

2009

## Pesticide Safety 2009 - New Insecticides and their Comparative Safety

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# **New insecticides and their comparative safety**

**AL Averill & MM Sylvia  
University of Massachusetts**

# New insecticides and their comparative safety

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- In cranberry, old chemistries are generally toxic to humans and other non-targets



- New chemistries are generally non-toxic to humans



- Variation in toxicity to other non-targets

# Why some insecticides are a human health risk

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- The nervous system of the insect is targeted by certain insecticides
- Human and insect nervous systems have similar characteristics
- Organophosphates
  - Diazinon
  - Lorsban
  - Orthene
  - Imidan
- Carbamates
  - Sevin

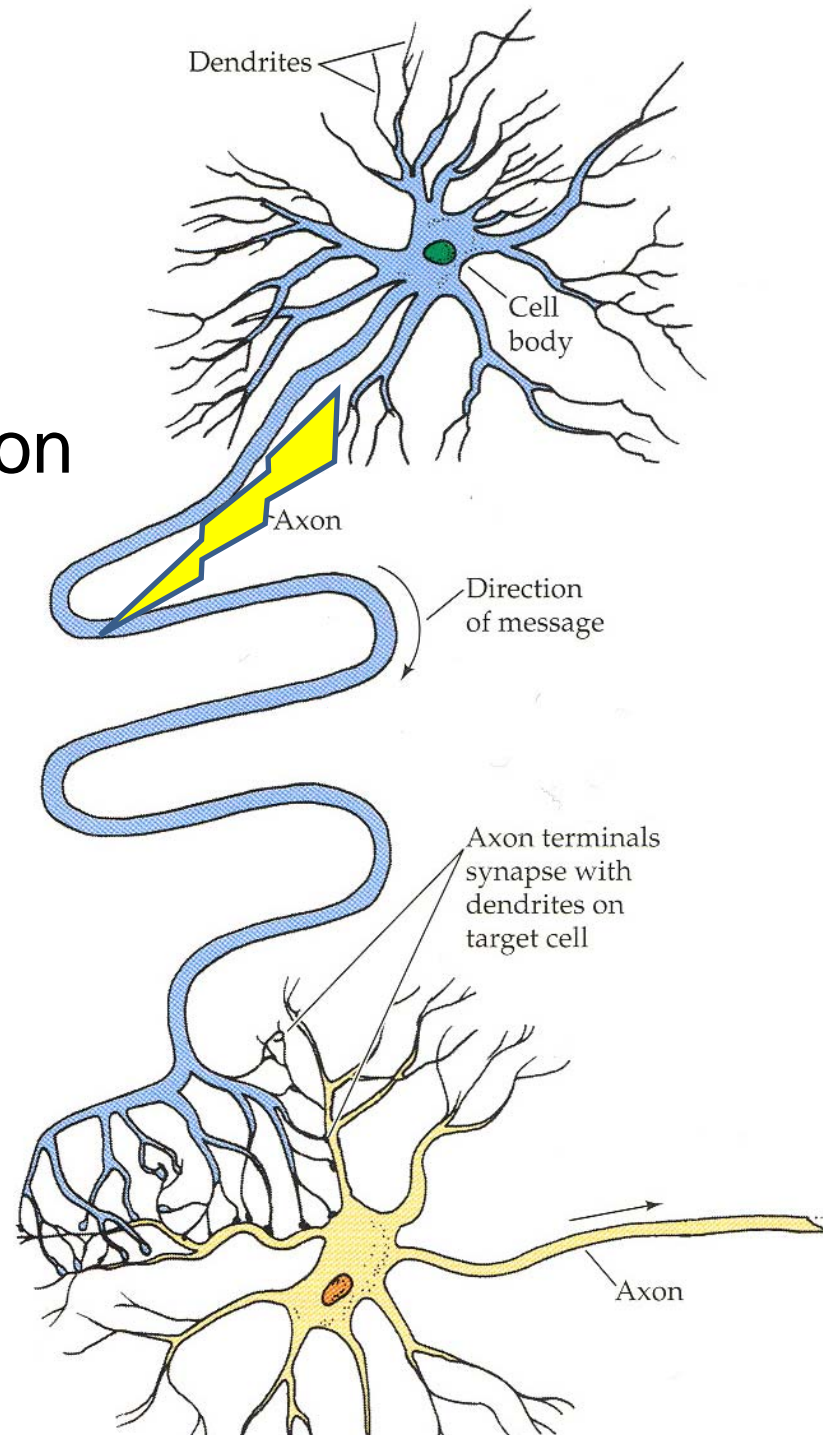
# Nervous system

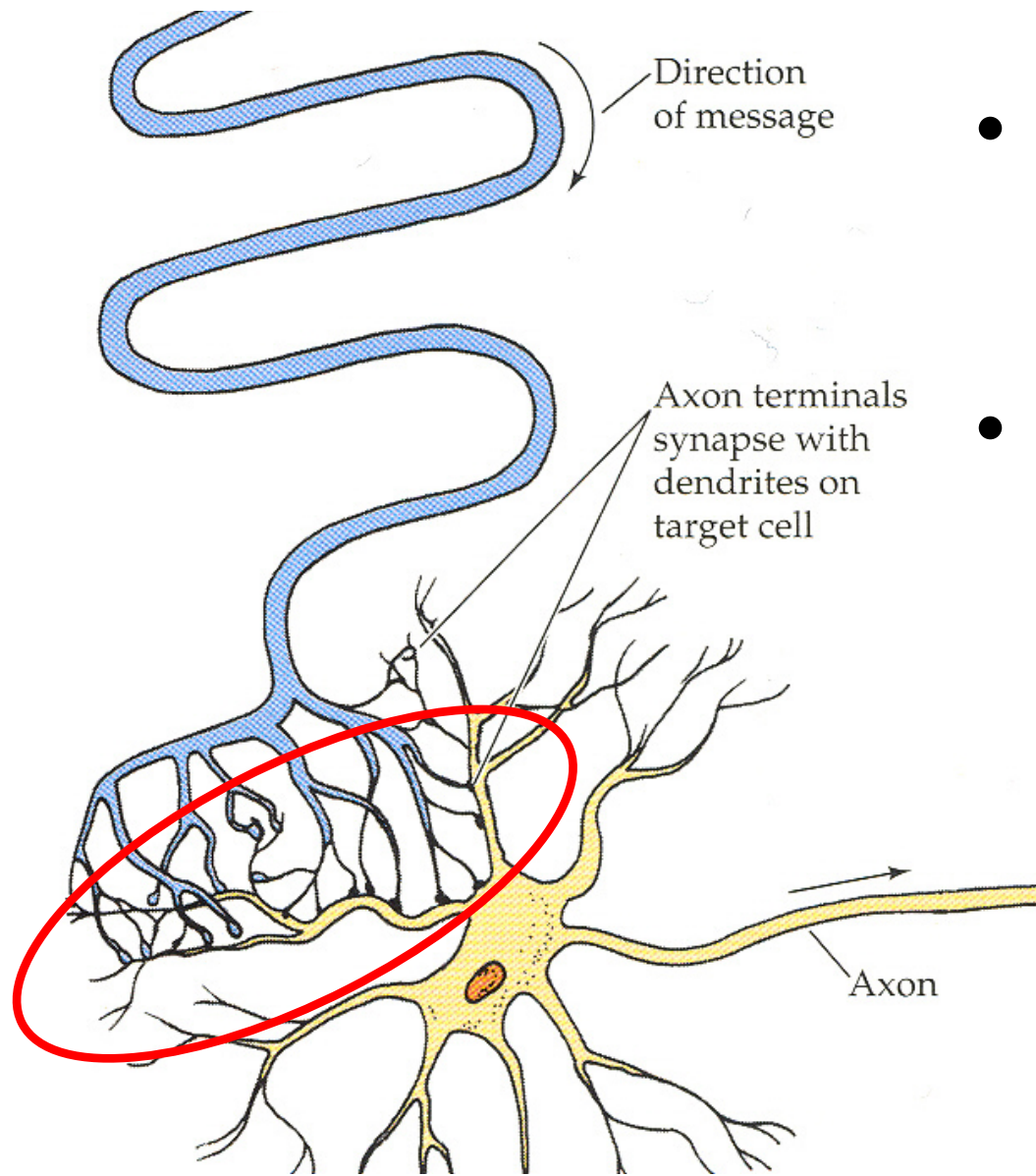
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- Neuron (nerve cell)
  - basic cell unit of system
- When neuron stimulated, electrical signal passes down axon of nerve



