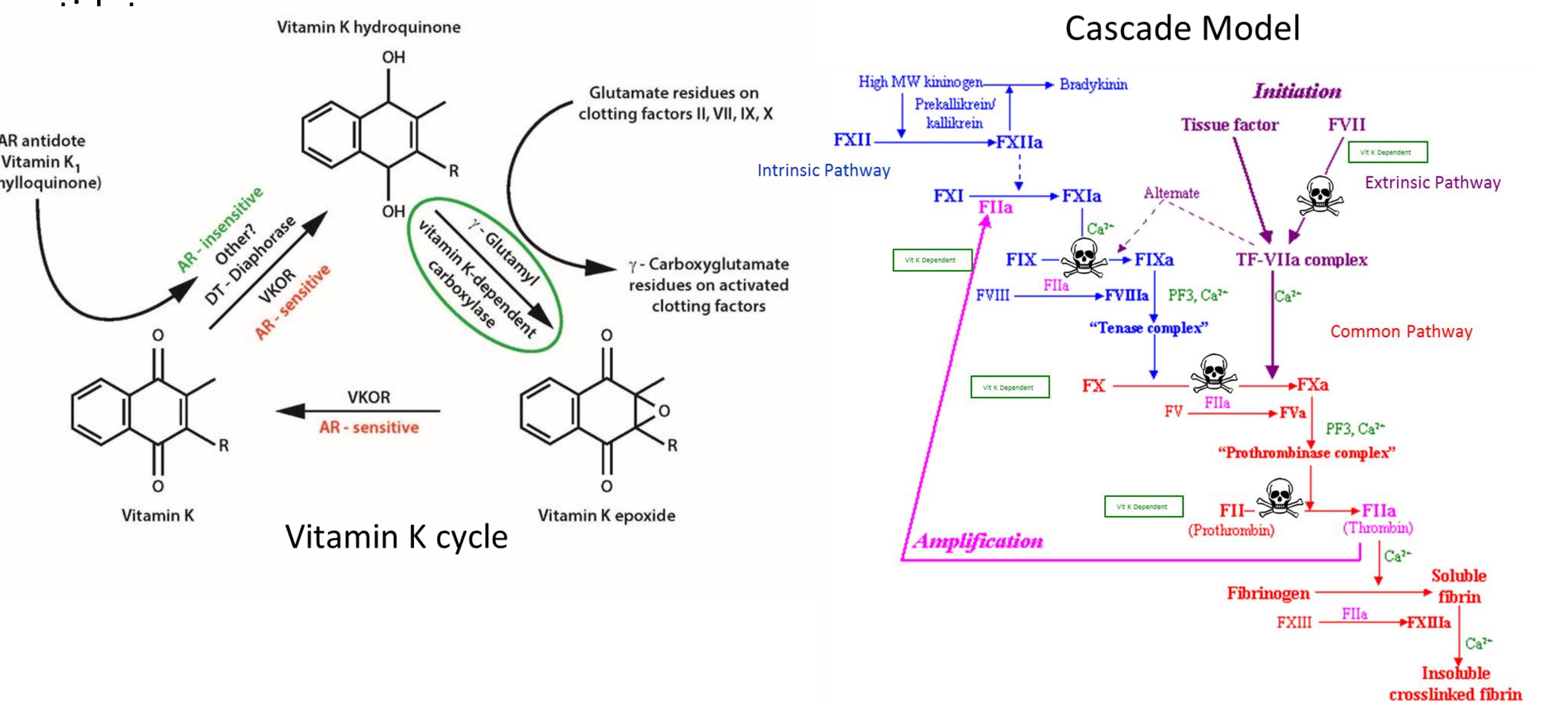
Global Use

ARs are the mainstay of rodent control in agriculture, urban and suburban settings, and island restoration projects.



