Pesticide Resistance Prevention

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Defining resistance

'A heritable change in the sensitivity of a pest population that is reflected in the repeated failure of a product to achieve the expected level of control when used according to the label recommendation for that pest species'

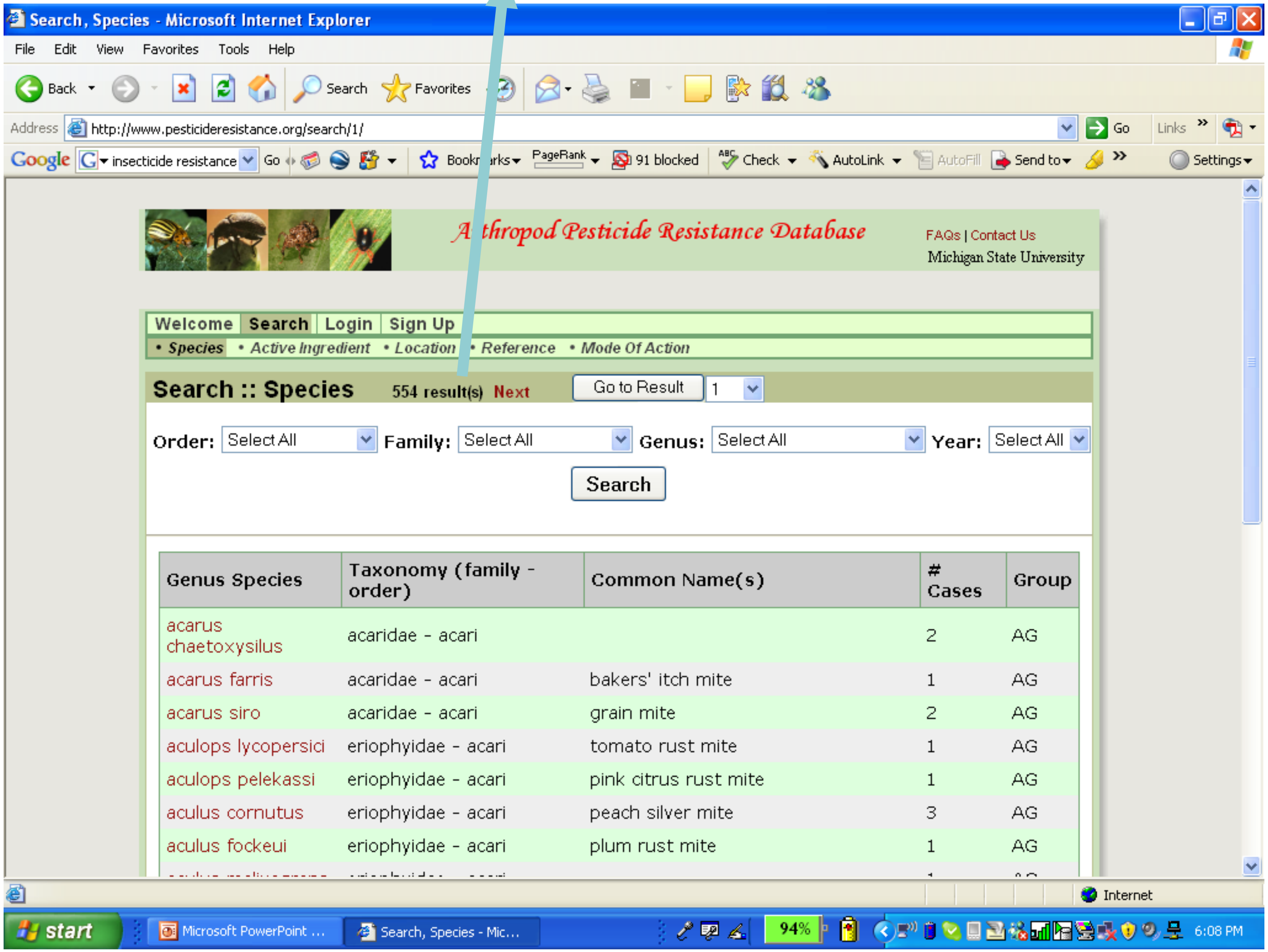
IRAC, 2007

BUT, resistance often begins a long time before 'field resistance' is detected

