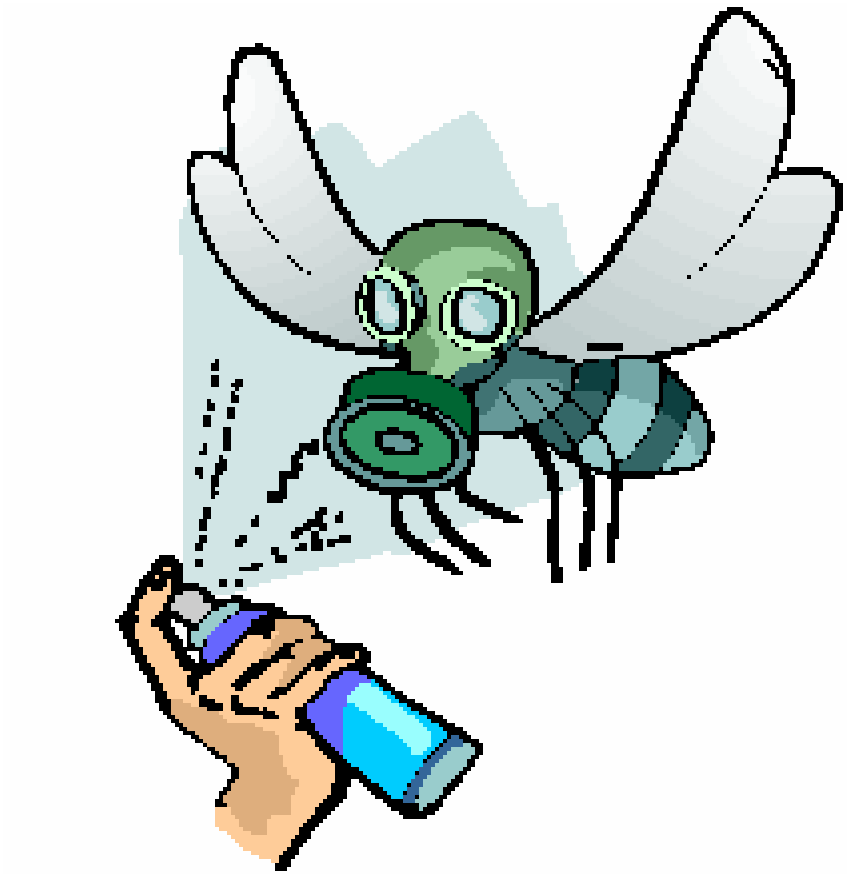
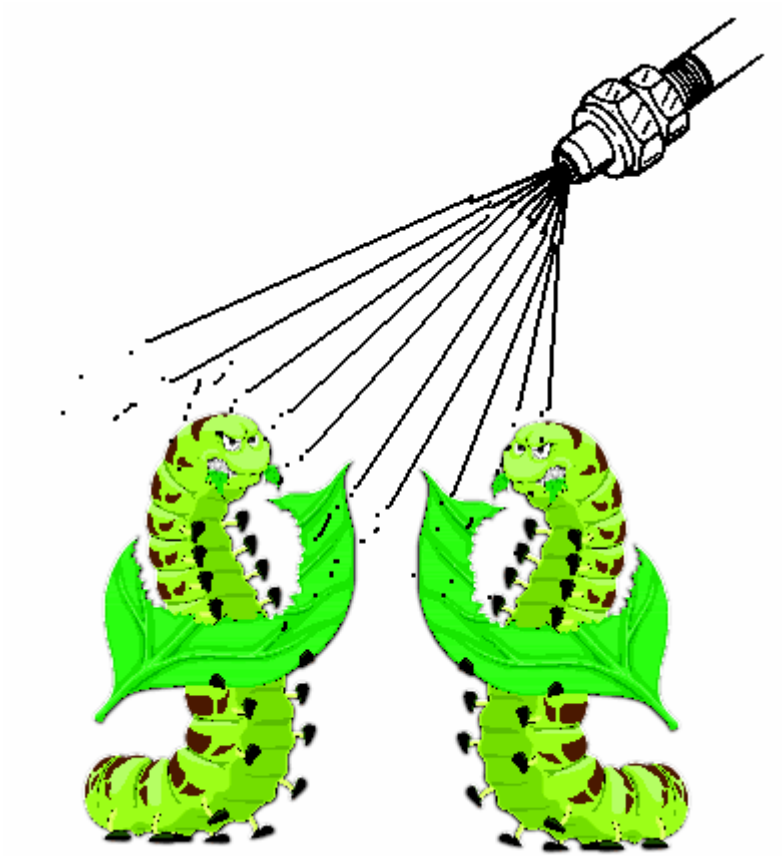
Highlights from Migration lecture

✓Why do Monarch butterflies migrate north in the spring?
✓Why is the majority of insect migration of short duration?
✓Using Kennedy's definition of insect migration explain the significance of; persistent, straightened out, and temporary inhibition to vegetative cues.
✓In the 1960s the paradigm for insect shifted from an emphasis on _____ to a concentration on _____.

✓Define; dispersal, accidental movement, and phoretic movement.

Insecticide Resistance: mechanisms and management



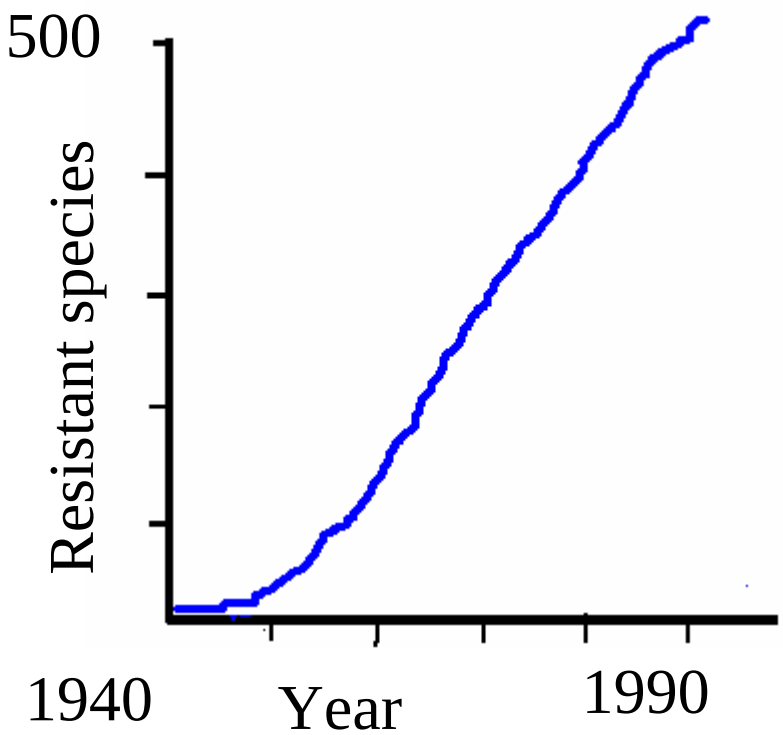


Outlines

- Resistance overview
- Evolution of resistance
- Factors promoting the development of insecticide resistance
- Resistance mechanisms
- IRM (Insecticide Resistance Management)
- Bt crop resistance management

Resistance overview: history

- 1908 San Jose scale to lime sulfur in Washington
- 1946 DDT resistant houseflies in Sweden
- 1997 over 500 arthropod species resistant to 1 or more insecticide groups



Resistance overview: what is it?

