Section 8: Problems with insecticides

- The introduction of "modern" insecticides (chlorinated hydrocarbons or organochlorines) beginning in the 1940s revolutionized insect control. They were:
  - Broad-spectrum
  - Long-lasting
  - Inexpensive
  - Effective

  Therefore, they were used extensively until the 1960s.

Silent Spring

- Publication in 1962 of "Silent Spring" written by Rachel Carson caused scientific and public voice to be raised in criticism of these insecticides.
- Criticisms were based on
  - Toxicity to humans,
  - Toxicity to wildlife,
  - Tolerance of some strains to these insecticides.

Over time, and with a shift in pesticide chemistry, these issues remain, though the order of importance is likely reversed.

Also, note problems with malaria eradication, p. 108-110 of text.

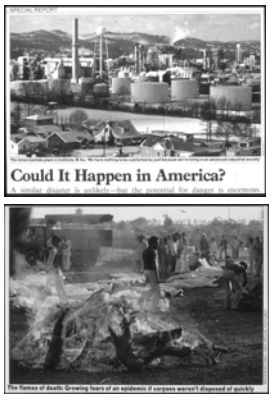
Toxicity

- Insecticide development has focused on chemicals that disrupt nerve transmission - probably because they work so quickly.
- However, nerves of all animals are similar, so all susceptible to poisoning.
- Principal reason humans not often affected is that toxicity is dose-dependent. Small animals such as insects easily poisoned with small doses, but so long as human contact is minimized, acute toxicity among humans is infrequent.

Human toxicity

- Principal hazard occurs among those who mix or apply pesticides (lots of exposure), or possibly from long-term exposure.
- Some poisonings occur when applicators don't, or can't, read pesticide labels (illiterate, wrong language, children, or ignore warnings).
- Few poisonings from legitimate, according-to-the-label applications.
- Occasionally a disaster occurs, as in Bhopal, India in 1984, when an insecticide release from a manufacturing facility killed 3,800 residents living near the plant. Eventually, more that 20,000 people were affected, the world's largest industrial disaster.

The potential for large-scale poisoning exists, though it is unlikely.



More human toxicity

- Many problems occurred when DDT was replaced by more toxic products (OPs, ecologically preferable because degrade quickly). Any time a new, more toxic product is introduced, this risk develops.
- Chronic exposure more speculative. Difficult to demonstrate effects of chronic exposure of low levels of insecticide on longevity, reproduction and general health of humans.
- Chronic exposure to high levels (workers who handle, or drink contaminated well water) has more substantive record of problems.

Children should not be allowed access to insecticides and should be taught to recognize such toxicants at an early age.

Particular care should be taken when handling large volumes of pesticide or restricted use materials, due to the greater risk of injury.

Effects on wildlife

- Insecticides can affect all wildlife, though most apparent on birds.
- Both acute and chronic toxicity.
  - Consumption of treated seed or granular formulations by birds.
  - Treatment of water, or adjacent to water, leading to fish kills.
  - Biomagnification of insecticides and poisoning of top predators.
  - Endocrine disruption due to similarity of chlorinated hydrocarbons to vertebrate hormones.

This grouse was found dead following an application of dieldrin. According to Rachel Carson, bird mortality was very common after application of chlorinated hydrocarbons. Technology now allows rapid determination of poisonings, and such mortality rarely occurs and is easily documented.

However, some formulations can lead to wildlife toxicity, hence the recent elimination of the registration for diazinon granules, which were sometimes eaten by geese.

Lake Apopka alligators

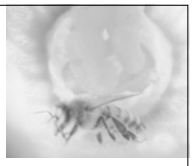
- Spill of the acaricide dicofol in 1980.
- Dicofol contains DDT and isomers, and one of its breakdown products, DDE, blocks action of testosterone.
- Ratio of testosterone: estrogen affected.
- Feminizing effect on embryos; poor egg hatch.
- Male genes not activated, prevents normal development of penis and sex glands.
- May explain decline in alligator populations.

Unhealthy alligators indicate an unhealthy environment, and even though alligators can be pests, their decline in Lake Apopka signifies the need to restore the lake to a healthier state.

"Invisible" wildlife

- Biggest problem may be effects on "invisible" wildlife - non-target insects such as predators, parasites and pollinators.
- Destruction of beneficials causes "resurgence" - environment freed of pests and beneficials is recolonized first by pests, and initially becoming over-abundant.
- Insecticides often induce problems with mites, scale insects, aphids and whiteflies - sometimes others.

Other problems



- Pollinators, especially bees, affected by insecticide use.

  Remedies: remove bees or restrain flight, cover during application, time when flights minimal.

- Insecticides can affect plants directly by suppressing metabolism, and causing spots or blemishes. Toxicant or formulation?

