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Insecticide Failures? Let’s Talk Resistance…

Ian MacRae
Dept of Entomology
University of Minnesota

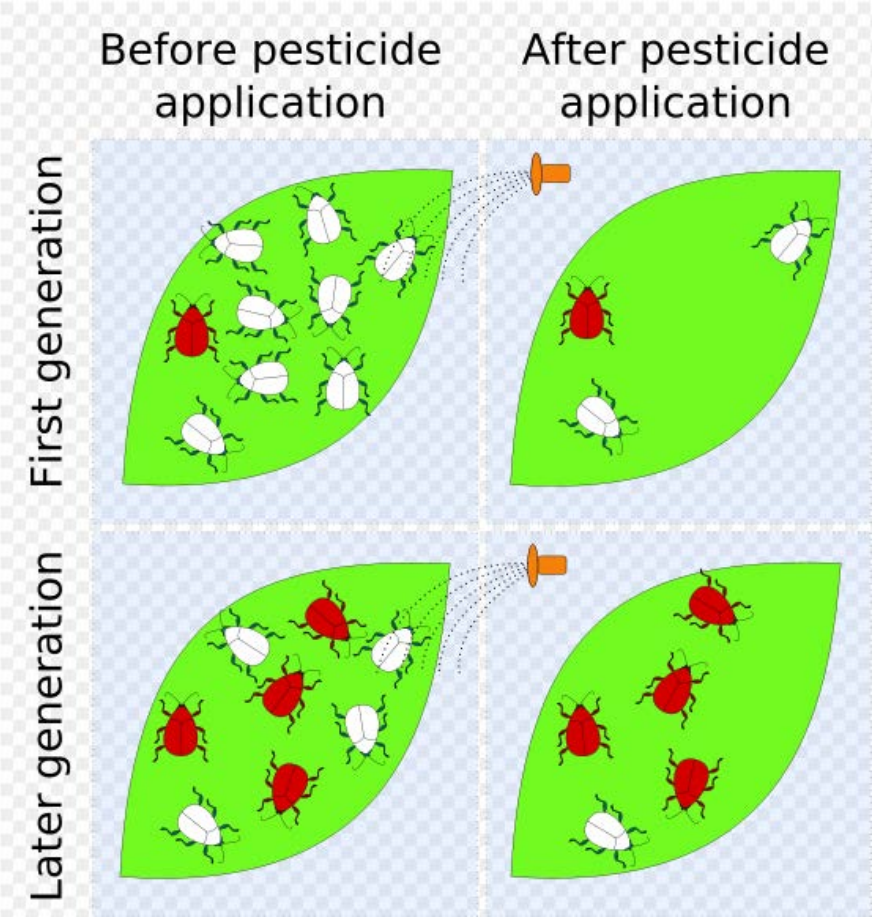
Insecticide didn't work? Hitting it again didn't work? Doubling the rate didn't work?

- Must be resistance, right?
- Product failures aren't always ***resistance***
  - ***Clean the tank? Store the product correctly? Old product? Climate?***
- How to know it's resistance..

Genetic nature of resistance

- Resistance is ‘pre-adaptive’
  - It results from genes already in the population
  - Sounds counter-intuitive
- A genetic trait in some individuals in a population allows them to be less susceptible to a pesticide
  - Gene is usually a recessive & rare allele (~1/100,000 to 1/10 M individuals have it)
  - Why so rare? May be a ‘cost-to-fitness’ in the absence of the selection pressure

Development of resistance



http://en.wikipedia.org/wiki/Pesticide_resistance

- Resistant genes may be rare but if you apply selective pressure against the susceptible genotypes (insecticide applications) they're what's left to reproduce
- Some susceptible individuals will be left (poor application, immigrants, etc) so some offspring heterozygotes
- Successive applications (esp. in the same generation) will continue to select against susceptible genotypoes. Homozygotic resistant genotypes will make increasingly large increase proportion of population

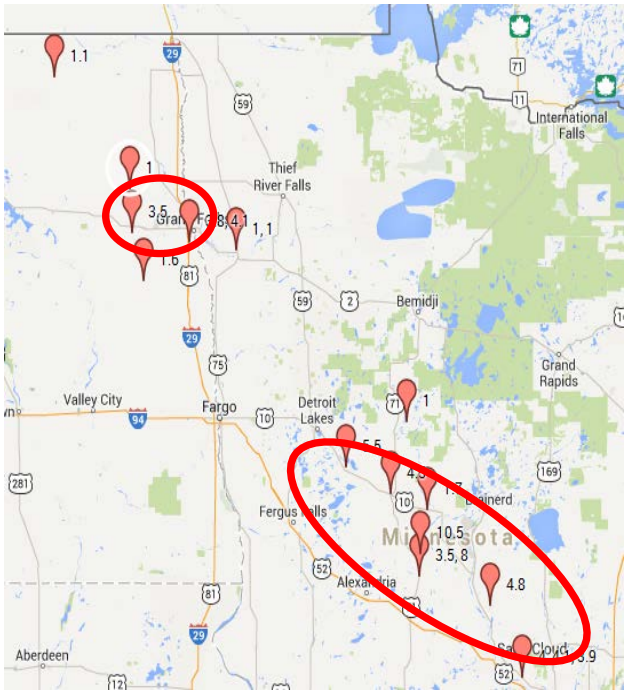
Selection pressure

- Over reliance on one chemistry
- Repeated applications same / subsequent generations (i.e. same season)
- Using same mode of action
  - It’s about more than just the name…

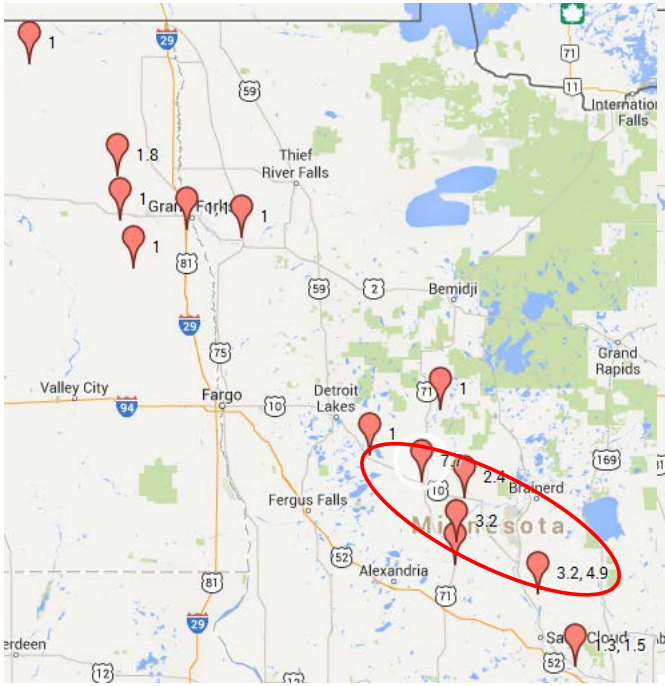
Seed treatments (insecticide)

- Popular
  - Neonicotinoids
    - Anthranilic diamides available but not yet widely used
  - Convenient
  - Seen as insurance…
  - Effective? (I'd argue not in all cases…)
  - Poor resistance management and leads to loss of product? ***YUP, it can***!