- WHO, 1957: “The development of an ability in a strain of insects to tolerate doses of toxicants which would prove lethal to the majority of individuals in a normal population of the same species”
- It’s about a pest population NOT an individual: Reduction in the susceptibility of a population to pesticides
- It’s genetically based: heritable reduction in susceptibility. It is not susceptibility difference (known as tolerance) between different stages or feeding on different plants

Resistance overview: the champions

Green peach aphid

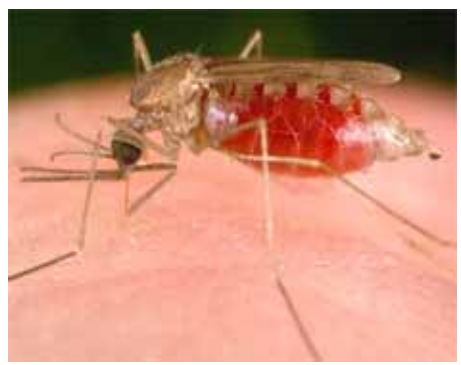
Colorado potato beetle

Diamondback moth

Whitefly

spider mite

cotton bollworm

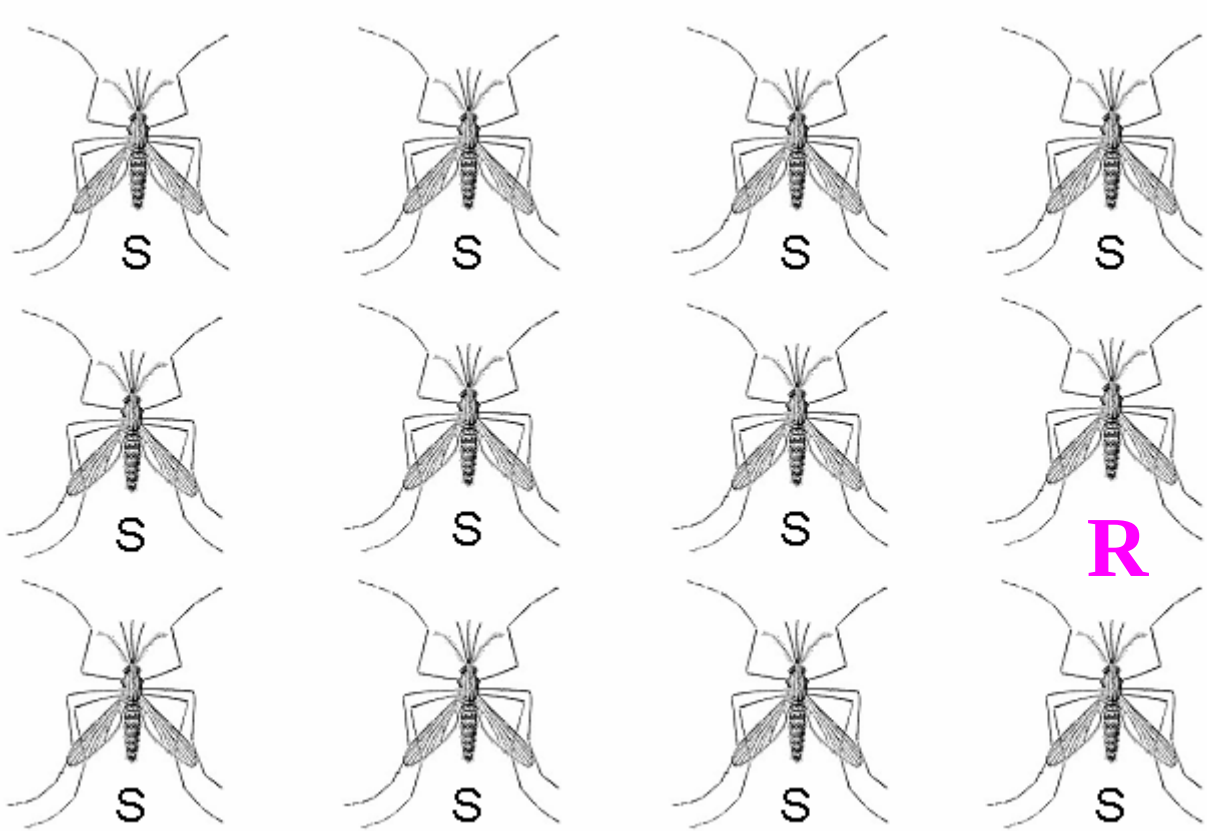


mosquito

Resistance overview: type of resistance

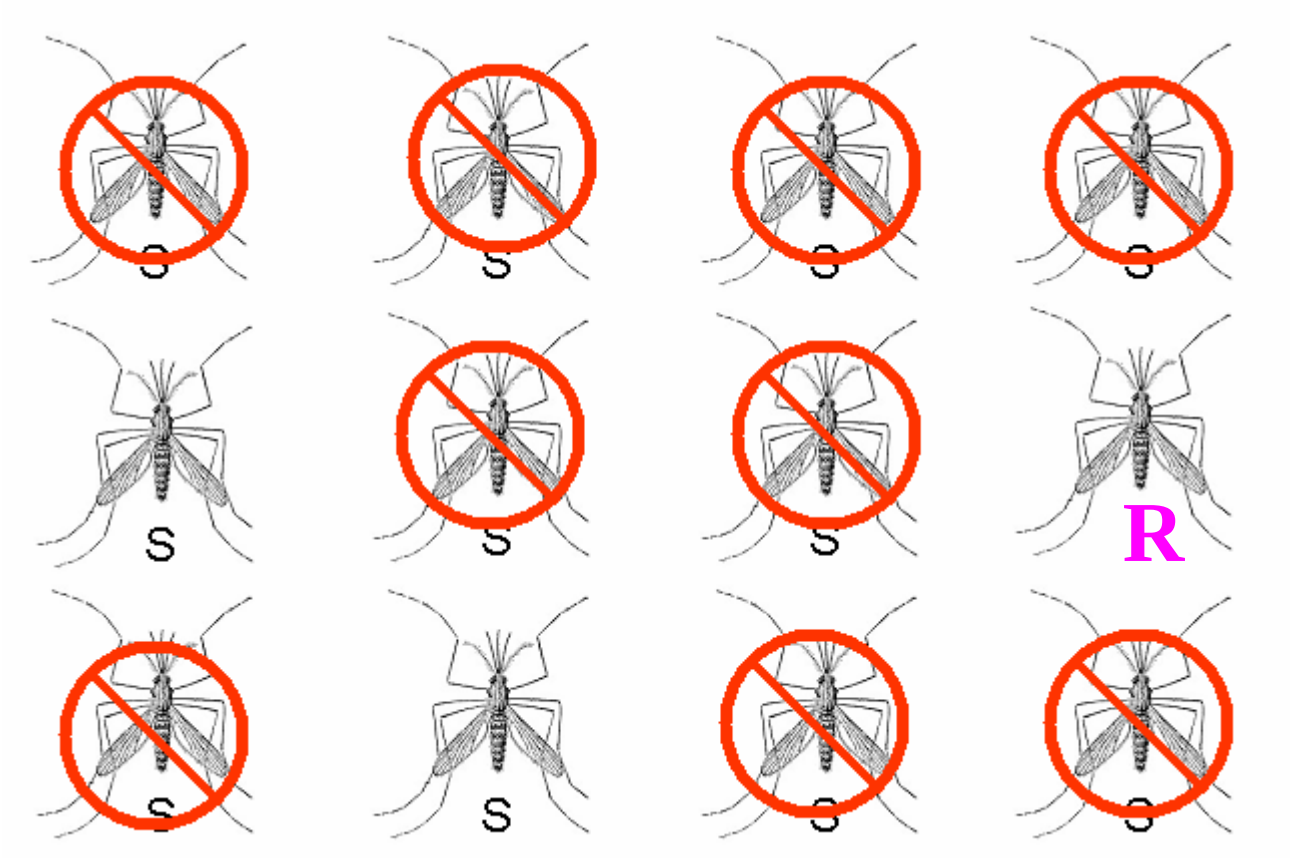
- Multiple resistance: resistance to multiple insecticides of different classes by multiple mechanisms. Consequence of sequential application of insecticides (Pesticide treadmill)
- Cross resistance: resistance to one insecticide leads to resistance to another yet unused insecticide. Usually the two insecticides belong to the same class and share identical or similar mode of action
- Negative cross-resistance: resistance to insecticide A leads to susceptibility to B and vice visa

Evolution of Insecticide resistance
How does it occur?