Stimulus creates electrical activity that travels along axon to next neuron





- Space exists between neurons
- Gap = synapse

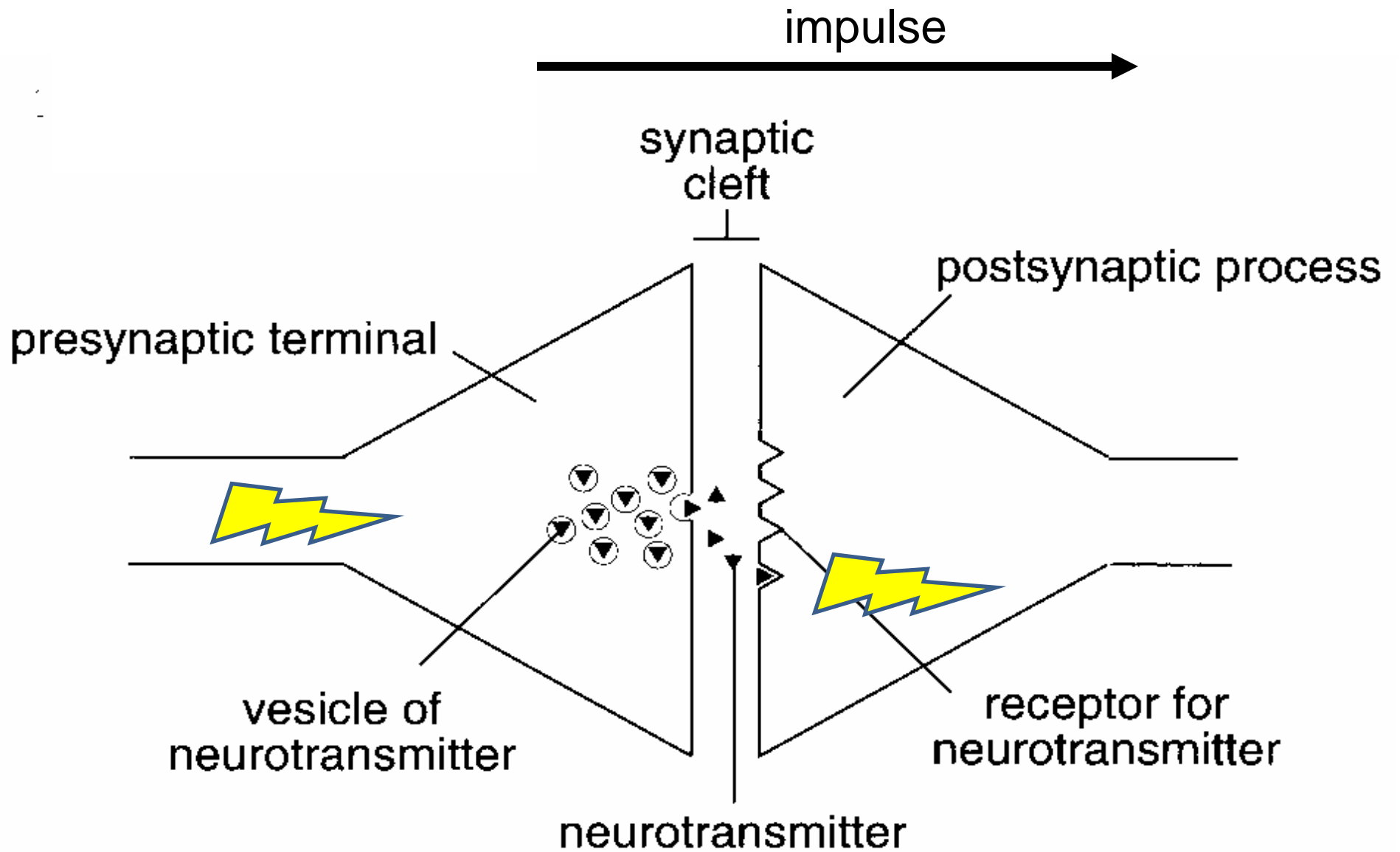
# Synapse

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- Transmission across synapse is chemical
- Signal arrives>>>chemical released across synapse is called “neurotransmitter”
- Neurotransmitter travels to next neuron, next neuron stimulated




# Events at the synapse



# Synapse

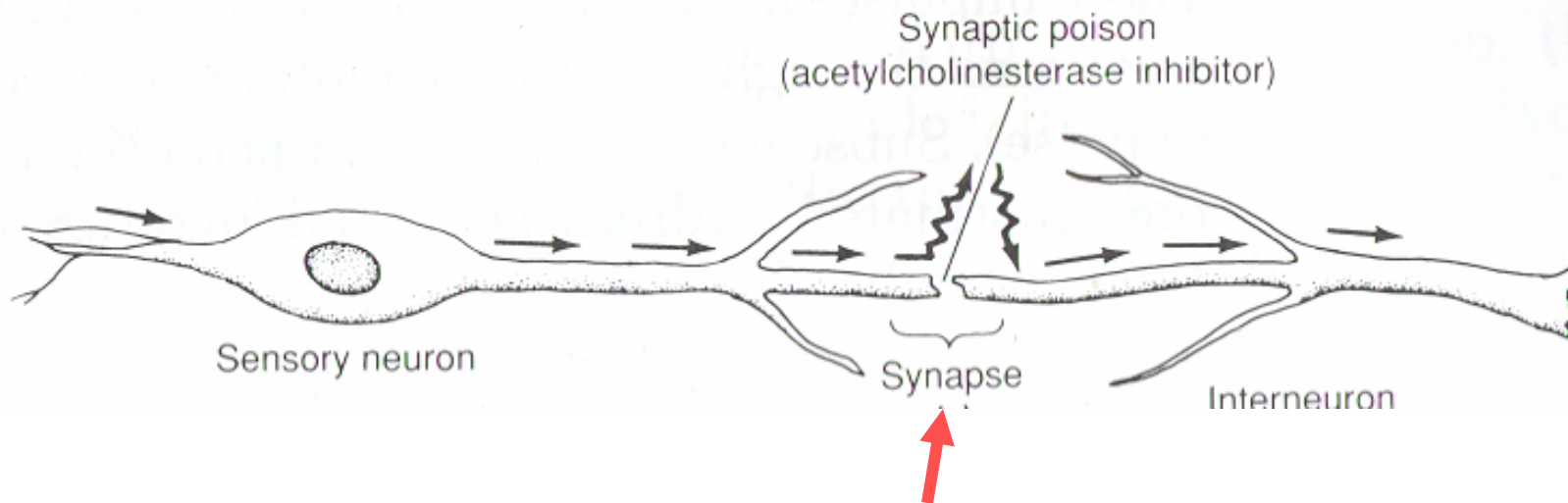
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- One neurotransmitter is acetylcholine
- Once job is done, acetylcholine needs to be broken down