Resistance to insecticides

- Although new classes of pesticides have overcome some traditional problems of use, loss of effectiveness (resistance) remains an important issue.
- Populations are genetically variable, with some individuals less sensitive to any insecticide. Continued use favors survival of the resistant individuals, and this genotype soon dominates.
  - Resistance is usually physiological, and due to
    - Decreased cuticular penetration
    - Target site insensitivity
    - Increased metabolism

    We usually attribute resistance to enhanced degradation of insecticides, which is the principal mechanism. Key components are the acetylcholine esterase enzyme, voltage-gated sodium channel, and GABA-gated chloride channel.
  - Enhanced microbial decomposition of insecticides occurs in some soils following long-term use of chemicals and build-up of microbial populations.

- Sometimes behavioral resistance (avoidance of contact) occurs.

• Insecticide resistance results from Darwinian evolutionary processes, and arises from rare random mutations or through migration of resistant individuals into pest populations. In response to selective pressure by insecticides, the frequency of resistance genes increases.

• Development of insecticide resistance is likely an inevitable process if high levels of selective pressure are imposed. This occurs most often in closed systems (greenhouses, buildings, isolated agricultural areas) or where insecticide is used almost continuously (lawns, vegetable and fruit crops).

Resistance

- Resistance is most common in moths, beetles, Hemiptera/Homoptera, flies, mites and ticks.
- Number of resistant species increased from about 45 in 1944, to 450 in 1990.
- Cross-reaction (cross-resistance) is a problem:

  - Within classes of insecticides, resistance to one product may signal resistance to another.

  - Also, among classes with similar modes of action, resistance in one class leads to rapid appearance of resistance in another (aka “multiple resistance”).
- Beneficial insects also may develop resistance, but more slowly.

The following table shows an alarming pattern of resistance:

Pattern of development of insecticide resistance on Colorado potato beetle populations, Long Island, New York (after Forgash 1984).

Insecticide	Year introduced	Year first failed
Arsenicals	1880	1940
DDT	1945	1952
Dieldrin	1954	1957
Endrin	1957	1960
Carbaryl	1959	1963
Azinphosmethyl	1959	1964
Monocrotophos	1973	1973
Phosmet	1973	1973
Phorate	1973	1074
Carbofuran	1974	1976
Oxamyl	1978	1978
Fenvalerate	1979	1981
Permethrin	1979	1981

Potato beetle resistance

• Colorado potato beetle resistance is not typical, though not the worst case either.

• The rapid development of resistance to most classes of insecticides (chlorinated hydrocarbons, organophosphates, carbamates, pyrethroids) shows how challenging it can be to manage an insect using insecticides alone. Also, the occurrence of resistance to several insecticides occurring almost simultaneously shows the potential problems of cross-resistance.

Approaches to managing insecticide resistance

- Alternate materials from different classes, including introduction of new classes.
- Use lower rates; don't try to eliminate all insects with high doses.
- Switch life stage targeted if holometabolous insects.
- Allow susceptible genotype to survive (refugia).
- Add synergist to help block detoxification.
- Find other sources of mortality (BC, HPR).

Managing resistance

Insecticide-resistant "superbugs" occur in various taxa

- No easy solution to resistance problem, and considerable disagreement about how best to manage.
- Best solution may be not to depend on insecticides - use occasionally - only when truly needed.

Questions

- Why were chlorinated hydrocarbons (organochlorines) used so extensively from their introduction until the 1960s (and beyond in some cases)?
- What was Rachel Carlson's contribution to changing the pattern of insect control?
- How do insecticides affect humans. How does such exposure occur? What can be done to prevent it?
- How do insecticides affect wildlife (including beneficial insects)?
- Describe the insecticide resistance problem in pests, How can insecticide resistance be managed?

Insecticides and disease transmission

- Control of vectors with insecticides is often considered as a principal means to suppress disease transmission to plants and animals (including humans), but insecticide-based vector control has its limitations.
- Both with plant and animal pests, the insecticide must reach the potential vector early enough to kill the pest before the disease is transmitted. This may not always be feasible, primarily because it is not always possible to treat the host with an insecticide that works quickly enough to block transmission.

Transmission of diseases

- Insects vector many types of pathogens, including fungi, bacteria, mollicutes, protozoa, nematodes, and viruses. There are 3 principal modes of transmission:
  - Insects vector disease passively, by walking through or feeding in an infected area of the host that contains bacteria or fungal spores. The pathogens may be sticky and cling to the insect.
  - Some insects transmit bacteria, fungi, or viruses by feeding on infected host tissue and carrying the pathogen on their mouthparts when they visit other (uninfected) hosts.
  - Some insects ingest pathogens with the sap or blood, and the pathogen circulates in the insect's body, with or without multiplication in the host. The pathogen reaches the insects's salivary glands and then is secreted during a feeding event.