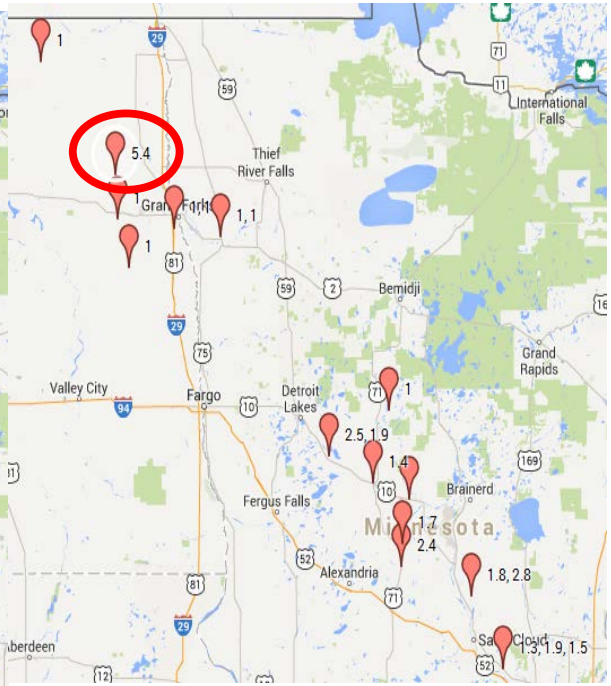
Imidacloprid



Clothianidin



Thiomethoxam



- Patterns of neonicotinoid insecticide resistance in MN/ND

Prophylactic treatments

- Popular
  - ‘Just in case…’, ‘clean up the field’, ‘decrease future applications’
  - Convenient
  - Seen as insurance…
  - Effective? (again, I’d argue not in all …)
  - Poor resistance management and leads to loss of product? ***YUP, it can***!
    - sometimes results in unexpected problems…

TSSM resistance in MN, 2012

	Susceptibility (LC50)		Comparative Resistance Rate
TEST	**CO (Susceptible)**	**MN (Suspected Resistant)**	**MN/CO**
Residual test (Vial Test)	**3.21** Lower Limit = 0.10 Upper Limit = 6.32	**34.64** Lower Limit = 17.28 Upper Limit = 52.19	**10.81**
Exposure Test (Dip Test)	**3.62** Lower Limit = 1.72 Upper Limit = 5.52	**18.46** Lower Limit = 7.95 Upper Limit = 28.97	**5.10**

2013 Data

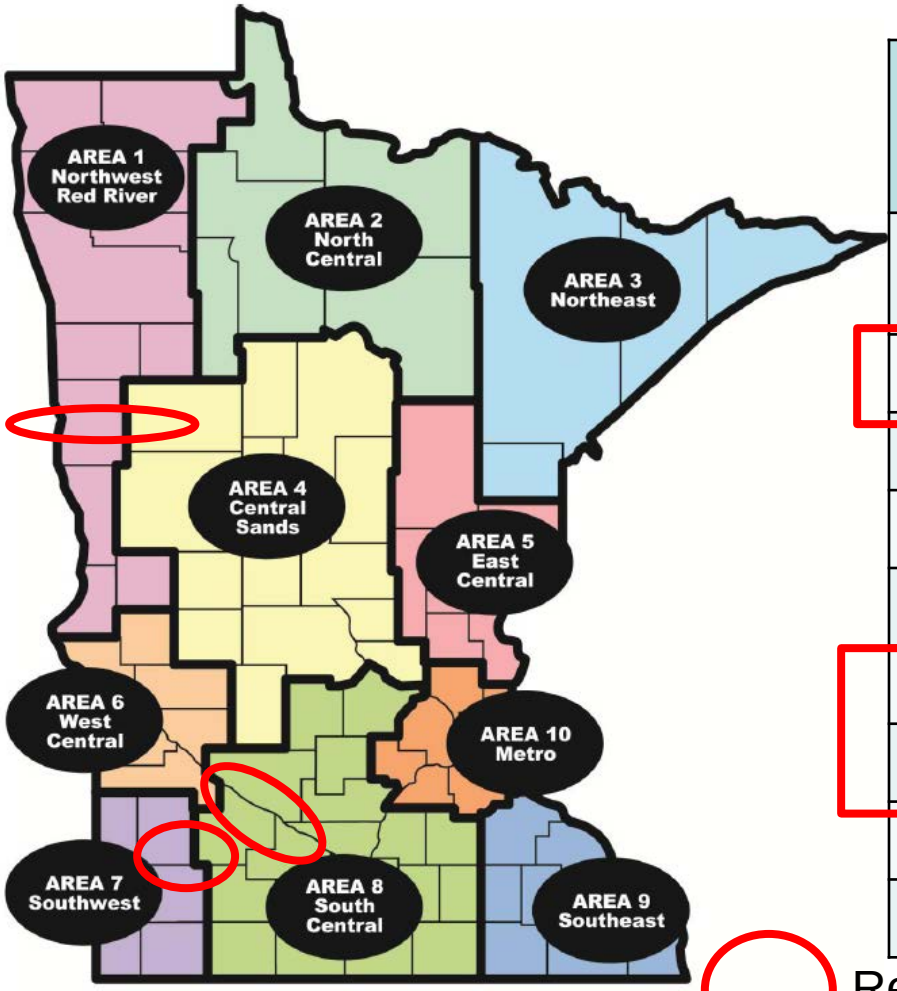
- All 2013 trials done with vial method
- 2013 data showed similar results to 2012
  - Chlorpyrifos still effective in most areas
  - Exceptions where it was used for soybean aphids (especially prophyplactic applications)

Level of Resistance	Resistance Ratio
Susceptible	0X – 3X
Minor resistance	3X – 5X
Low resistance	5X – 10X
Medium resistance	10X – 40X
High resistance	40X – 160X
Extremely high resistance	>160X

Location	Resistance Ratio
Hawley	**3.85***
Georgetown	**1****
Crookston	**1****
Dillworth	**1.95**
Foxhome	**3.5**
Rice	**1****
Fergus Falls	**1****

*Calculated after removal of outlier data in 0 an 0.5 rates. Outliers had high rates of mortality (100% after 24h), probably as a result of handling
**Any ratio under 1:1 was reported as 1 to indicate susceptibility of that population.

Chlorpyrifos application in MN



PMA	% crop area applied	Avg no. appl/yr	Avg rate/appl.	Avg rate/yr	Total lbs a.i. applied in PMA
State	26%	1	.45	.45	65.5K (avg/8.2K lbs/PMA)
PMA1	23%	1	.45	.45	18.6K
PMA4	26%	1.1	.45	.45	3.6K
PMA5	23%	1	.39	.39	538
PMA6	28%	1	.41	.41	8.3K
PMA7	31%	1	.53	.54	10.3K
PMA8	30%	1	.44	.44	21.8K
PMA9	9%	1.1	.56	.59	1.6K
PMA10	21	1	.35	.35	913