Acetylcholine  Acetic acid +  
choline

Enzyme **cholinesterase**, breaks down acetylcholine  
**Stops signal**

# Synaptic poisons



Organophosphates, carbamates, poison system by acting at the synapse

# Mode of action against insects

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- Inhibit breakdown of neurotransmitter
- Bind up breakdown enzyme, cholinesterase
- Neurons continue to fire
- Nerves continuously send message to muscles
  - Twitching
  - Convulsions
  - Seizures



# Why some insecticides are a health risk

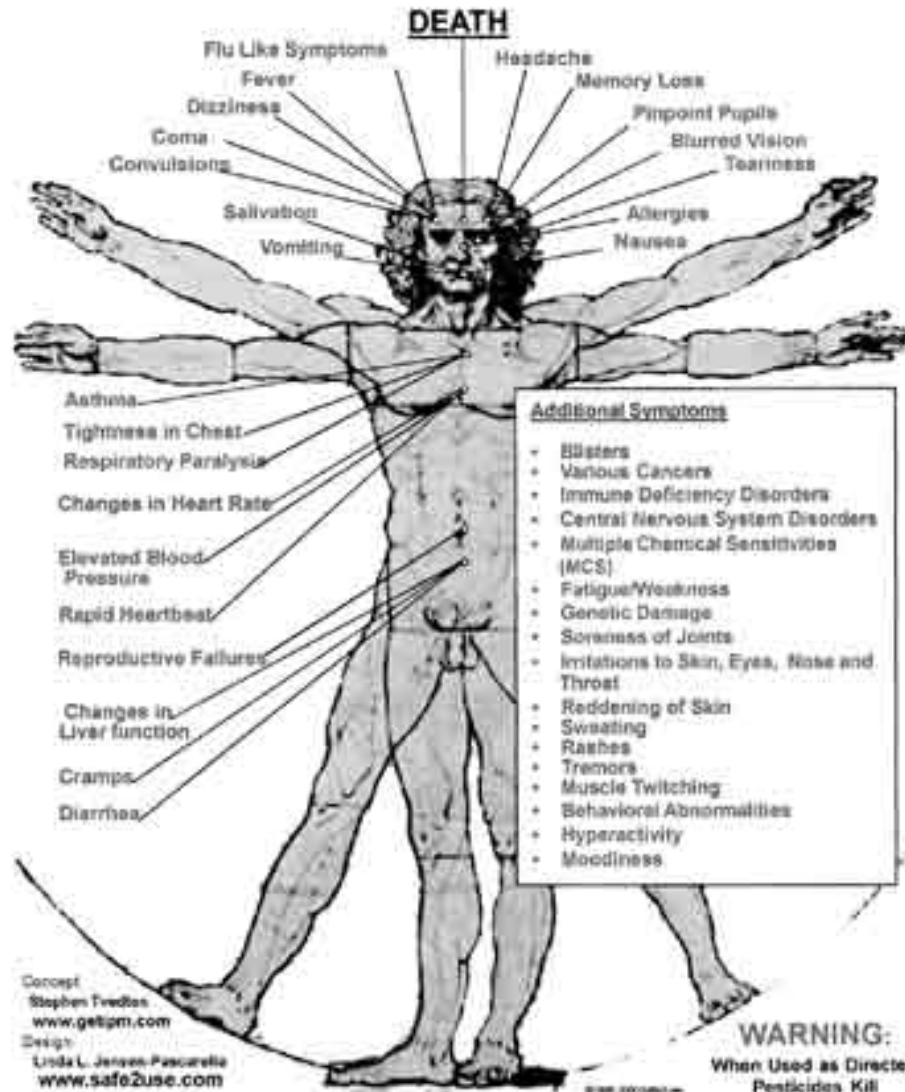
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- Organophosphates
  - Diazinon
  - Lorsban
  - Orthene
  - Imidan
- Carbamates
  - Sevin
- Organophosphate poisoning not reversible
- Body must manufacture new cholinesterase
- Carbamates somewhat reversible; some enzyme recovered



# Symptoms of Pesticide Poisoning

Sources: National Cancer Institute, National Academy of Science,  
Office of Technology Assessment, EPA, OSHA



Concept  
Stephen Twedden  
[www.getipm.com](http://www.getipm.com)

Design  
Linda L. Jensen-Pascarella  
[www.safe2use.com](http://www.safe2use.com)