Transmission of diseases

- Diseases are most effectively transmitted by insects with piercing-sucking mouthparts; passive transmission is not very effective.
- Routes of transmission by piercing-sucking insects:
  - Non-persistently transmitted, stylet-borne
  - Semi-persistently transmitted, foregut-borne
  - Persistently transmitted, circulative
  - Persistently transmitted, propagative
- Thus, we effectively have non-persistent, semi-persistent, and persistent pathogens.
- Non-persistent diseases are quite important with respect to plants, but generally not so important in disease transmission to animals.

Time parameters in epidemiology

- Non-persistently transmitted diseases are transmitted to the host quickly (a few seconds), and disease acquisition is also brief (no latent period). Ability to transmit is limited to brief period of time.
- Semi-persistent diseases require minutes to transmit, and persist longer (hours) in the insect. No latent period.
- Persistent-circulative diseases require minutes to hours to acquire and transmit. Short latent period. Persists for 30 days to life of insect.
- Persistent-propagative diseases requires hours to days to acquire, latent period of weeks, persists for life of insect, may be passed to egg.

Important vectors

- Plant viruses: aphids, leafhoppers, planthoppers, whiteflies, thrips, mealybugs
- Plant bacteria: flies, beetles, bees, wasps, ants, leafhoppers, psyllids
- Plant fungi: beetles, aphids, scales, flies, bugs
- Plant nematodes: beetles
- Animal viruses: mosquitoes, midges, sand flies, deer flies, lice, fleas, ticks
- Animal bacteria: sand flies, lice, fleas, ticks, mites
- Animal nematodes: black flies, midges, sand flies
- Animal protozoa: mosquitoes, sand flies, tsetse flies, triatomine bugs, ticks,

Insecticidal control of vectors

- For rapidly transmitted diseases, there often is not enough time for the insecticide that has been applied to the host to have an effect. The disease is transmitted before the insecticide kills the vector.
- Effective protection of hosts occurs when the insect vectors are colonizers of the hosts, and when most of the transmission occurs by host-to-host movement of the vectors (secondary transmission), or when persistent diseases are involved (requiring prolonged periods of feeding).
- Protection is not very effective when transient, non-colonizing vectors, or non-persistent diseases are involved.
- Insecticides work principally by keeping insect populations from building to high numbers within a population of hosts, and by limiting among-host spread of a disease. They rarely keep disease from being inoculated into a host population.

How can insecticides be used more effectively for disease management?

- Insecticides often the principal tool for disease management, despite their limitations.
- Use insecticides as part of a management plan. Also, use repellents, trap-out or intercept vectors, or target stages (immatures?) before they attain vector capability.
- Examples include:
  - Mosquito repellents in addition to adulticides.
  - Reflective mulches to slow invasion of insecticide-treated crops.
  - Insecticide-treated or untreated barrier crops.
  - Mosquito control with larvicides; treatment of weed hosts.

Other means of avoiding diseases

- Use stylet oils on crop plants to avoid viruses.
- Reduce the propensity of crop invasion by reducing the source (non-crop or non-sprayed area).
- Use a host-free period to break disease cycle, or eliminate breeding sites of vectors to eliminate insects.
- Use host that are less attractive to vectors.
- Reduce the number of vectors with biocontrol or host-plant resistance.
- Culture only disease-free hosts to reduce among-host transmission.
- Prune or rogue disease from host populations.
- Eliminate volunteer crops, and wild hosts of diseases or vectors.

Viruses vectored to cucurbits

- Insect-borne viruses are generally the most severe type of diseases in crop plants because many are stylet-borne, non-persistent diseases that are difficult to control through vector protection. Disease symptoms may be expressed in the foliage (above) or fruit (below). If the crop is protected during the early stages of growth, however, the early most-valuable portions of the crop may not be affected.

Questions

- Can you describe the 3 principal modes of disease transmission?
- Can you describe the persistence of diseases on insect mouthparts and how that affects transmission.
- If you compare the vectors of plant disease with vectors of animal diseases, what similarities and differences do you note?
- When is insecticide useful for preventing disease transmission? When is it ineffective?
- What other steps can be taken that complement insecticide use for disruption of disease transmission.

Questions from supplementary readings

- Reading 8, Insect-vectored plant pathogen mgmt.
  - Can you distinguish between primary and secondary spread of pathogens?
  - Can biological control be used to manage plant pathogen transmission?
  - Can fungicide affect insects and spread of disease?
  - What form of host plant resistance is desirable for curtailing disease spread?
  - How can urbanization affect spread of disease to crops?

More questions from supplementary readings

- Reading 10, Plant viruses and insects
  - Can you name the most important taxa of plant viruses?
  - Plant viruses seem to be increasing in number. Why?
  - What are the principal functions of saliva in piercing-sucking insects?
  - Can you describe some benefits to insects of having piercing-sucking mouthparts? Some advantages to the insect pathogens?

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