Reports of Chlorpyrifos failure 2012

Double the rate

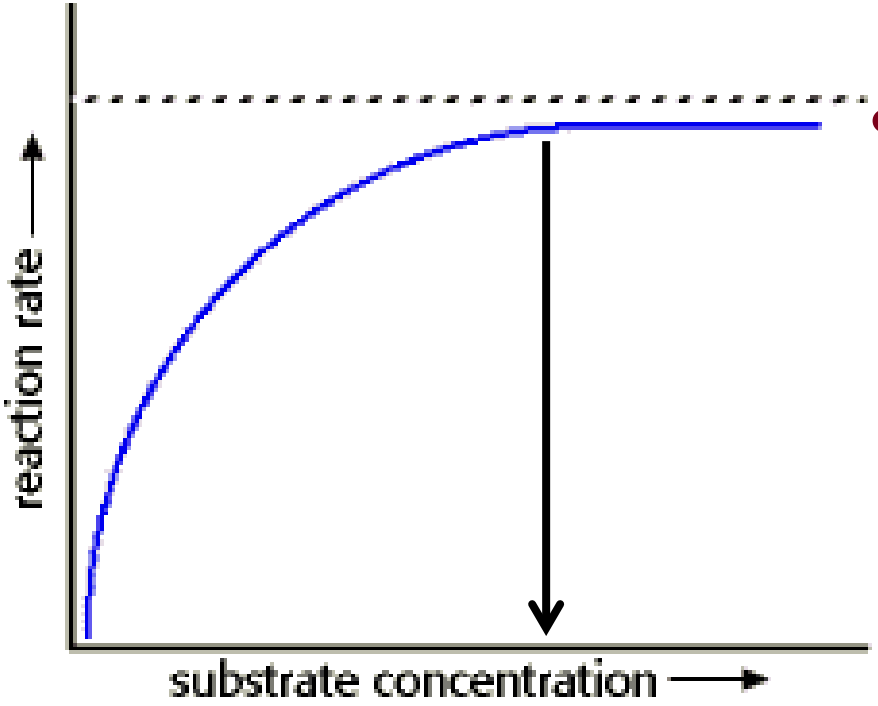
- Sorry, I can’t think of a single thing that’d sell this idea to anybody….
  - Poor resistance management and leads to loss of product? ***YUP, you betcha***!

Fastest car in the world

- 269.86 mph
- ~1200 bhp quad turbo-charged W-16
  - takes 280 bhp to get to 155mph
  - Takes remaining 920 bhp to get the last 100mph

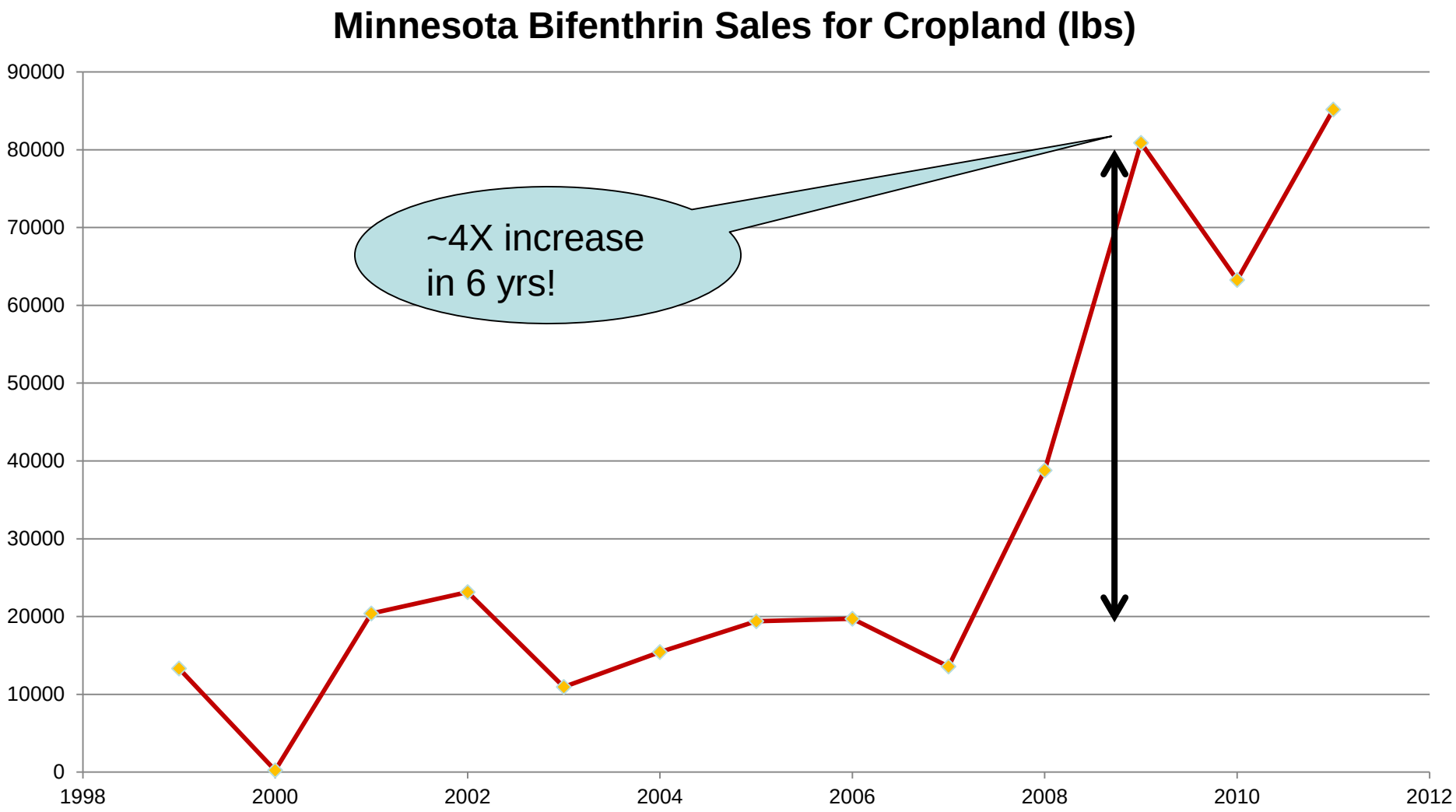
Yes, I know the Hennessy Venom GT did 270.49mph (0.63mph faster) at the Kennedy Space Center in 2014, but only in 1 direction so it didn't make Guinness WBR….

Law of diminishing returns

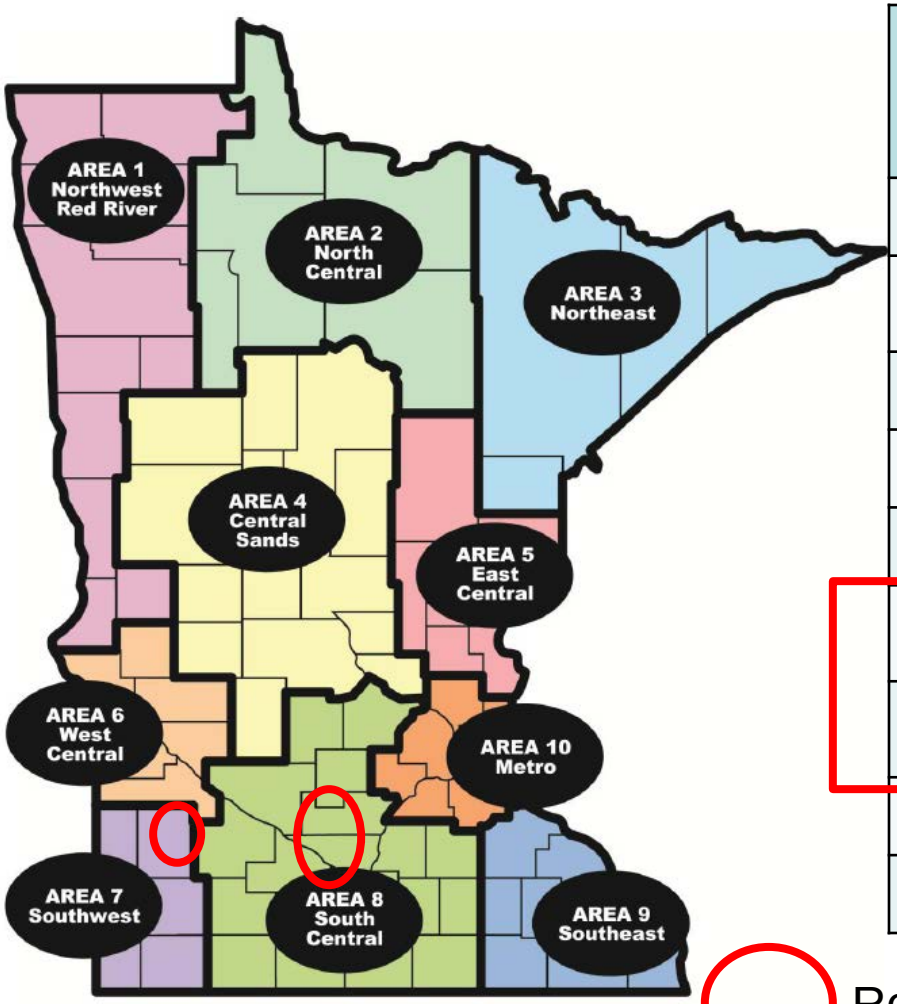


- Takes more and more input to get same amount of output
  - Same with many chemical Rx
- Insecticides formulated to get max return from rate used, doubling label rate will not double no. killed
  - efficacy maxed AMAP at label rate)