**WARNING:**  
When Used as Directed  
Pesticides Kill

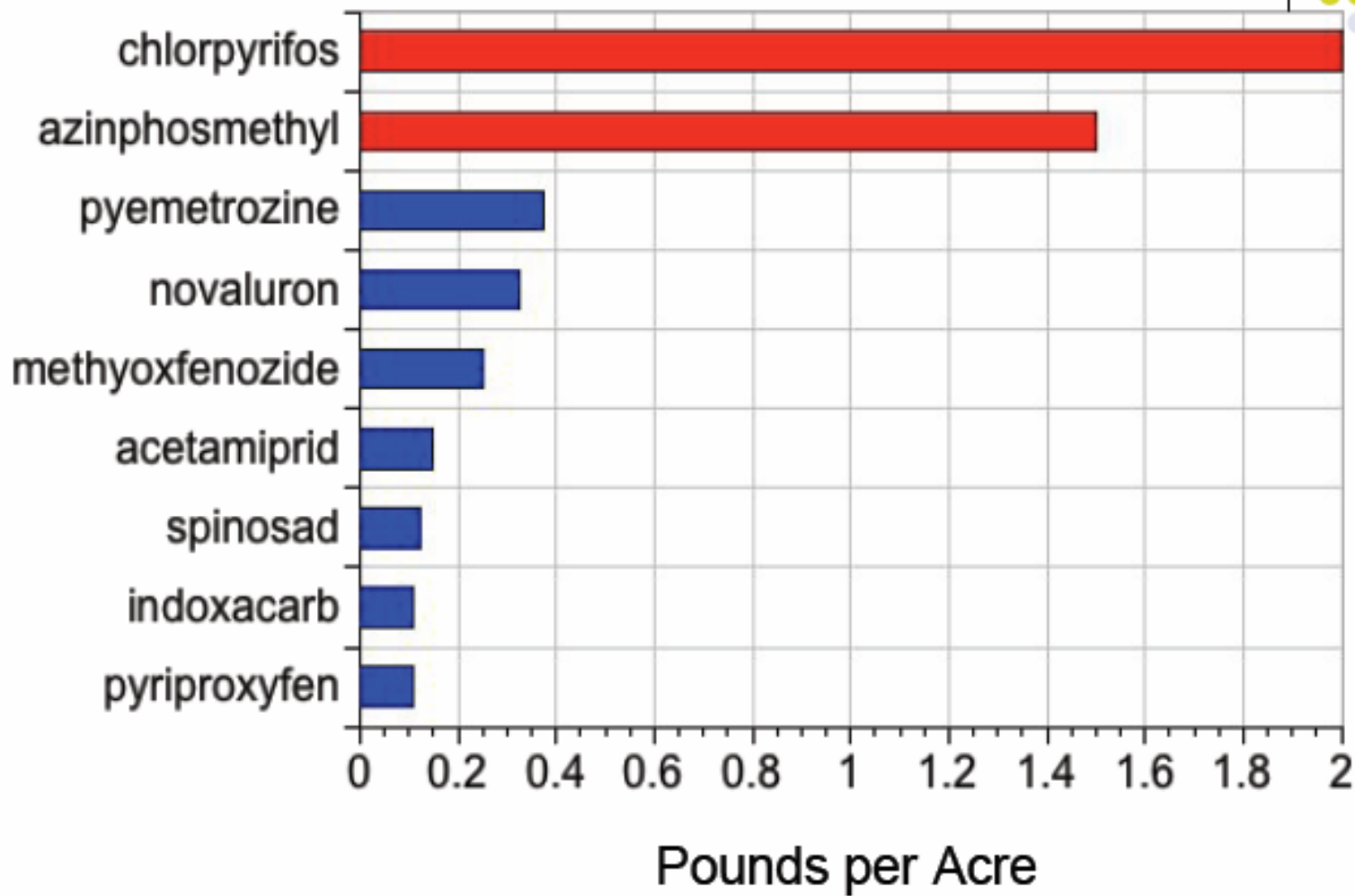


# Overview, new compounds

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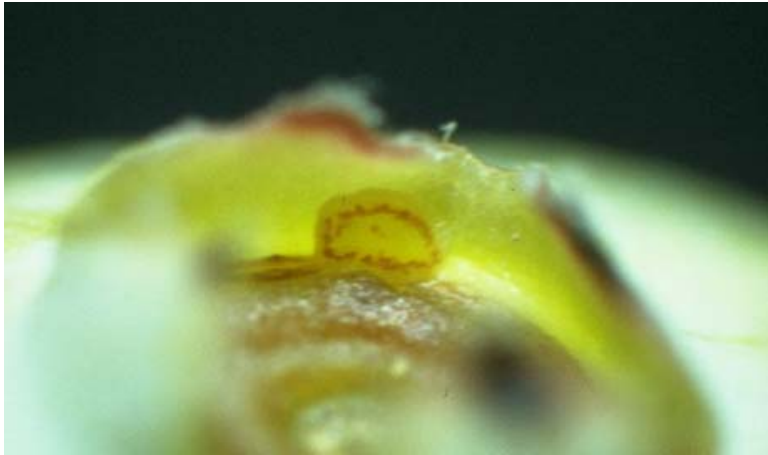
- Different chemistries!
- Usually reduced risk
  - Very low mammalian toxicity
  - Low ecotoxicity
  - Selectivity
- Often good residual, often systemic
  - Low application rate

## Application Rate of Reduced Risk Insecticides



# ACTIVITY!

Screening  
program:cranberry  
fruitworm and new  
insecticides





Worked at EB abandoned bed with  
mega-infestation of fruitworm,  
YHFW





# Control



# Field trial: methods

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- 5 replicates/treatment
- 150 gal/A with CO<sub>2</sub> backpack sprayer
- Two applications
  - 7/10 and 7/19
- Fruit randomly sampled after second spray





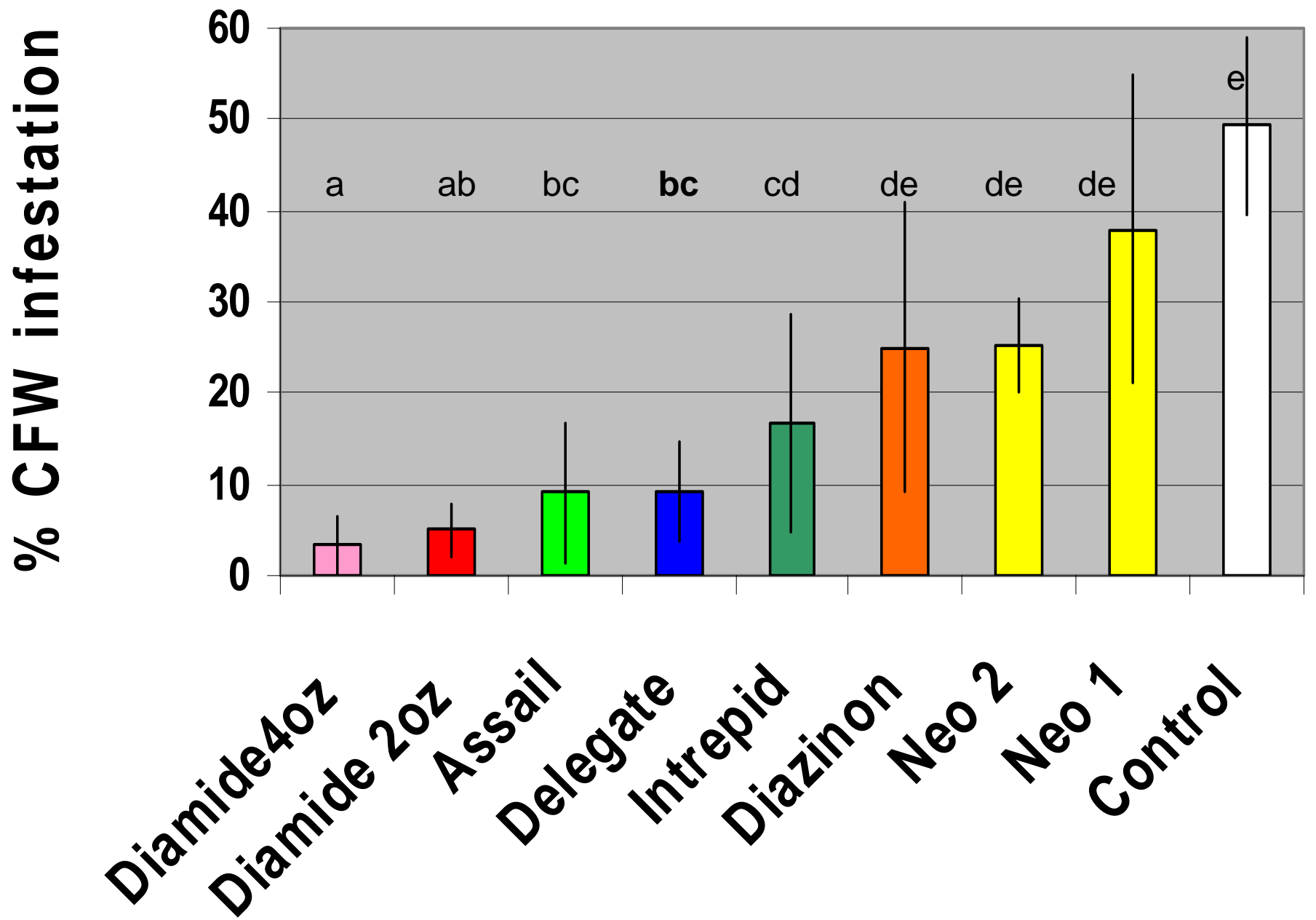
First spray: timing based on out-of-bloom counts  
Second spray: 9 days later