Mode of Action

ARs harm a wide range of predatory and scavenging non-target species because they affect the blood clotting cascade, a highly conserved physiological mechanism among vertebrates. ARs bind to vitamin K epoxide reductase (VKOR) and impede activation of clotting factors, which eventually results in coagulopathy. Vitamin K₁ is an effective

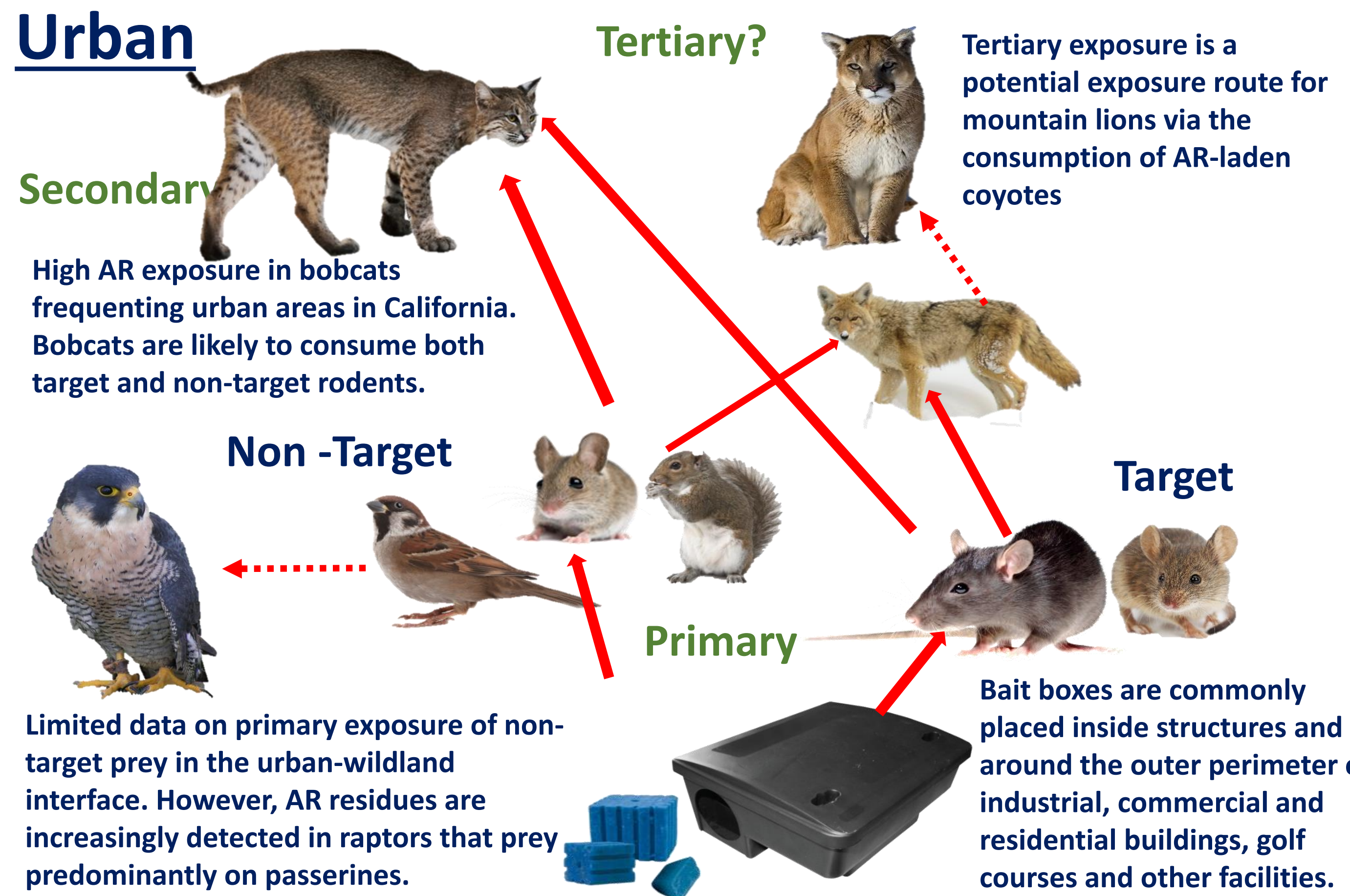


Inter-specific toxicological trends

- Most toxicological data are for common lab species
- Mammals generally more sensitive to ARs than birds
- Second-generation ARs (SGARs) more toxic and persistent than first-generation (FGARs)
- Causes of large inter and intra species variation in sensitivity to ARs are unclear