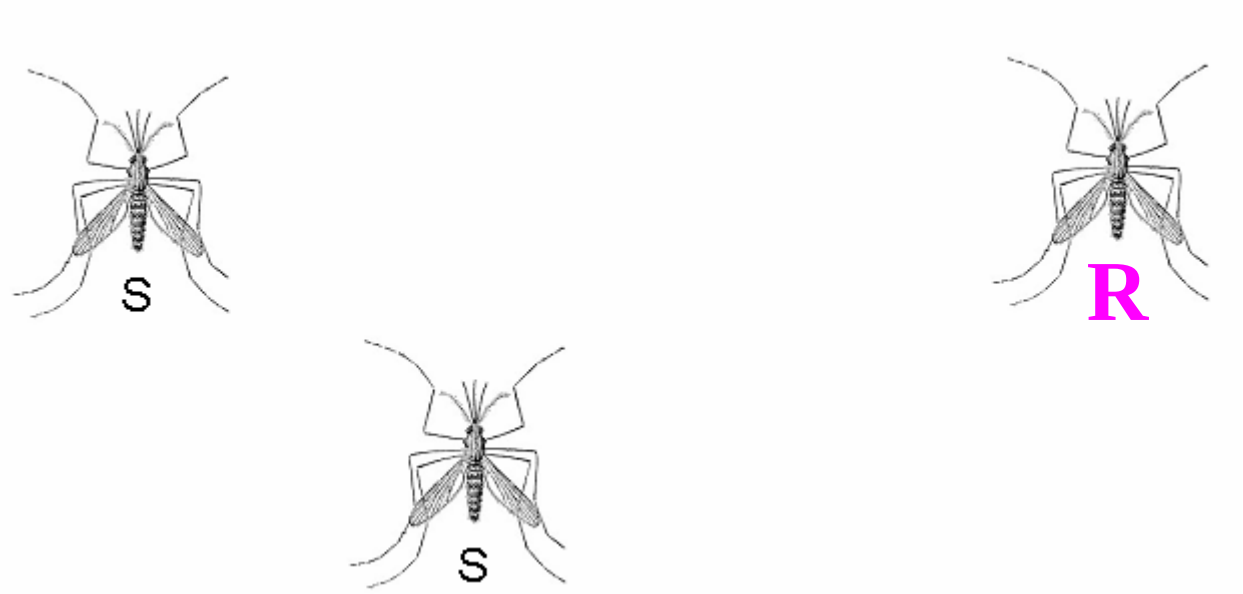
Individuals in a population are never equally susceptible to an Insecticide. Although initially rare, insecticide-resistant R genotype is present. Frequency is 1/12 = 0.083

Evolution of Insecticide resistance



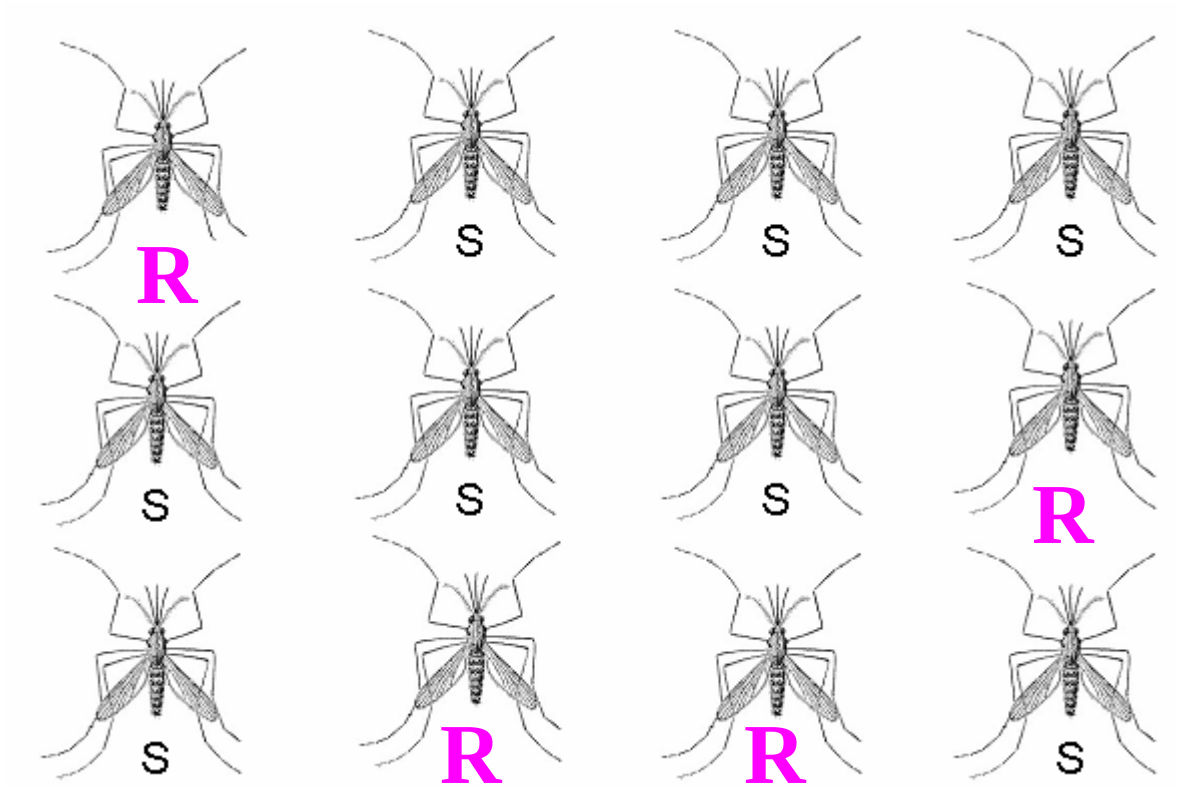
An insecticide is used, leaving insecticide-resistant individuals (their R genotype) and some susceptible individuals (S genotype)

Evolution of Insecticide resistance



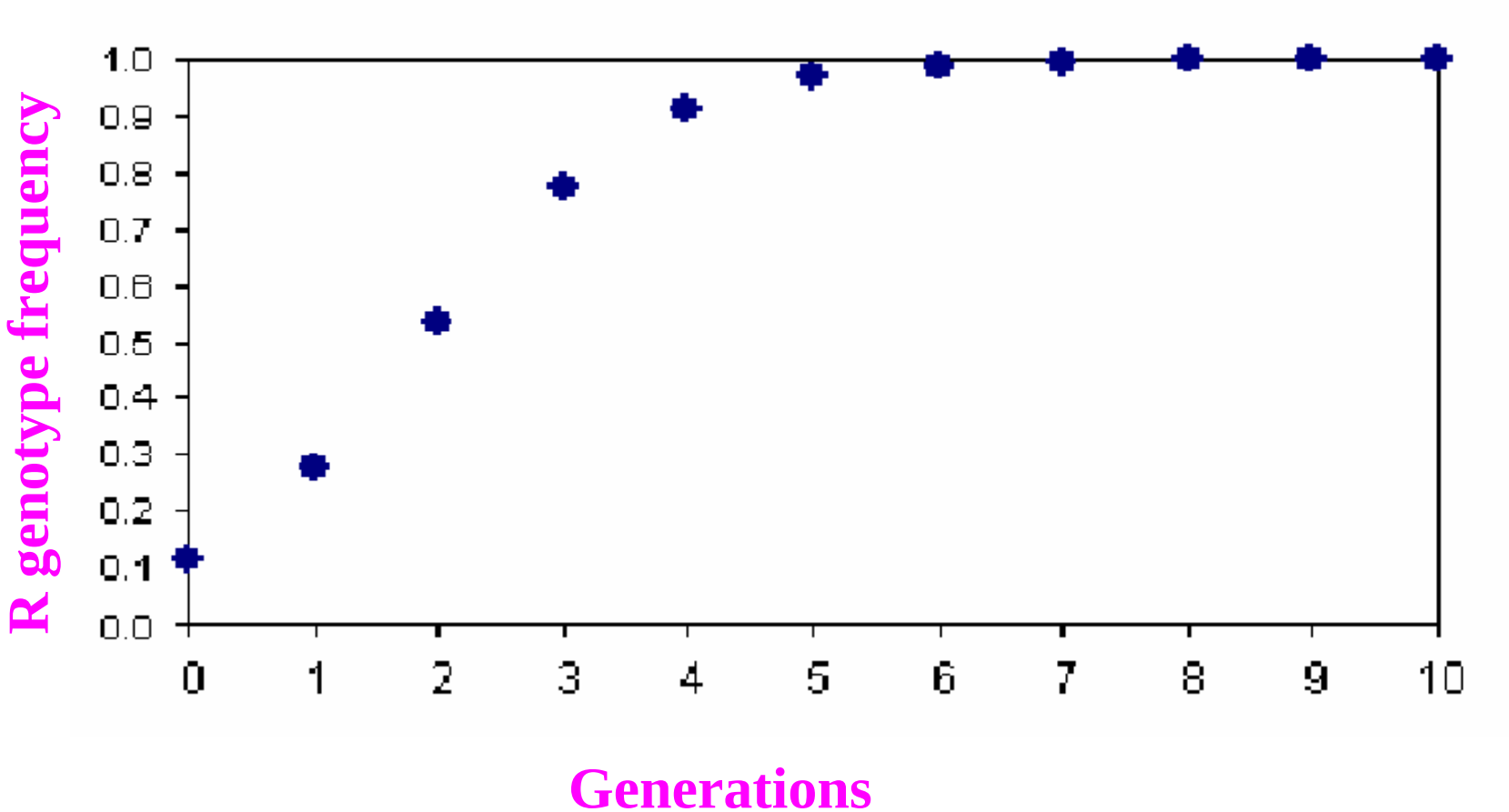
Now, the frequency of resistant R genotype is 1/3 = 0.333