# Treatments

- 3 neonicotinoids
  - **Assail, Neo 1, Neo 2**
- Molting hormone mimic
  - **Intrepid**
- 1 anthranilic diamide
  - 2 rates: **Diamide 2 oz** and **Diamide 4 oz**
- New spinosyn
  - **Delegate**
- Industry standard
  - **Diazinon**
- Control--untreated







**Delegate**

**Diamide  
4 oz**

**Intrepid**

**Diamide  
2 oz**

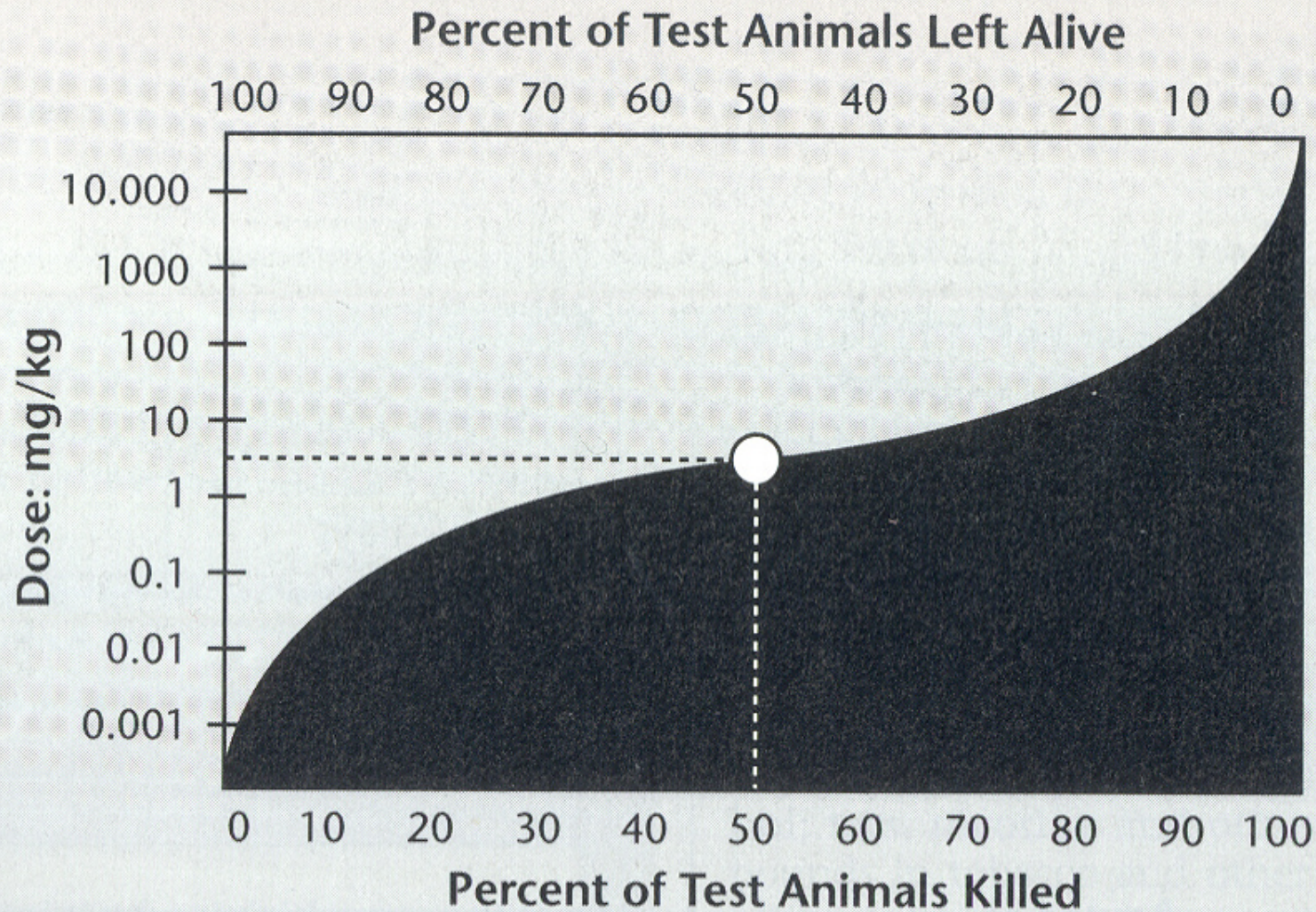
**Diazinon**

# New insecticides in cranberry

– Tipworm compound	2012	
– HGW (2 <sup>nd</sup> generation)	2012	Diamide
– Zone II Neonic	2011	Neonicotinoid
– E2Y (1 <sup>st</sup> generation)	2011	Diamide
– Actara +type	2010	Neonicotinoid
– Assail (acetamiprid)	2008	Neonicotinoid
– Delegate (spinetoram)		2008 Spinosyn
– Avaunt (indoxacarb)	2007	
– Actara (thiamethoxam)		2005
		<b>Neonicotinoid</b>
– Intrepid (methoxyfenozide)	2004	IGR
– Admire (imidacloprid)	2004	Neonicotinoid
– Spintor (spinosad)	2002	Spinosyn
– Nexter, Pyramite (pyridaben)	2001	



## Measuring the Acute Toxicity of Pesticides



Dose/response tests: lethal dose that kills 50% is  $LD_{50}$

# HUMAN TOXICITY

new vs old

neonicotinoid →

IGR →

spinosyn →

Formulation	Oral LD50 (mg/kg)	Dermal LD50
Guthion	4.4	155
Lorsban	223	222
Assail	1064	>2000
Advion	1277	>5000
Esteem	4253	>2000
Intrepid	>5000	>2000
Rimon	>5000	>2000
Fulfil,	>5000	>2000
Success	>5000	>2000