Bifenthrin use (sales) in MN



Bifenthrin application in MN

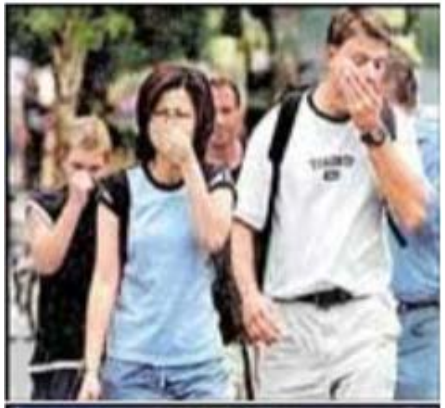


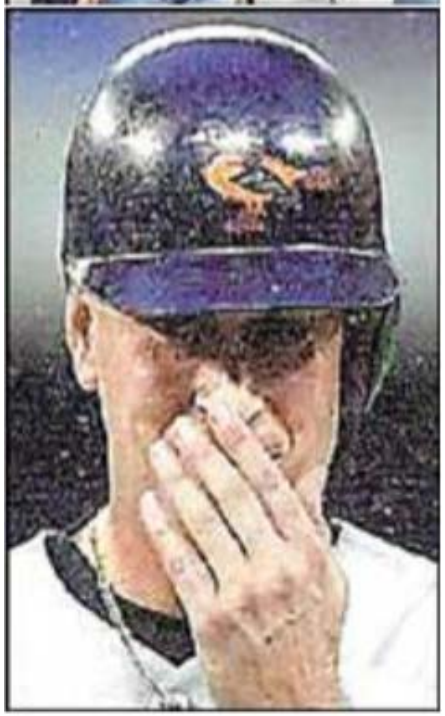
PMA	% crop area applied '09 '11	Avg no. appl/yr '09 '11	Avg rate/appl. '09 '11	Avg rate/yr '09 '11	Total lbs a.i. applied '09 '11
State	7% 4%	1 1	.08 .06	.08 .06	502 1567
PMA1	0 2%	0 1.1	0 .06	0 .07	0 369
PMA4	2% 4%	1 1	.07 .06	.07 .06	47 121
PMA5	0	0	0	0	0
PMA6	0 6%	0 1.1	0 .05	0 .05	0 197
PMA7	2% 9%	1 1	.09 .07	.09 .07	122 328
PMA8	3% 5%	1 1	.08 .07	.08 .07	333 552
PMA9	0	0	0	0	0
PMA10	0	0	0	0	0

Reports of Bifenthrin failure 2015

And aphids move…

- Your aphids aren't just ***your*** aphids…
- Soybean aphids move, *a lot!*
- 200, southern Ontario invaded by soybean aphid
  - One massive flight
  - Remember the Sturgis Dispersal Event (B. Potter)