Evolution of Insecticide resistance



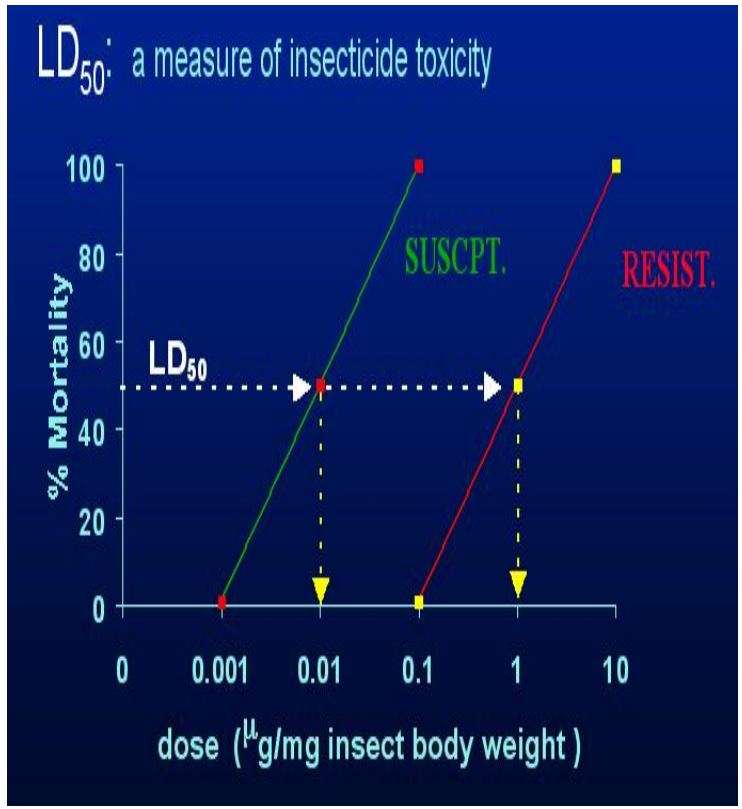
If R genotype reproduces as well as S genotype, in the next generation the frequency of R will be the same as the survivors in the preceding generation, i.e. 0.333

Evolution of insecticide resistance



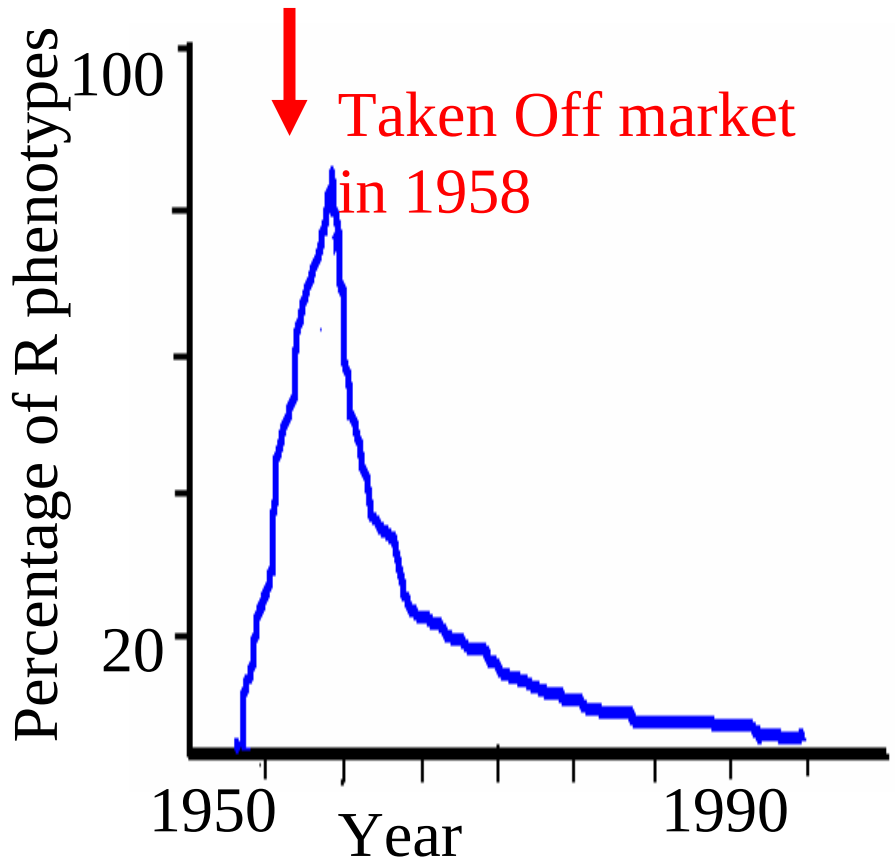
Evolution of insecticide resistance

- When LD50 ratio ≥ 10, resistance occurs
- Resistance: occur through insecticide selection
- Selection acts on genetically-based variation in susceptibility which arise from:
  - Mutation, the source of all new genetic variation
  - Genetic recombination that rearranges genetic variation
  - Gene flow from populations having different allelic frequencies



Persistence of resistance

- Resistance take a while to develop suggest R alleles are initially rare
- The rarity of R allele suggests it comes with reduced fitness
- Thus R allele should fall to a very low frequency without insecticide selection
- Example: dieldrin resistance in Australian sheep blow fly declines due to low overwintering survival



Some resistance are fixed

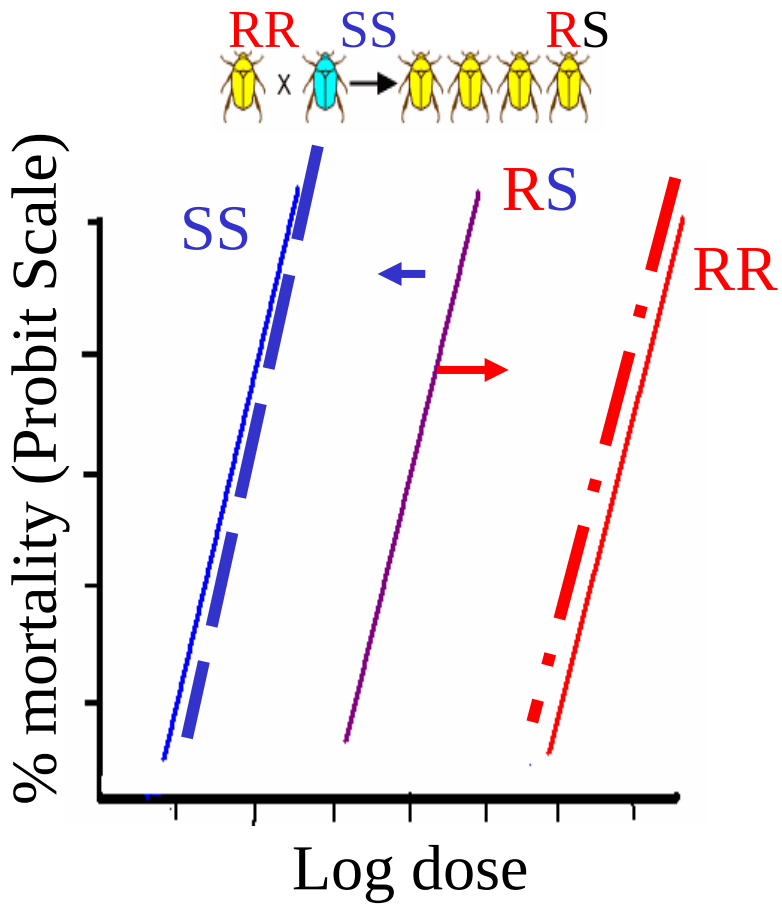
- DDT resistance fixed in many insects
  - Housefly
  - Drosophila
  - Pink bollworm
- Dicofol (structurally similar to DDT) resistance in European red mite
- Bendicarb (a carbamate) resistance in many strains of German cockroaches

Factors influencing evolution of resistance

- Genetic factors
- Biological factors
- Operational (application) factors

Genetic Factors influencing evolution of resistance

- Number of R gene: quicker if one major gene
- Dominance of R alleles: quicker if R allele is dominant
- Initial frequency of R alleles: the higher the quicker
- Fitness of R alleles: quicker if no fitness cost