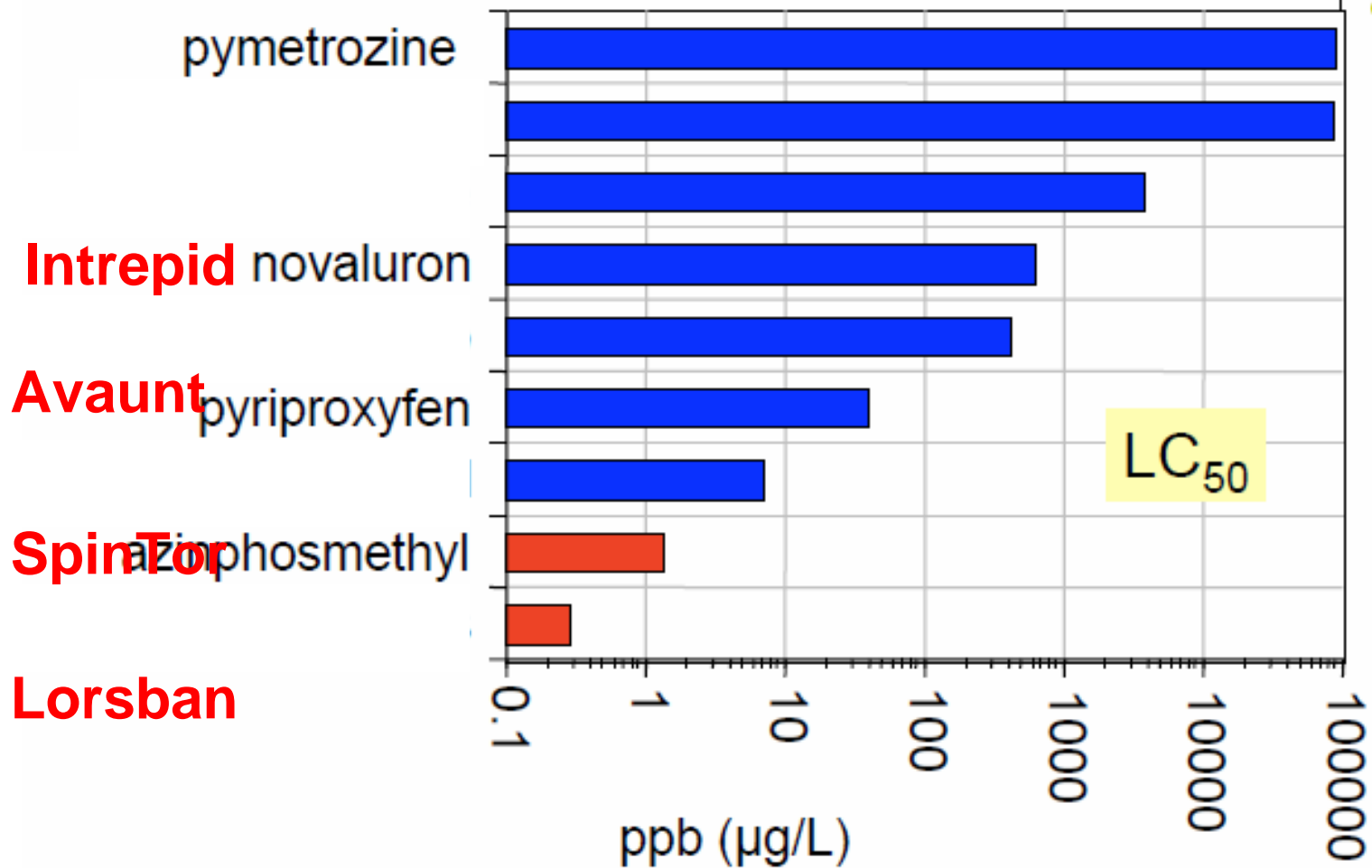




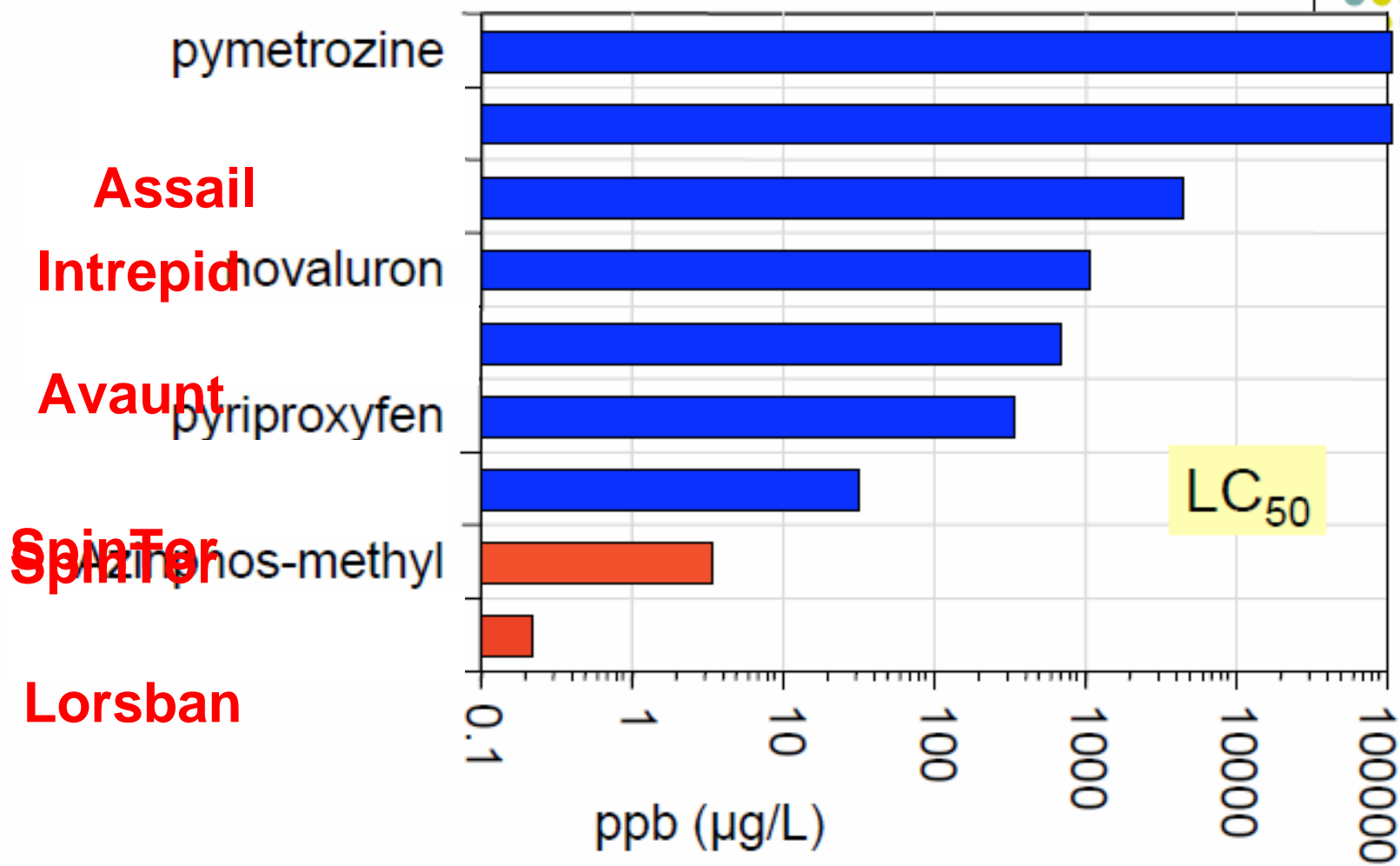
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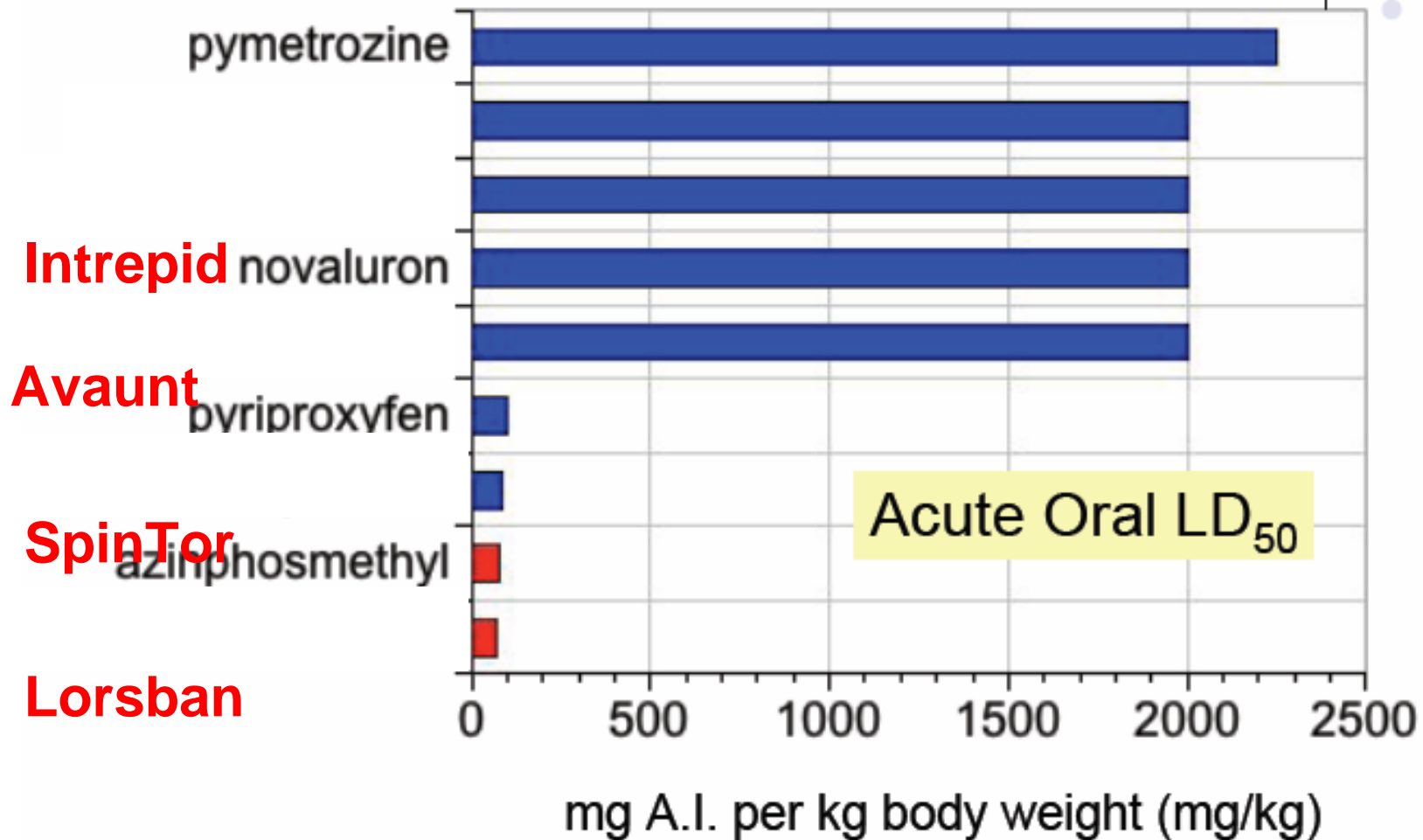
# Toxicity of Reduced Risk Insecticides to *Daphnia*