First detected

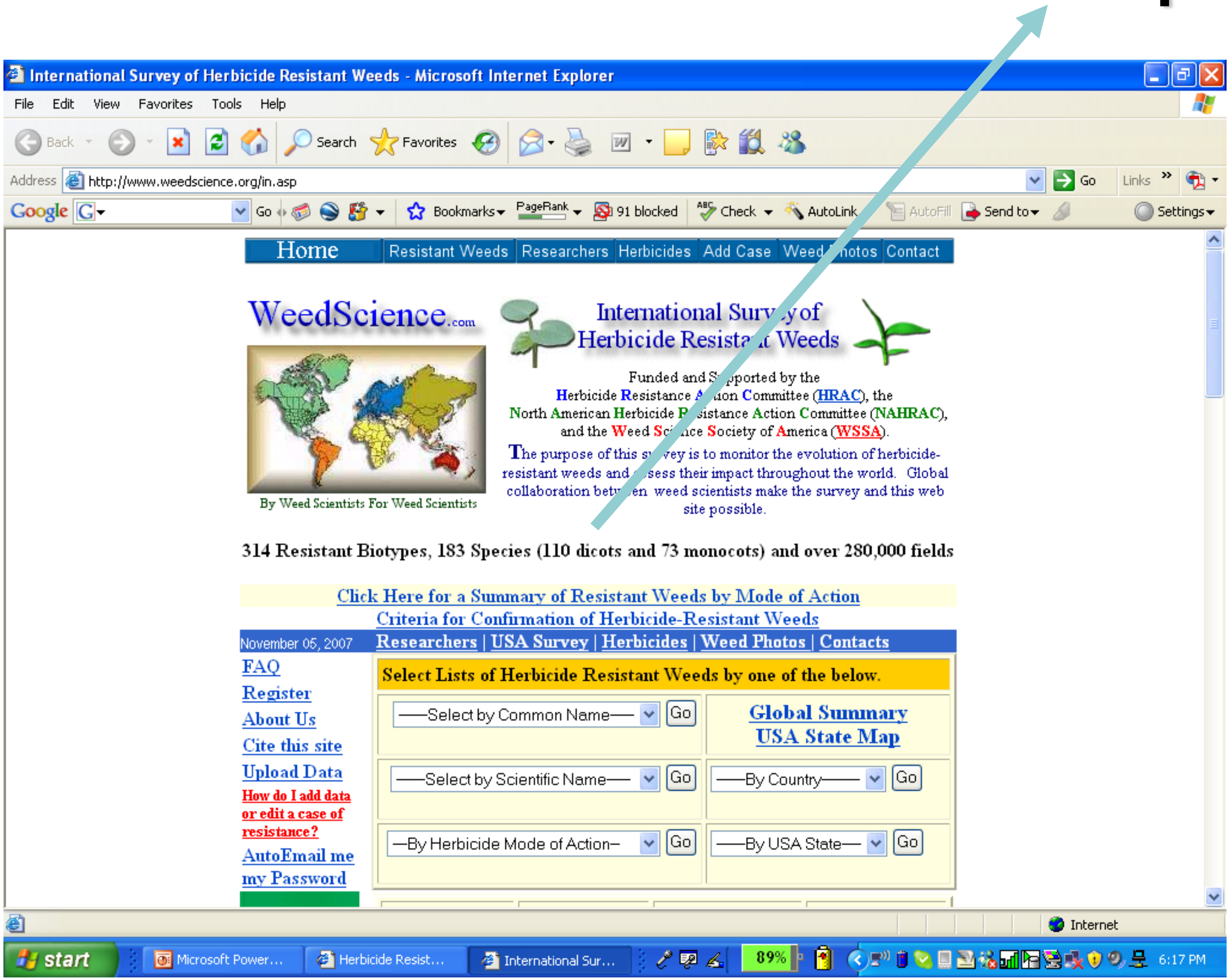
- **Insecticides: 1940's**
  - Use pattern
  - Insect distribution
- **Herbicides: 1950's**
  - Use pattern
- **Fungicides: 1970's**
  - Appearance of systemic fungicides
  - Use pattern

Arthropod pesticide resistance database includes >500 species



http://www.pesticideresistance.org/

Weed resistance database includes >250 species



http://www.weedscience.org/

Why is resistance difficult to detect?