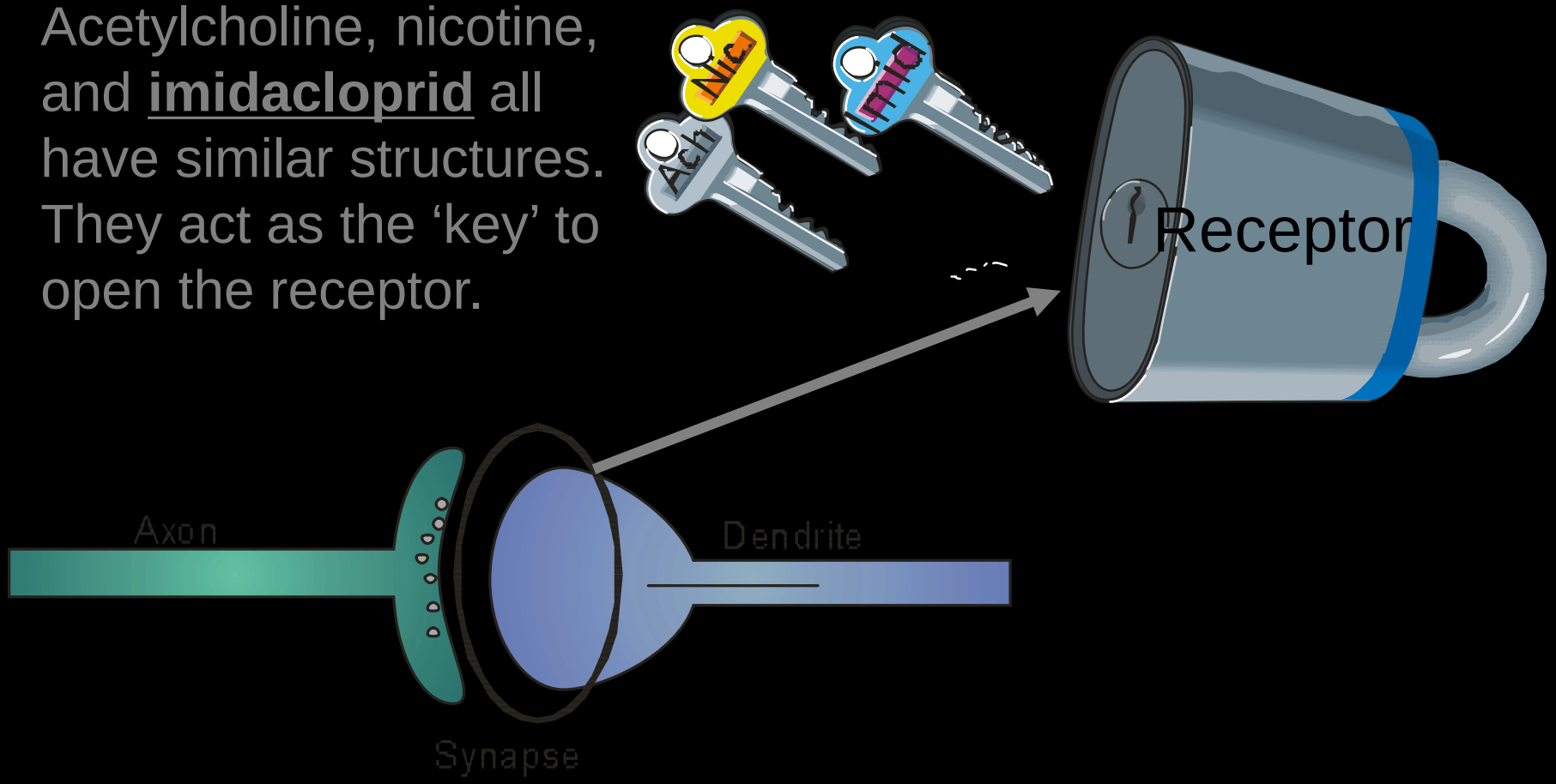


Insecticides - Nervous system

Acetylcholine Mimics

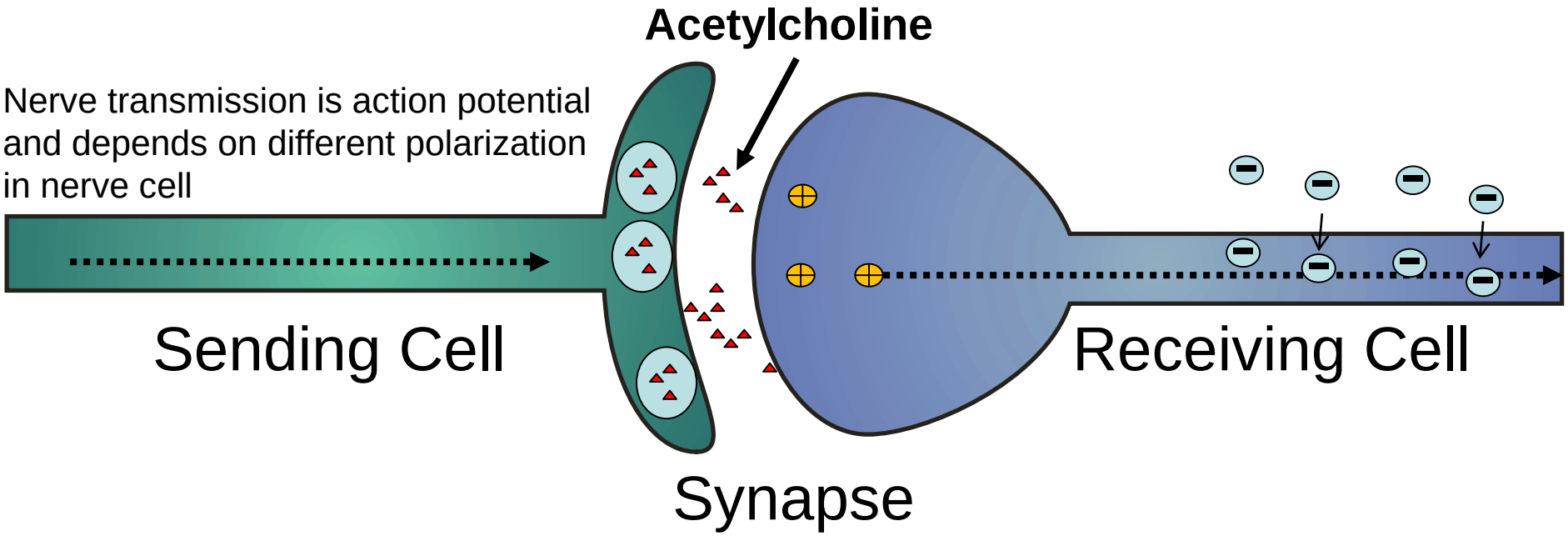
Mode of Action

Acetylcholine, nicotine, and **imidacloprid** all have similar structures. They act as the ‘key’ to open the receptor.



Insecticides – Neonicotinoids moa

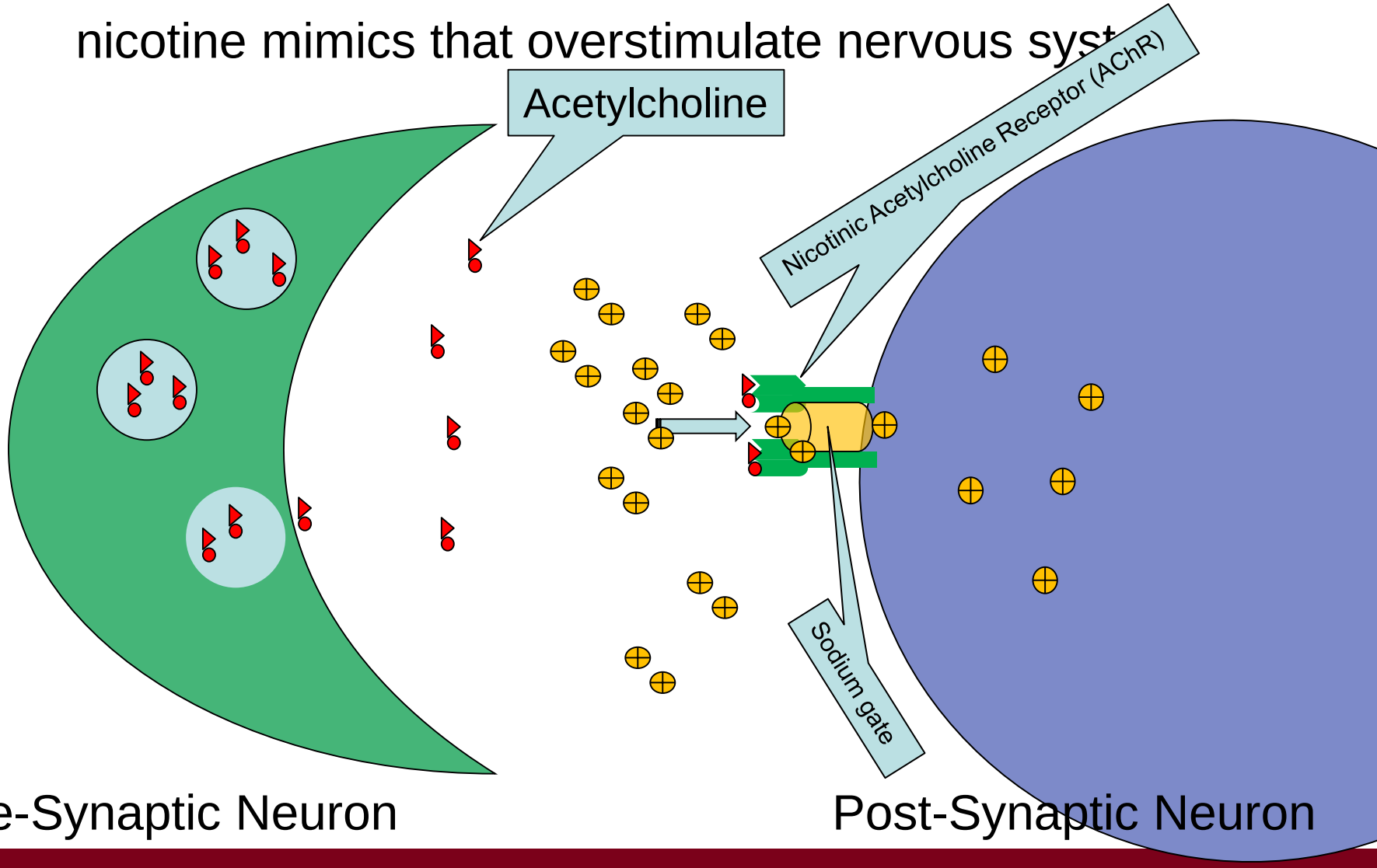
nicotine mimics that overstimulate nervous system



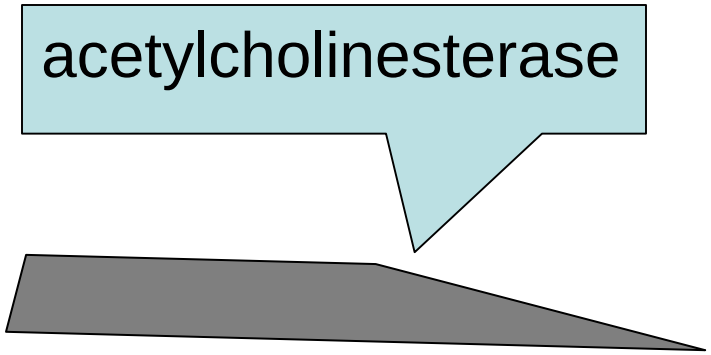
Acetylcholine binds to Nicotinic Acetylcholine Receptors (AChR) which trigger AChR ion channels to open, NA^+ enters post-synaptic neuron, polarizing nerve and initiating action potential.