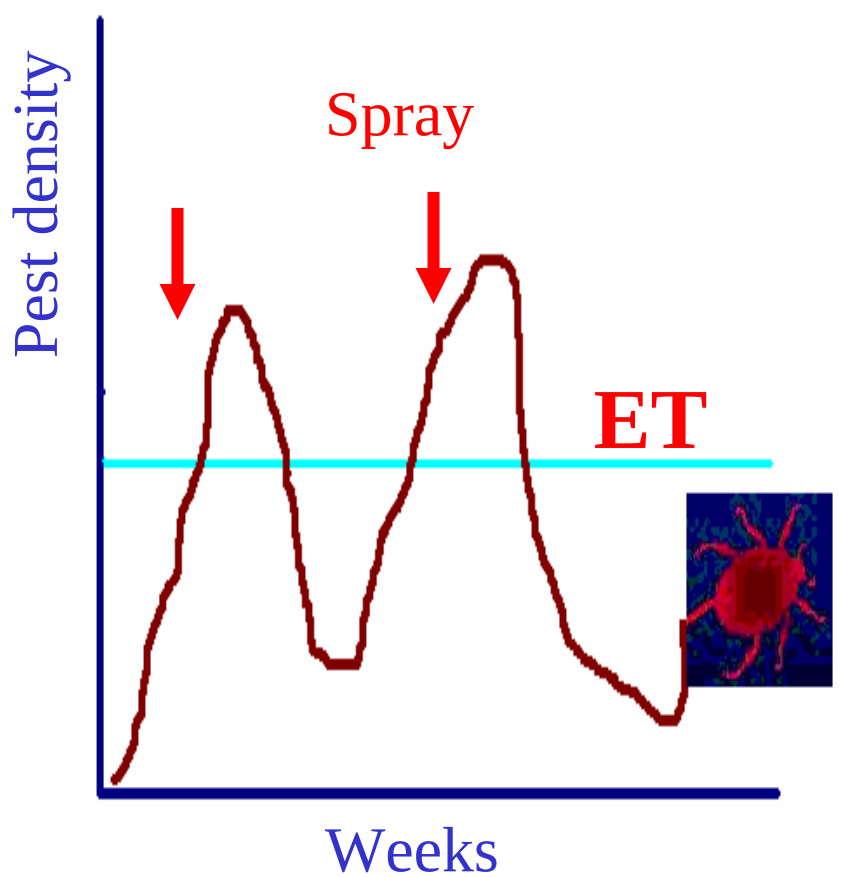


Biological factors that promote resistance

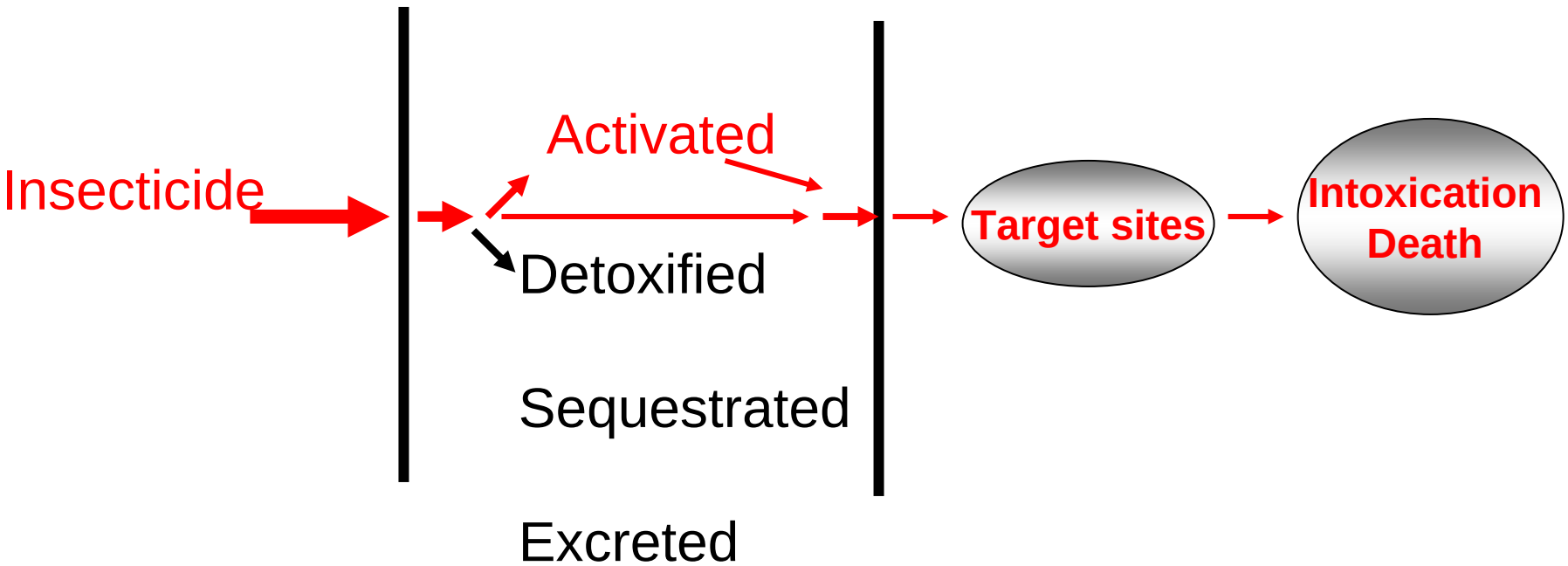
- Short generation time
- High fecundity
- No (or little) migration occurs between populations
- The species is highly mobile, increasing the possibility of exposure to insecticides

Operational factors that promote resistance

- Low economic threshold (ET)
- Use the same insecticide every generation
- A large geographical area is treated
- High dose
- No refuge exists
- Long persistent insecticide or slow-release formulations
- Use insecticides related to one used earlier



Resistance mechanisms: how do insects become resistant ?



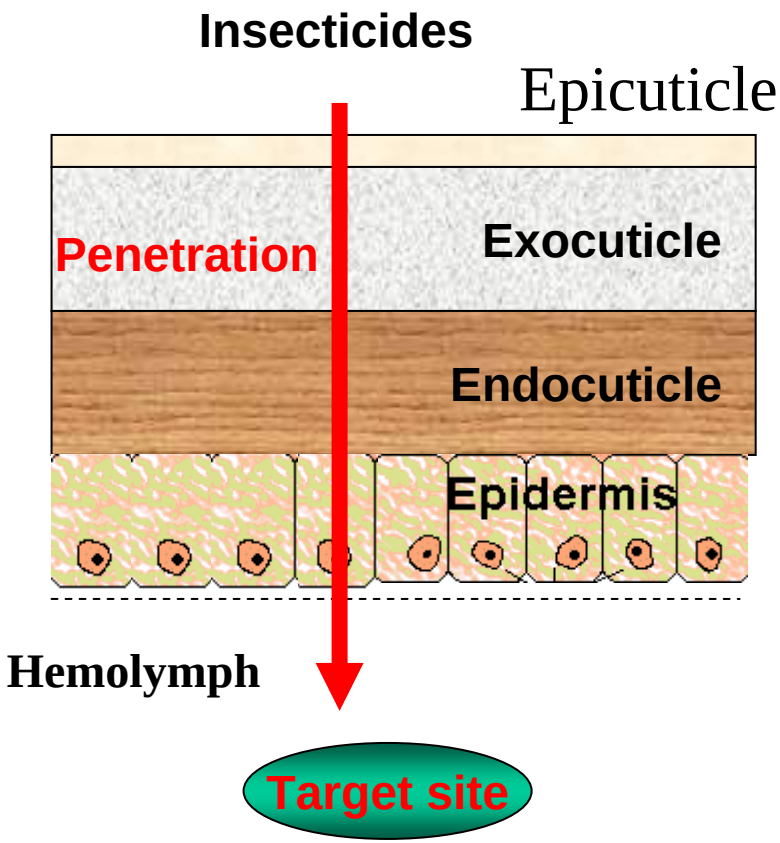
Behavioral resistance: avoiding toxin

- Detect and avoid the toxin
- Anopheles mosquito: SS lives and bites inside home, but RR remains outdoor and flies into house to bite because DDT was applied to interior walls
- Houseflies: avoid treated surface
- Cockroaches: avoid treated surface and baits
- Diamondback moth: avoid permethrin