- Resistant pests are mixed with susceptible pests while resistance is building up
- Resistant pests can spread from heavily sprayed regions or even close neighbors
- Market shifts, price, availability of pesticides may suddenly alter selection pressure for resistance
- % control is rarely measured or even noticed, and gradual decreases in efficiency may go unseen
- Not all pesticides give ‘miraculous’ control, and we do not have high expectations for them!

Other reasons why efficiency may be poor

- **Regional**
  - Unusually serious pest outbreak
  - Favorable weather for the pest
  - Unfavorable weather for the pesticide
- **Local**
  - Poor targeting of the spray
  - Poor calibration
  - Worn or inappropriate nozzles
  - Inefficient chemical AI, &/or poor storage conditions
  - Fewer natural enemies

INSECTICIDES AND MITICIDES

http://www.irac-online.org/

INSECTICIDE RISK FACTORS

- High pest reproduction (multiplication)
- Large number of generations a year
- High genetic variability
- Isolation, enclosure
- Low immigration by susceptible pests
- High proportion of population exposed
- Frequent applications

THE THREE INGREDIENTS FOR EVOLUTION, CAPACITY FOR INCREASE, HERITABLE VARIATION AND SELECTION PRESSURE

RESISTANCE MANAGEMENT

- **Minimize selection for resistance by one 'type' of insecticide**
    - **Sequences or rotation of 'Mode of Action' (MoA) groups**
    - **Apply each MoA group during one stage of crop growth or pest development**
    - **Avoid treating successive generations of pests with same MoA group**
- **Avoid spraying where possible; use IPM**
- **Predators and parasites do not select for resistance: they represent a non-specific MoA group**

Natural enemies are exposed to pesticides, as well as pests, but they rarely become resistant

WHY?




MAXIMIZE BIOLOGICAL PEST SUPPRESSION

To conserve pesticides!




Every 1% increase in pest resistance increases the job that natural enemies have to do

How many insecticide mode of action classes are there?

8, 18, 28, 48?

Insecticide Mode of Action Classification:
A key to effective insecticide resistance management

Insecticide Resistance Action Committee

IRAC website: www.irac-online.org

Introduction

IRAC promotes the use of a Mode of Action (MoA) classification of insecticides as the basis for effective and sustainable insecticide resistance management (IRM). Insecticides are allocated to specific groups based on their target site. Reviewed and re-issued periodically, the IRAC MoA classification list provides farmers, growers, advisors, extension staff, consultants and crop protection professionals with a guide to the selection of insecticides or acaricides in IRM programs. Effective IRM of this type preserves the utility and diversity of available insecticides and acaricides. A selection of MoA groups is shown below.

Effective IRM strategies: Alternations or sequences of MoA

All effective insecticide (and acaricide) resistance management (IRM) strategies seek to minimise the selection for resistance from any one type of insecticide or acaricide. In practice, alternations, sequences or rotations of compounds from different MoA groups provide sustainable and effective IRM. This ensures that selection from compounds in the same MoA group is minimised. Applications are often arranged into MoA spray windows or blocks that are defined by the stage of crop development and the biology of the pest(s) of concern. Local expert advice should always be followed with regard to spray windows and timings. Several sprays of a compound may be possible within each spray window but it is generally essential to ensure that successive generations of the pest are not treated with compounds from the same MoA group. Metabolic resistance mechanisms may give cross-resistance between MoA groups, and where this is known to occur, the above advice must be modified accordingly.