Wildlife exposure pathways

Ecological factors that drive AR uptake by non-target species depend on the landscape and the management of habitats within that landscape. An example of exposure pathways in an urban landscape is shown below



Primary Exposure

Non-target primary exposure is more prevalent in granivorous and herbivorous species that will feed on cereal baits. The extent of exposure in non-targets is driven in part by species traits (dietary and habitat preferences, home range size, mobility) and such exposure can lead to acute mortalities, and in some instances declines in species abundance.

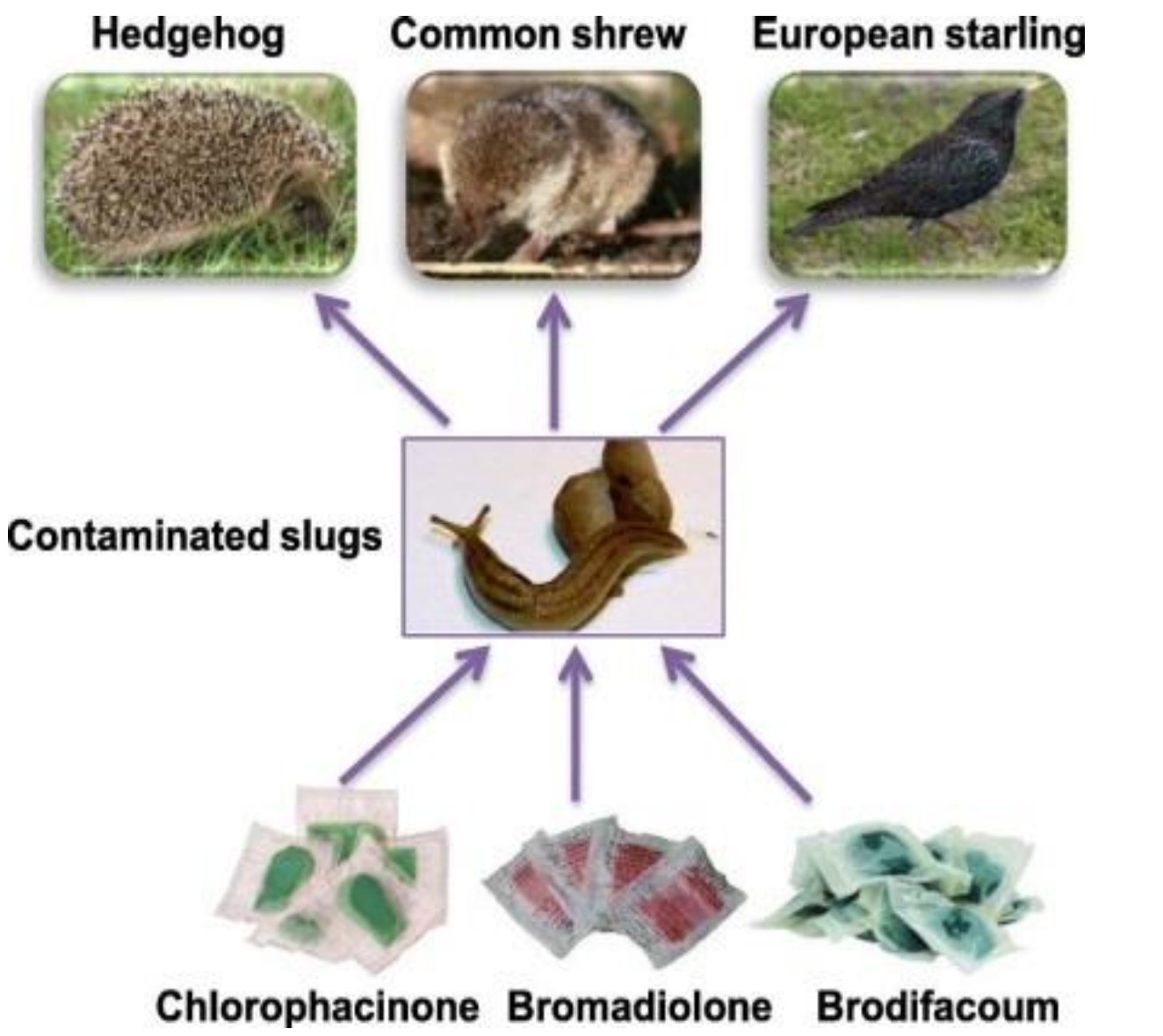


Horned lark droppings at a prairie dog burrow that was treated with Rozol (green)

Robin and brown rat eating bait

Secondary Exposure

AR exposure in predators would seemingly reflect opportunistic feeding and highly variable nature of primary exposure in prey. However, the longer life span and larger foraging areas of predators, coupled with the bioaccumulation of ARs in tissues, suggest that secondary exposure is integrated both temporally and spatially. Notably, 58% of predators (2414 out of 4187 specimens) analyzed worldwide have AR tissue residues, evidence that secondary exposure to ARs is a global phenomenon.



A high degree of AR exposure in shrews and hedgehogs (predominantly insectivorous) suggests significant 2° exposure through eating contaminated insects and possibly carrion.

Signs of Toxicosis

Ante-mortem signs (lethargy, unresponsiveness) are not specific to AR toxicosis. Pallor of mucous membranes and hemorrhage can be indicators of potential AR toxicosis, and in combination with prolonged blood clotting time, are more diagnostic. While AR residues demonstrate exposure, unequivocal diagnosis requires a combined approach of clinical investigation, measurement of blood parameters, pathological evaluation and forensics.