Run from insecticides

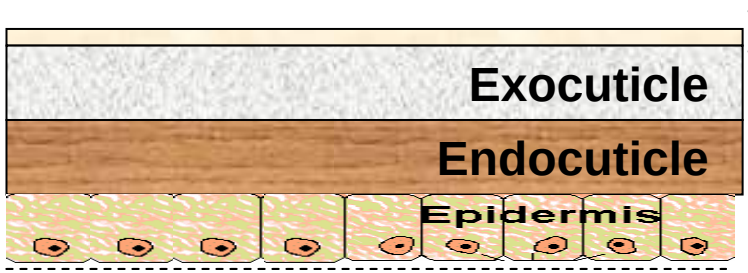
Reduced penetration

- RR individuals have a *pen* gene that reduces penetration of insecticides 2 to 3 fold due to
  - Modified composition
  - Modified structure
- Protects pests from a wide range of contact insecticides
- Usually present along with metabolic and/or target mechanisms to amply resistance

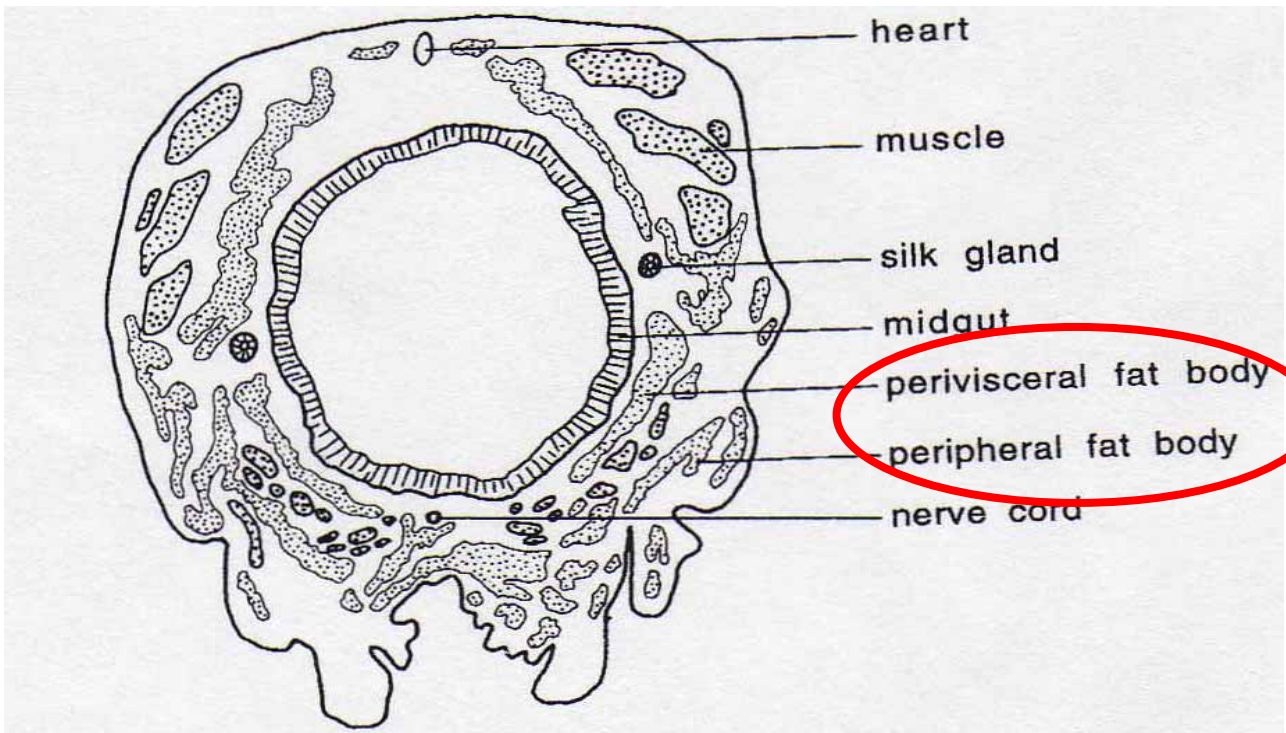


Increased Sequestration

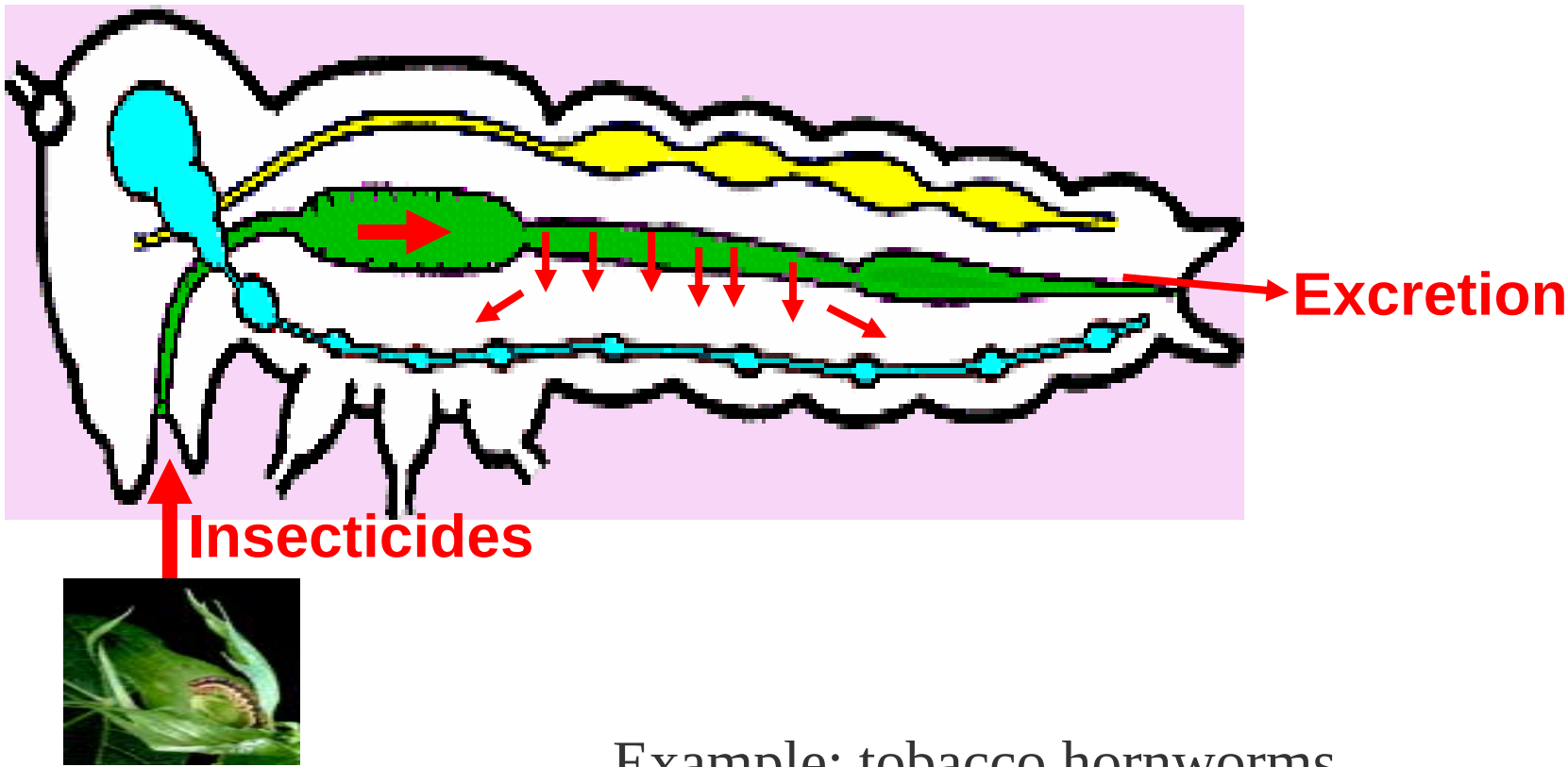
- **Integument**



- **Fatbody**



Increased direct excretion



Example: tobacco hornworms

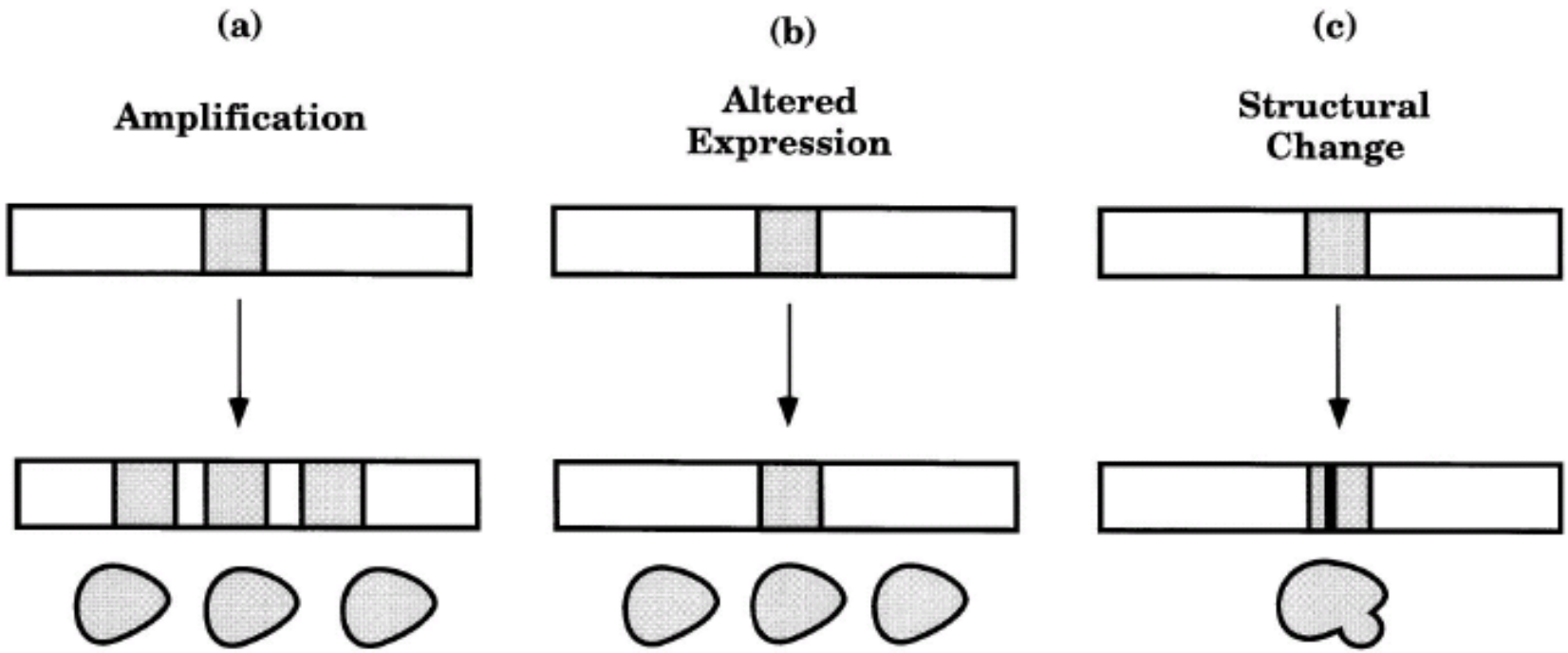
Increased Metabolism

- The most common resistance mechanisms, called metabolic resistance. RR individuals have more enzymes or more efficient enzymes
- P450: Cytochrome P450 monooxygenases, involved in metabolism and resistance to all classes of insecticides
- Esterases: involved in resistance to organophosphates (OP), carbamates (Carb), and Pyrethroids
- GST: involved in resistance to DDT, OP and Py

Reduced target sensitivity

- Target site insensitivity: the 2nd most common mechanisms, called altered target-site resistance
- AchE insensitivity : resistance to OP and Carb
- Sodium Channel insensitivity: knockdown resistance (kdr) to DDT and Py. Super kdr
- GABA receptor (Cl^- channel) insensitivity : resistance to cyclodiens, fipronil, and avermectins
- AchR insensitivity: resistance to nicotin, neonicotinoids and spinosad

Molecular mechanisms of resistance



Esterase in mosquitoes green peach aphid

P450, GST, esterase

esterase, Target sites

Resistance mechanisms: summary

- Behavioral resistance
- Penetration resistance
- Increased sequestration and direct excretion
- Metabolic resistance: P450, esterase, GST
- Altered target-site resistance: AchE, AchR, sodium channel, GABA receptor

Insecticide resistance management (IRM)