Moulting & Metamorphosis

Group 18 Ecdysone agonist / disruptor
Diacylhydrazines (e.g. Tebufenozide)
Group 7 Juvenile hormone mimics
JH analogues, Fenoxycarb, Pyriproxyfen, etc

Midgut

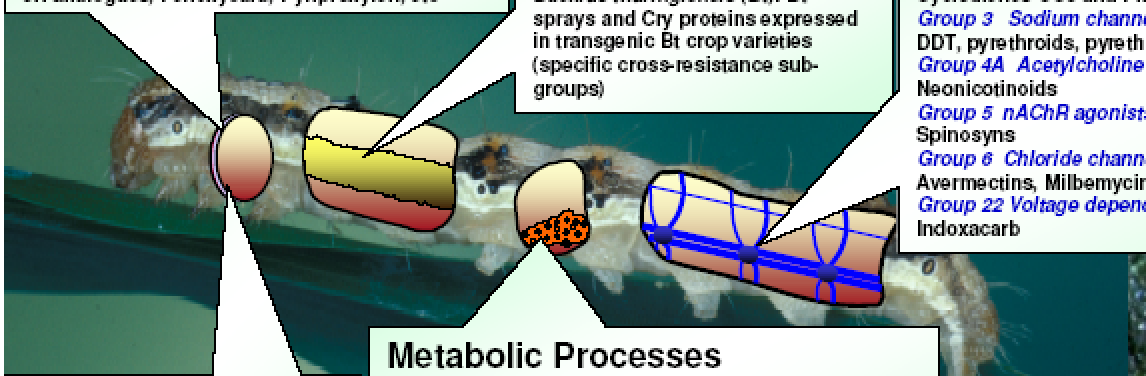
Group 11 Microbial disruptors of insect midgut membranes
Toxins produced by the bacterium *Bacillus thuringiensis* (Bt): Bt sprays and Cry proteins expressed in transgenic Bt crop varieties (specific cross-resistance sub-groups)

Nervous System

Groups 1A & B Acetylcholinesterase (AChE) inhibitors
Carbamates and Organophosphates
Group 2 GABA-gated chloride channel antagonists
Cyclodienes OCs and Phenylpyrazoles (Fiproles)
Group 3 Sodium channel modulators
DDT, pyrethroids, pyrethrins
Group 4A Acetylcholine receptor (nAChR) agonists
Neonicotinoids
Group 5 nAChR agonists (Allosteric) [not group 4A]
Spinosyns
Group 6 Chloride channel activators
Avermectins, Milbemycins
Group 22 Voltage dependent sodium channel blocker
Indoxacarb

Non-specific MoA

Group 9 Compounds of non-specific mode of action (selective feeding blockers)
Pymetrozine, Flonicamid, etc.



Cuticle Synthesis

Groups 15 and 16 Inhibitors of chitin biosynthesis
Benzoylureas (Lepidoptera and others), Buprofezin (Homoptera)

Metabolic Processes

Many groups acting on a wide range of metabolic processes including:
Group 12 Inhibitors of oxidative phosphorylation, disruptors of ATP
Diafenthiuron & Organotin miticides
Group 12 Uncouplers of oxidative phosphorylation via disruption of H proton gradient - Chlorfenapyr

Non-specific MoA

Group 10 Compounds of non-specific mode of action (mite growth inhibitors)
Clofentezine, Hexythiazox, Etoxazole

Metabolic processes

Group 20 Mitochondrial complex III electron transport inhibitors
Acequinocyl, Fluacrypyrim, etc
Group 21 Mitochondrial complex I electron transport inhibitors
Rotenone, METI acaricides
Group 23 Inhibitors of lipid synthesis
Tetronic acid derivatives

v4, October 2005

INSECTICIDE CLASSIFICATION

28 Mode of Action classes, plus ‘unknowns’

Class 1: Acetylcholine esterase inhibitors, 1A carbamates; 1B OP’s

Class 3: Sodium channel modulators, including pyrethroids

Class 4: Nicotinic Acetylcholine receptor agonists, 4A neonicotinoids

For apples and cherries: Kaiser et al, EM 8951 2008 OSU Extension

http://ir.library.oregonstate.edu/xmlui/bitstream/handle/1957/20525/em8951.pdf?sequence=1