# Toxicity of New Insecticides to Fish (Rainbow Trout)



# Toxicity of Reduced Risk Insecticides to Birds



# New insecticides in cranberry

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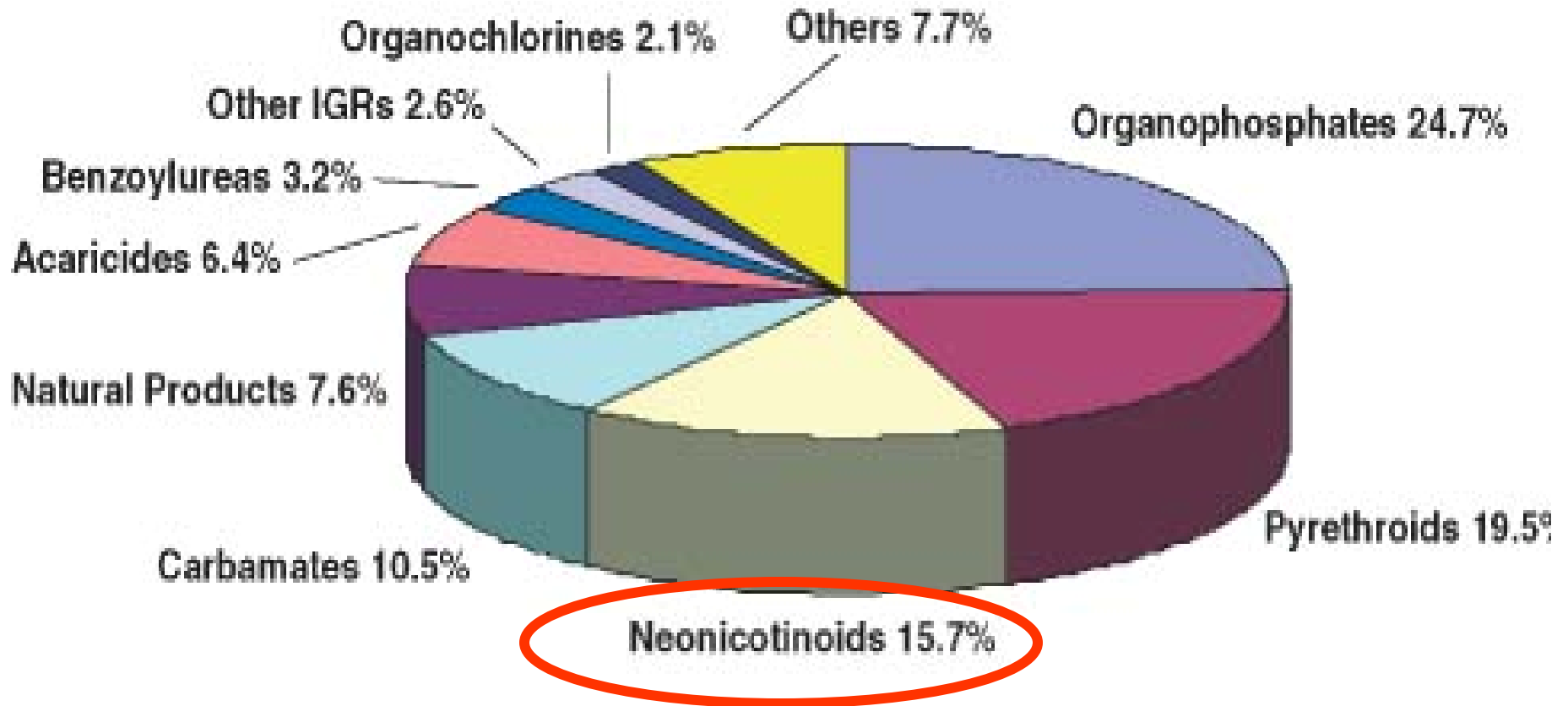
# Neonicotinoids

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- Now labelled in cranberry
  - Actara (thiamethoxam)
  - Admire (imidacloprid)
  - Assail (acetamiprid)

# Neonicotinoids

- Most important new class in 30 years



**Total Global Insecticide Sales 2003 = \$6,650**

[Source Phillips McDougall, November 2004]