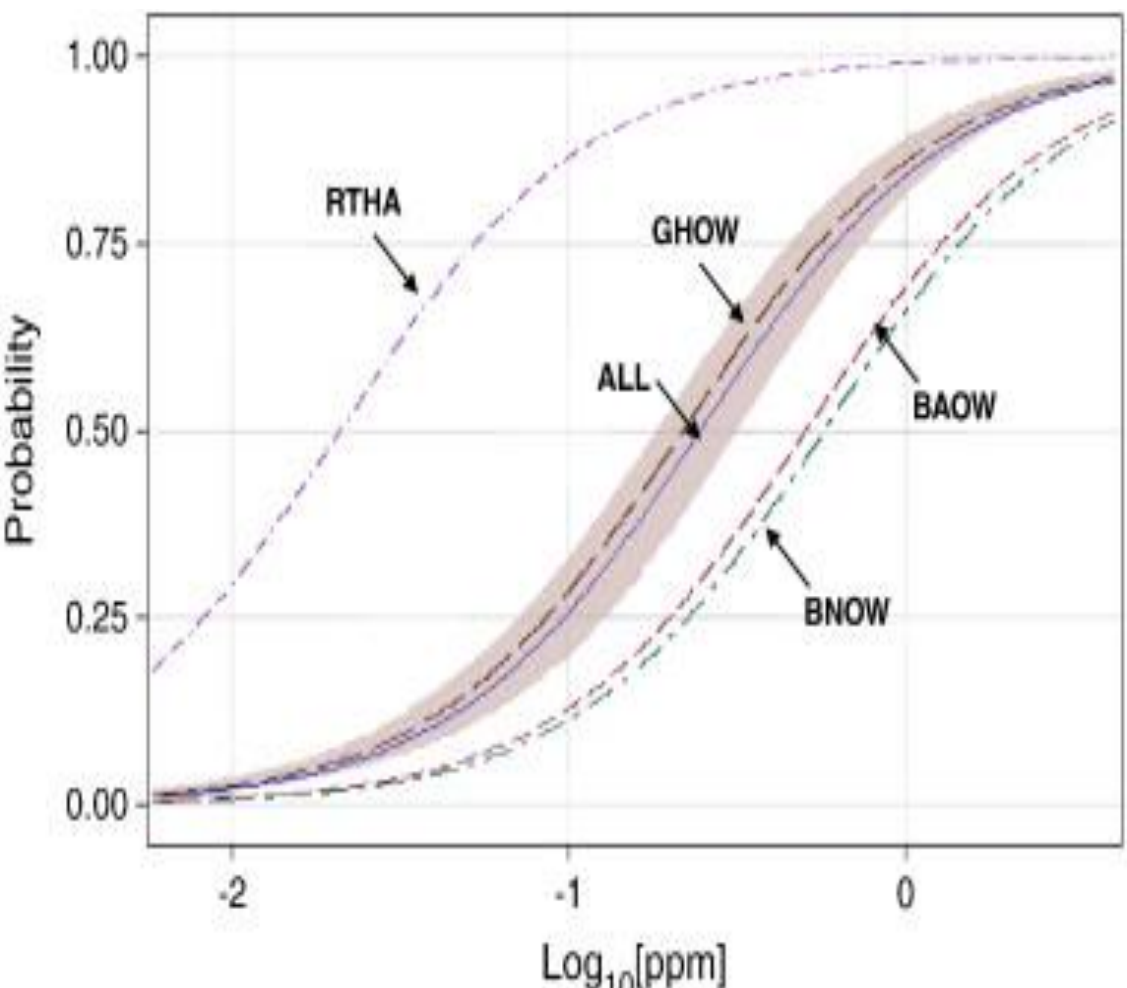


Oral cavity of a great horned showing extreme pallor of the mucous membranes due to blood loss and anemia

Population-level effects?

Some island eradications have led to AR-mediated population decline of predators. Current practices now involve capture, temporary removal and subsequent re-establishment of “at-risk” species.

Cause-effect relations leading to population declines from 2° exposure are difficult to detect and seemingly rare. Tissue residues in combination with other signs of intoxication may be used to estimate probability of AR-induced mortality in populations.



Probabilistic mortality curves associated with liver SGAR residues of various raptors and owl species

Resistance

Tolerance of rodents to ARs first became apparent in the 1950s. There is both a genetic (point substitutions on VKOR) and metabolic (increased expression of CYP3A) basis target species. There is some evidence that resistant target rodents carry greater AR burdens, and thus present a greater risk to non-target predators. Resistance monitoring is needed in order to adapt the use of rodenticides locally.

Regulation, alternatives and mitigation

The risk that ARs pose to wildlife is evident from widespread implementation of regulation while continued authorization reflects concerns about the consequences of limiting effective rodent control and its associated benefits. There are alternatives to ARs (bromethalin, cholecalciferol etc) but they also pose significant hazard. Forcing a shift from ARs to other chemistries may substitute one set of risks for another.

Given the risk to wildlife from ARs, mitigation measures are justified. These may include:

- use of protected bait stations
- replacing permanent baiting with pulsed baiting
- restricting AR use by non-professionals
- avoid treating areas with high density of non-targets
- integrated pest management



Key needs and information gaps

- Better tools to estimate AR-induced mortality in wildlife populations
- Knowledge of sub-lethal effects and their ecological significance
- Extent/importance of AR exposure in lower vertebrates and invertebrates
- Information on wildlife exposure in urban habitats, where AR use is greatest
- Effectiveness of mitigation and IPM approaches for rodent control