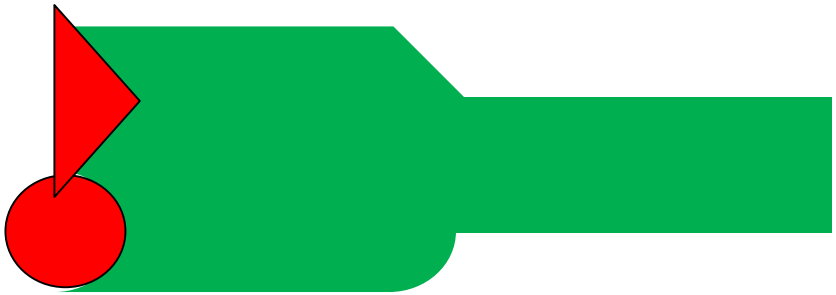
Insecticides – Neonicotinoids moa

nicotine mimics that overstimulate nervous syst



Post neural impulse transfer

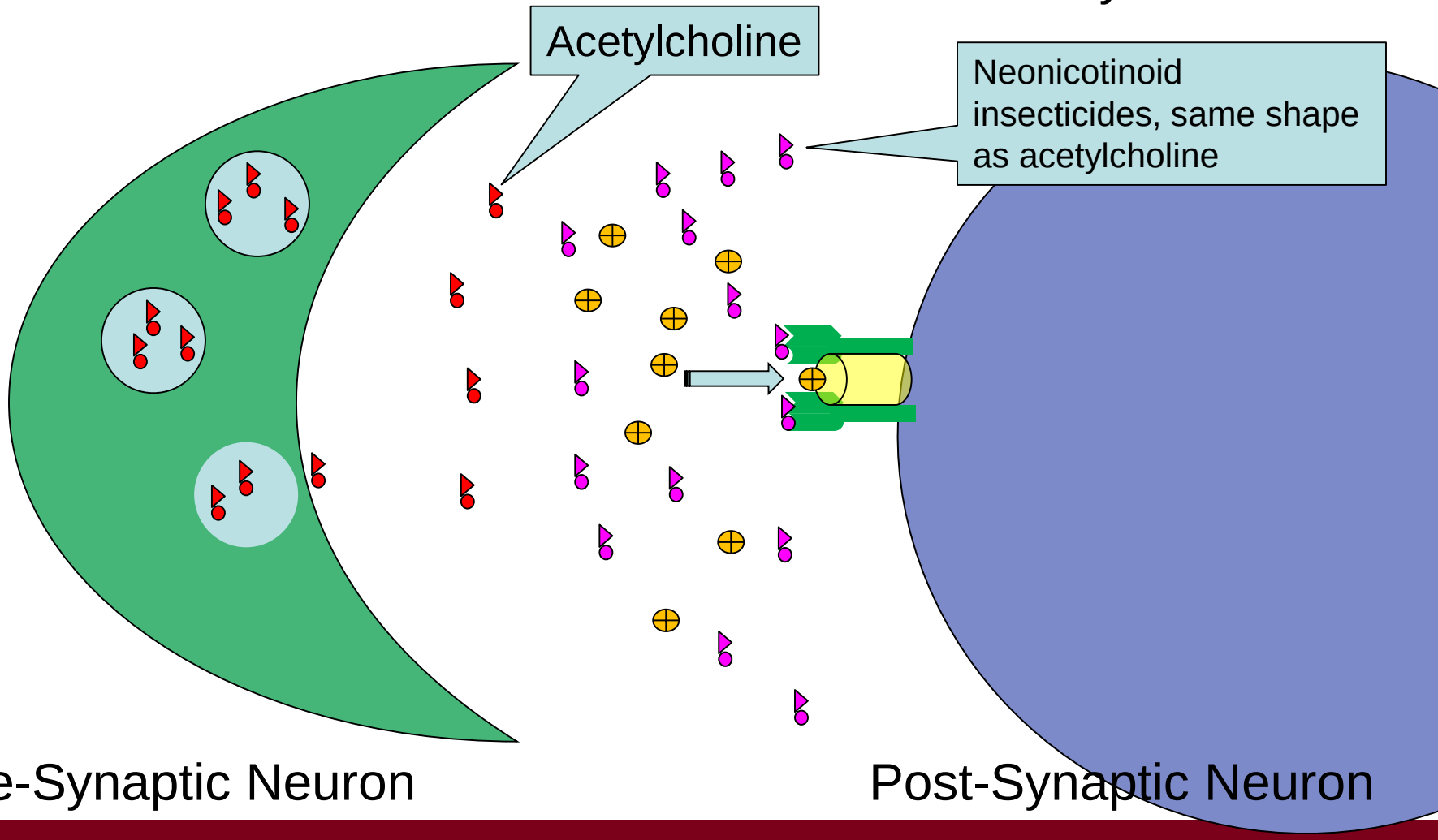




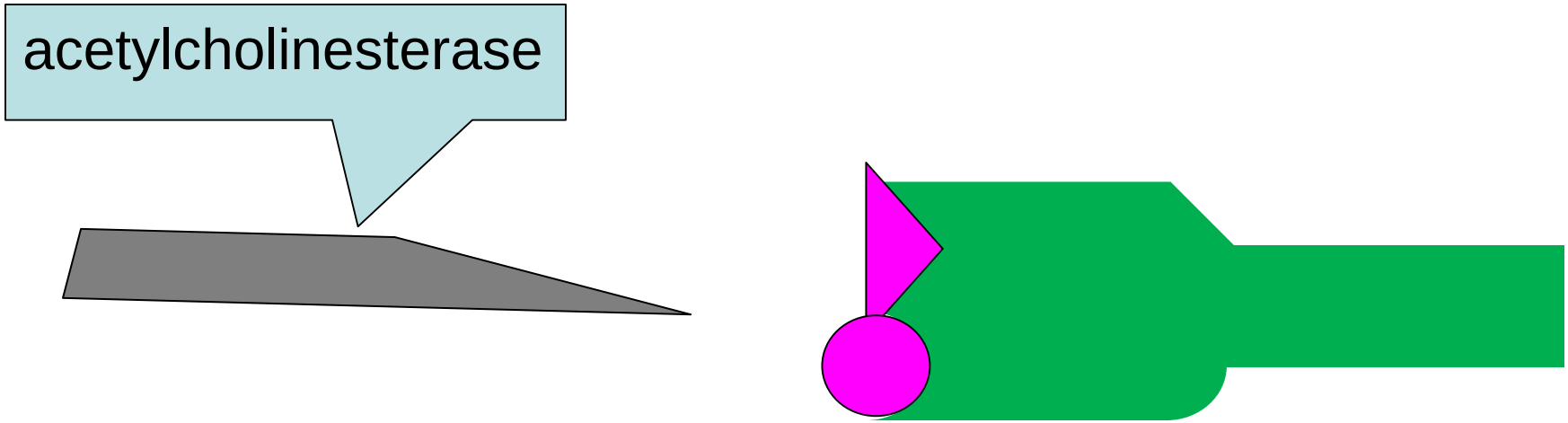
In normal nerve transmission, acetylcholinesterase splits acetylcholine into acetyl and choline groups freeing the Nicotinic Acetylcholine Receptors to receive another impulse