What can you do about resistance?

- Historically, growers respond to resistance by
  - Increasing dosage and frequency of application which only accelerate the development of resistance
  - Switch to another insecticide---- so begins the pesticide treadmill
- In theory, resistance can be minimized by
  - Increasing survivorship of SS individuals
  - Decrease survivorship of resistant individuals (R allele)
- Three IRM strategies
  - Moderation
  - Saturation
  - Multiple attack

IRM by moderation

- Strategy: Conserve susceptible alleles by reducing selection pressure in the following ways
  - higher ET, low dosage, less frequent application
  - local, rather than area wide applications
  - Preserve refuge by treating only "hot spots" in a field
  - Use short residual insecticide and avoid slow-release formulation
- Good for environment and natural enemies, but not practical for high value crops (fruits, veg.) and medical insects

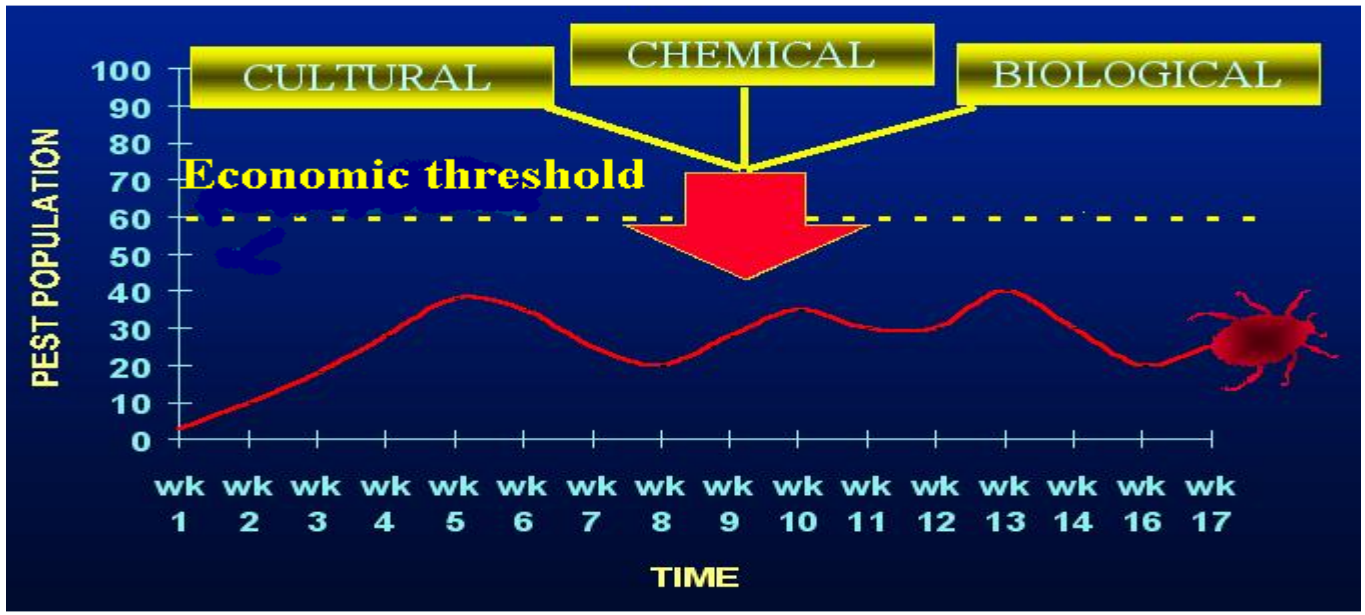
Management by Saturation

- Strategy: suppress resistant alleles by maximizing the mortality of R individuals in the following ways
  - High dosage
  - Apply at the most vulnerable stage of pests: age-specific expression of resistance, low Resistance level at younger stages
  - Synergists to inhibit detoxification enzymes
- Work if R allele is recessive and rare
- Environmental concern
- Necessary for high value crops/medical pests

Management by multiple attack

- Strategy: suppress resistant alleles by diversifying selection forces and complicating pests' adaptation in the following ways
  - Mixed use of insecticides at low dosage that differ in mode of actions. A-resistant individuals can be killed by insecticide B and vice visa.
  - Insecticide rotation
- Concern: cross resistance or multiple resistance
- Necessary for high value crop/medical pests