# Acute contact LD<sub>50</sub>: some neonicotinoids are very toxic to bees

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compound	µg/bee
Sevin	1.3
Guthion	0.063
Admire (neonic)	0.024

worse

# Not all neonicotinoids are as toxic: acute contact LD<sub>50</sub>

clothianidin	14 ng	Poncho
imidacloprid	18 ng	Admire, Provado, Gaucho
thiamethoxam	30 ng	Actara, Platinum
dinotefuran	75ng	Venom
acetamiprid	7.1 μg	Assail
thiacloprid	14. 6 μg	Calypso

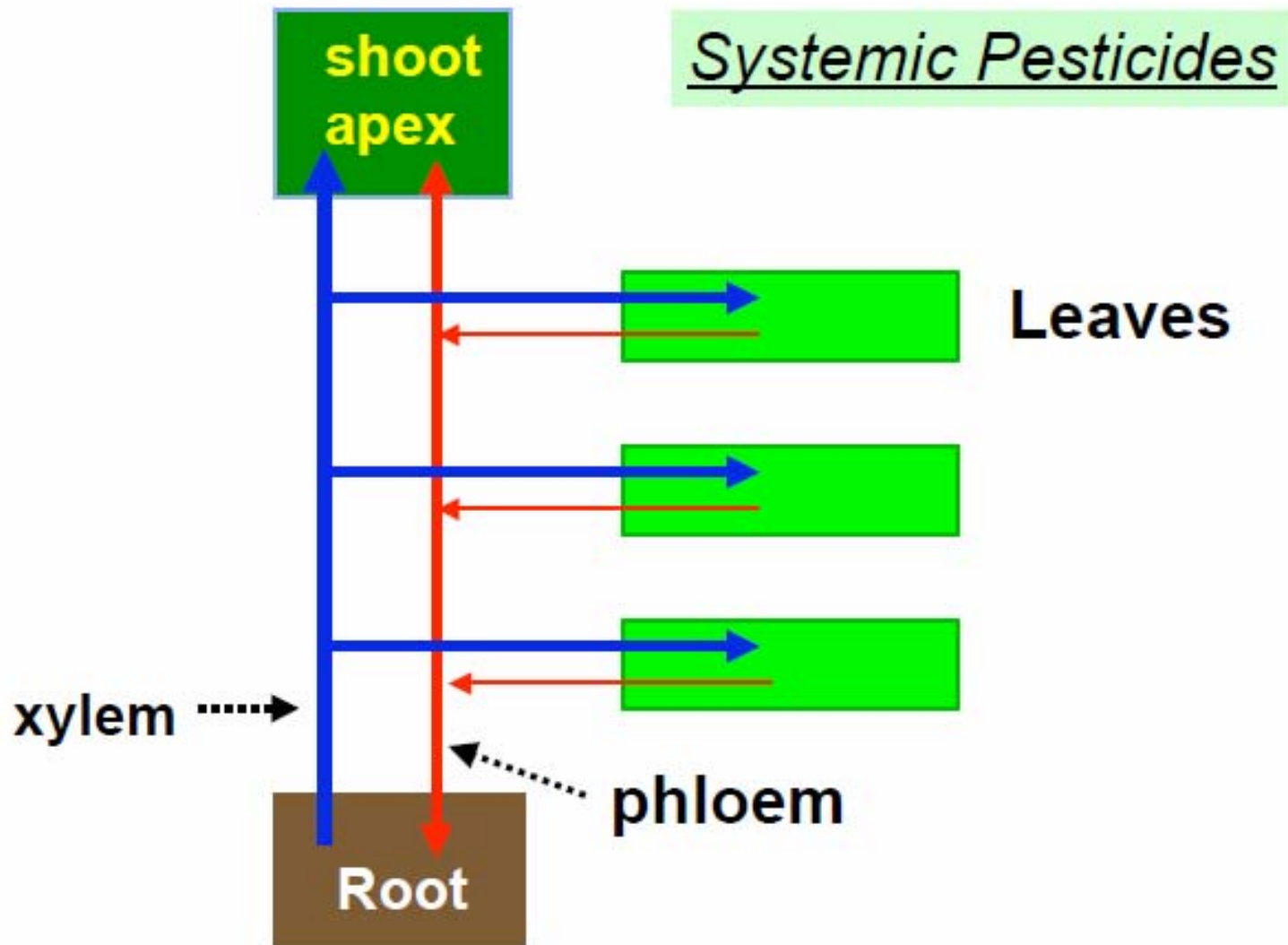


better

# Sublethal effects: pollen and nectar

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- Neonicotinoids are moderately water soluble and systemic
- Carried by the vascular system
  - Enter **xylem** (water transport), rapid movement into leaves
  - to flowers, via **phloem** – very low levels
    - ppb range



Once pesticides diffuse through the plant cuticle or into the roots, they can be carried by the vascular system to different regions

# Uptake in plants>>>pollen

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- Imidacloprid levels detected in other pollen: watermelon, corn, canola
- Registrant rule of thumb: < 5ppb expected in general
- Very, very low levels







Neonicotinoids implicated in Europe's bee problems,  
some  
compounds/use patterns banned, research base out  
of European labs until recently, role in US CCD?

# Sub-lethal effects on honey bee

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- Physiological or behavioral effects on individuals that survive an exposure to a pesticide
- Could impact worker bee functioning
  - Olfactory learning performance
  - Decreased foraging and activity at hive entrance
- Need to interface amount exposed to in lab with realistic field assays

# Contamination at colony

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- Contaminated pollen and nectar
  - Eaten by worker bees and larvae

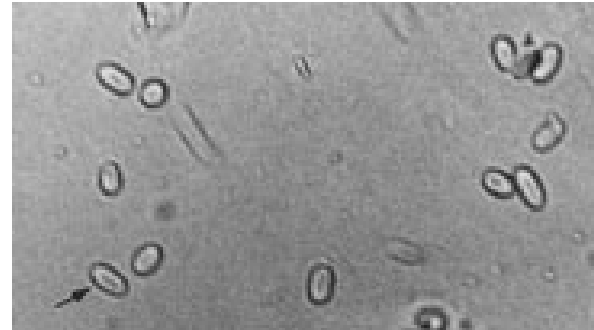




# Interactions: pathogen + neonicotinoid

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- J. Pettis and cooperators
- *Nosema apis*--microsporidian
- most widespread of the adult honey bee diseases; big problem
- infects ventriculus thereby causing dysentery





# Interactions: pathogen + neonicotinoid

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- Larvae exposed to imidacloprid for 2 months (in pollen at ppb level)
- Then challenged adults with *Nosema*
- Imidacloprid-exposed bees
  - Higher spore infection levels (>3x) vs. control



# Colony Collapse Disorder Status



# Colony Collapse Disorder Status

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There is growing evidence that a number of factors, including pathogens, pesticides, mites and other stress factors such as poor nutrition are most likely involved in the overall declining health of honeybee colonies in the US.

*June 2008 American Bee Journal*

# Colony Collapse Disorder Status

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- Seems to be adequate pollination
  - Migratory beekeepers are compensating
    - adding new queens
    - cleaning hives
    - not reusing combs
  - Keep up to date as to beekeeper concerns
- 