Insecticides – Neonicotinoids moa

nicotine mimics that overstimulate nervous system



Post-transfer



acetylcholinesterase can’t split neonicotinoid insecticides
so Nicotinic Acetylcholine receptors remain occupied,
NA+ gate stays open and nerve depolarizes (continuously sending nervous impulse
Imidacloprid, Clothianidin, etc work this way

Potential moa resistance (altered target site)

If a rare gene traits for a modified / different target site, insecticide can't bind to the site! Normal neural transmssions continue, insecticide doesn't work….
Just one type of resistance

Mechanisms of resistance

- Metabolic
  - Detoxification of toxin faster than susceptible insects (e.g. enzymatic)
  - Protein binding, doesn't reach target
  - Increased excretion rate
    - Insecticide not around long enough to act
- Altered target site
  - Site where toxin binds altered so toxin not as effective
    - Knockdown effect in houseflies – receptor site on nerve axon different 'shape'
- Penetration
  - Resistant insect may absorb insecticide more slowly, giving detoxification mechanisms nmore time to deal with it
- Behavioral
  - Insect's behavior may change to avoid insecticide exposure

Insecticide resistance management (IRM)

Standard Steps

- Detecting resistance
- Optimize non-chemical mortality (density dependent mortality: cultural control, biological control, pheromone confusion, etc)
- Reduce the selection pressure
  - Spray only when necessary (IPM)
  - Refuges
  - Rotate of Modes of Action (moa's)
    - More effective than tank mixing

Watch the mixed products that have only half rates of each ai

Tactics

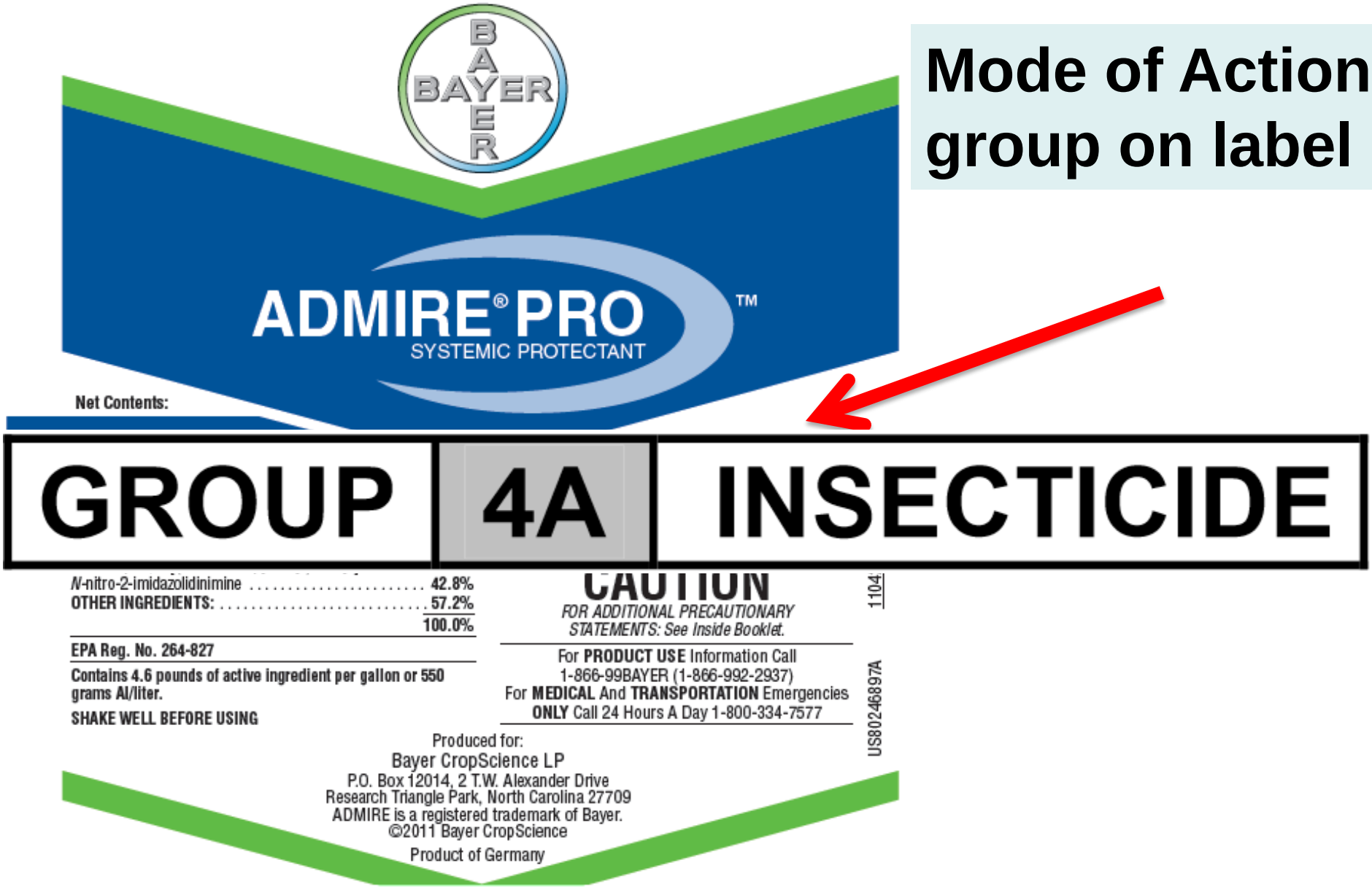
- **Don’t follow a seed treatment with a foliar of the same mode of action
- less frequent application or ai with brief persistence
- avoid slow-release formulations (tend to release at low rates)
- apply against adults after reproduction allowing susceptible genotypes to be passed on (esp if larvae/nymphs are damaging stage!)
- refugia are important
- utilize economic thresholds