The best solution: integrated Pest management (IPM)



- Combination of cultural, chemical and biological control tactics
- Diversifying selection forces and complicating adaptation
- Resistance to one tactics can be compensated for by other tactics

Resistance to crop rotation

- One generation per year
- Adults lay eggs in the cornfield. Diapausing eggs hatch the following spring and feed on corn roots
- Corn-soybean rotation
- Crop rotation no longer effective because
  - Northern Corn rootworm extend its egg diapause from one year to 1-5 years.
  - Western Corn rootworm leave corn field to lay eggs in soybean or other crop fields

Northern Corn rootworm

Western Corn rootworm

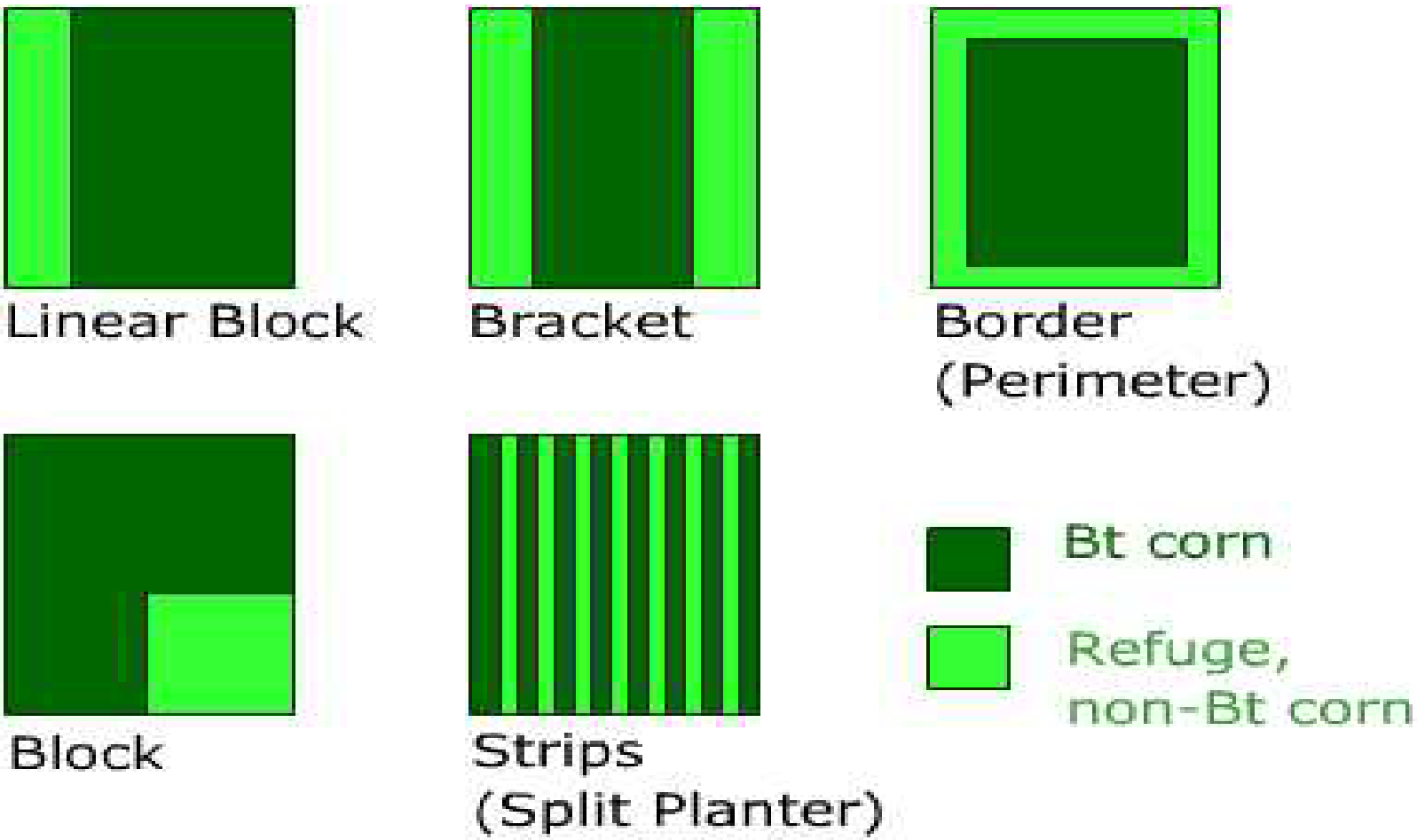
Bt-transgenic crops: high resistance risk

- Bt-transgenic crops: *Bacillus thuringensis* (Bt) toxin genes (e.g. Cry1Ac) introduced to plants
- Simplified toxin: one or two Bt toxins in plant vs. 5 toxins produced by bacteria
- Persistent selection: continual production in plant vs. sprayed Bt (degraded rapidly)
- Massive plantations
- High resistance risk

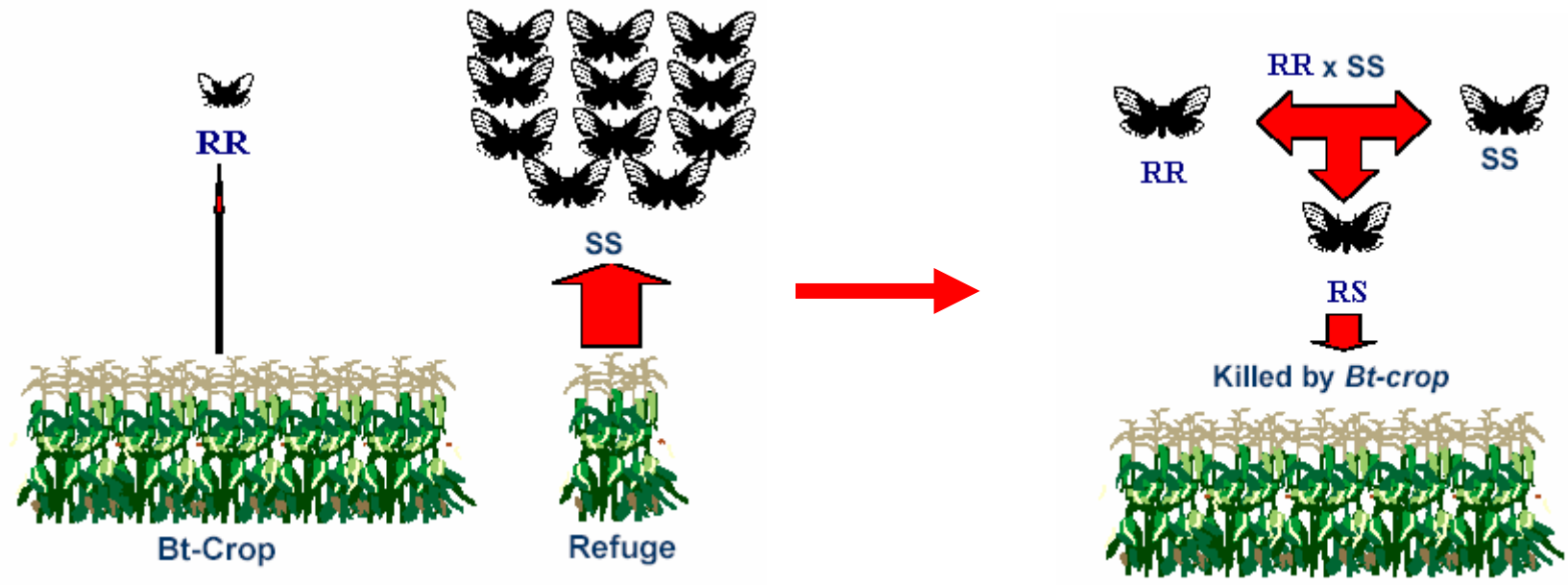
Bt crops IRM: assumptions and strategy

- High dose refuge strategy (EPA , 1996)
  - High dose: > 25 times the concentration required to kill susceptible individuals
  - Non-Bt crop refuge size: 20% (spray permitted) or 4% (no insecticide allowed)
- Assumptions
  - Resistance due to one gene
  - R allele is recessive
  - R allele frequency is low (< 1/1000)
  - R and S individuals mate randomly

Bt crops: refuge planting options



How does the strategy work?



Is the strategy really working?

- So far so good.
- Most target pests have not evolved resistance to Bt corn or Bt cotton after 12 years (from 1996)
- In Arizona, both the resistance and population of pink bollworm have declined


FINISH