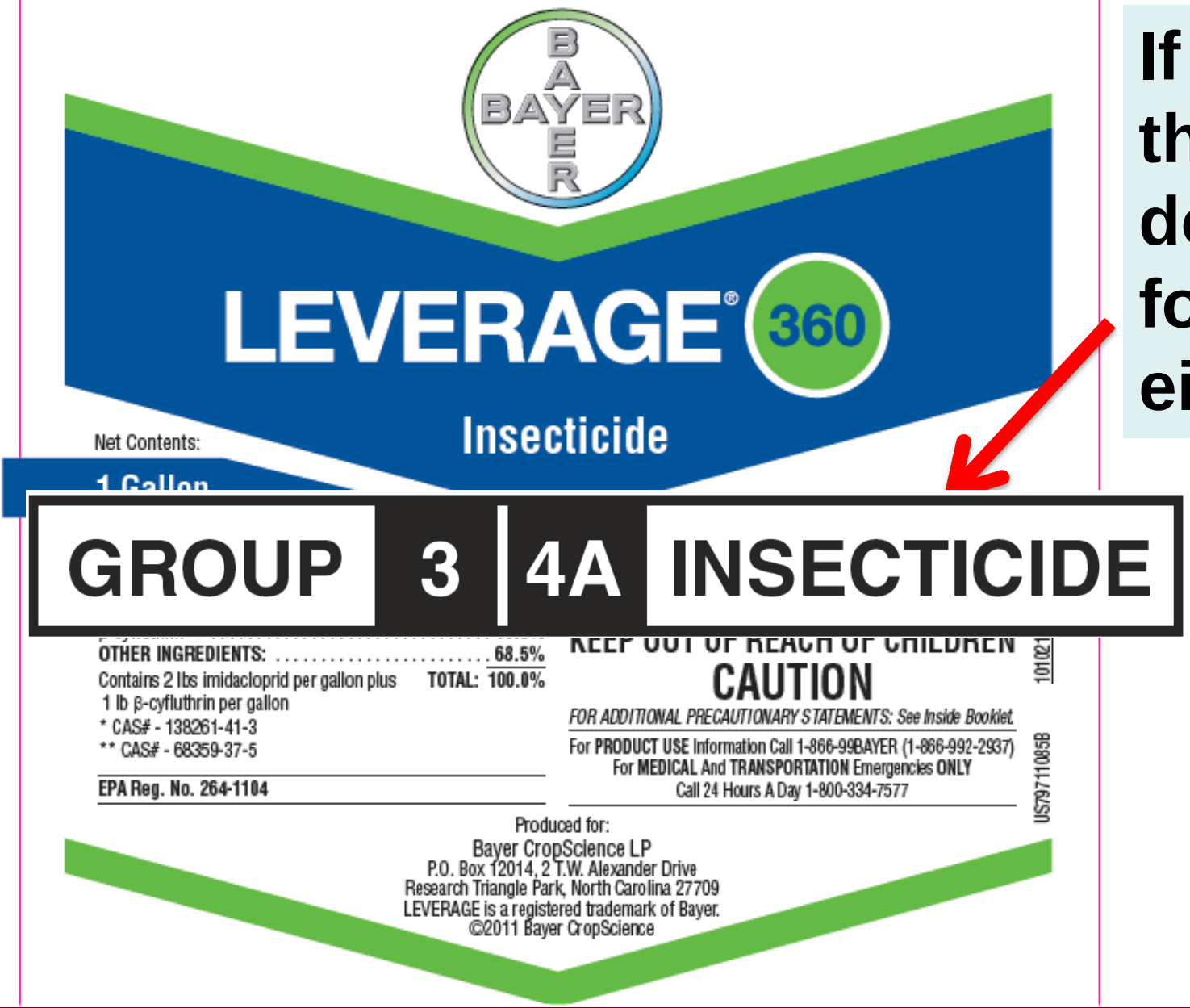
IRAC – Insecticide Resistance Action Committee

- International committee to coordinate IRM information and recommendations
  - Standardized testing methods, etc…

http://www.irac-online.org

Mode of Action group on label





If more than 1 moa, don't follow with either

Resurgence and Replacement

- Pest populations growing after application! Most associated with traditional insecticides but possible with other formulations
- Resurgence – a population, after being treated rebounds to numbers higher than before treatment
- Replacement – 2ndary pest outbreak. Another pest species population breaks out after treatment for 1st pest species.
- Possible reasons
  - reduction of natural enemies along with pest
  - direct favorable influences of pesticide on physiology and behavior of arthropods
  - removal of competitive species

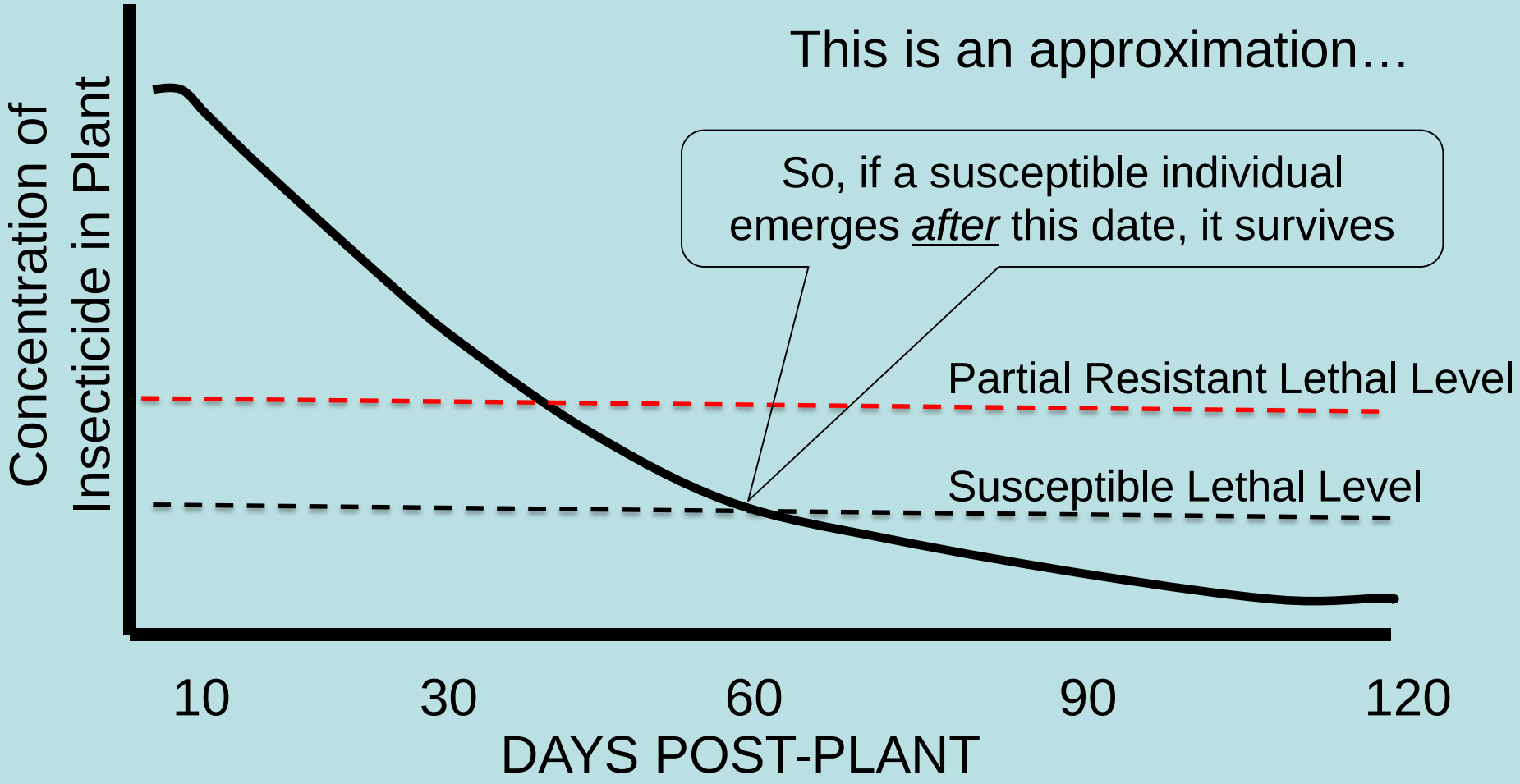
Reduction of natural enemies

- predators seem less impacted by insecticides than parasitoids. But disruption of natural enemies can be due to both direct and indirect pesticide effects
  - Direct - they get killed!
  - Indirect
    - starvation because of death of food source (prob most important)
    - death from 2dary poisoning from eating contaminated prey
    - repellency of insecticide

Other impacts – lag of natural enemies

- pest levels higher than tolerable but below potential, so grower sprays, removes prey items, predators die off because no food.
- Pest starts to recover but development of natural enemy populations lag behind because they have to wait for food supply to build.
- If they did develop, they'd probably bring pests back to previous level but before they can recover, grower sprays again. Over and over can remove natural enemy population more or less permanently! This a bad thing!

Other forms of resistance

- Resistance to Host Plant Resistance
  - ‘virulence’
- Behavioral
  - Crop rotations – northern corn rootworm, extended diapause
  - Extended emergence – Colorado Potato Beetle

Behavioral resistance?