FUNGICIDES

http://www.frac.info/frac/index.htm

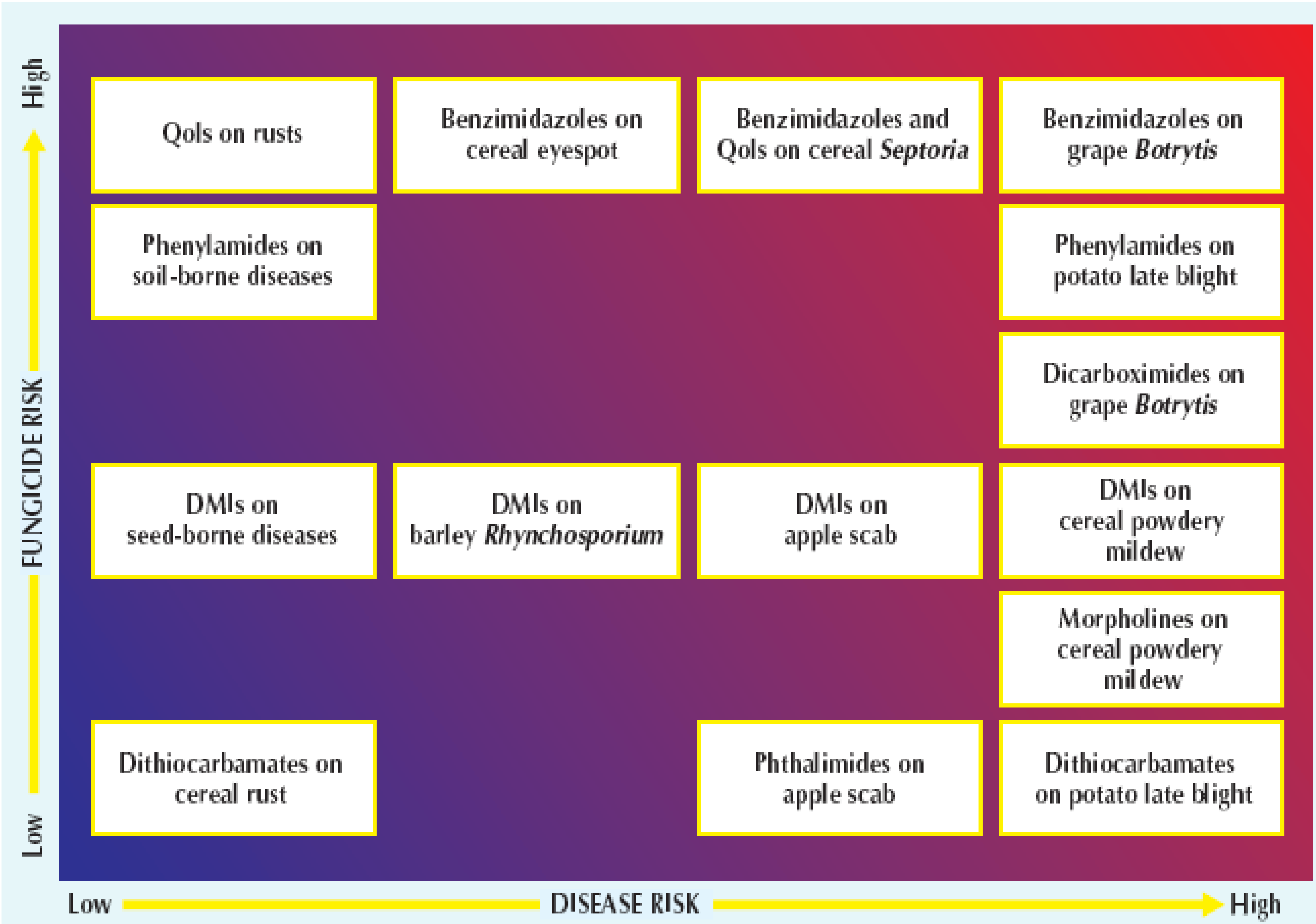
The time from marketing to onset of resistance is getting much shorter

Organomercurials, 40 years; Triphenyltins, 13 years; Carboxanelides, 15 years

vs

Quinone outside Inhibitors (e.g. Strobilurins), 2 years; Melanine biosynthesis inhibitors, 2 years

FUNGICIDE RISK FACTORS

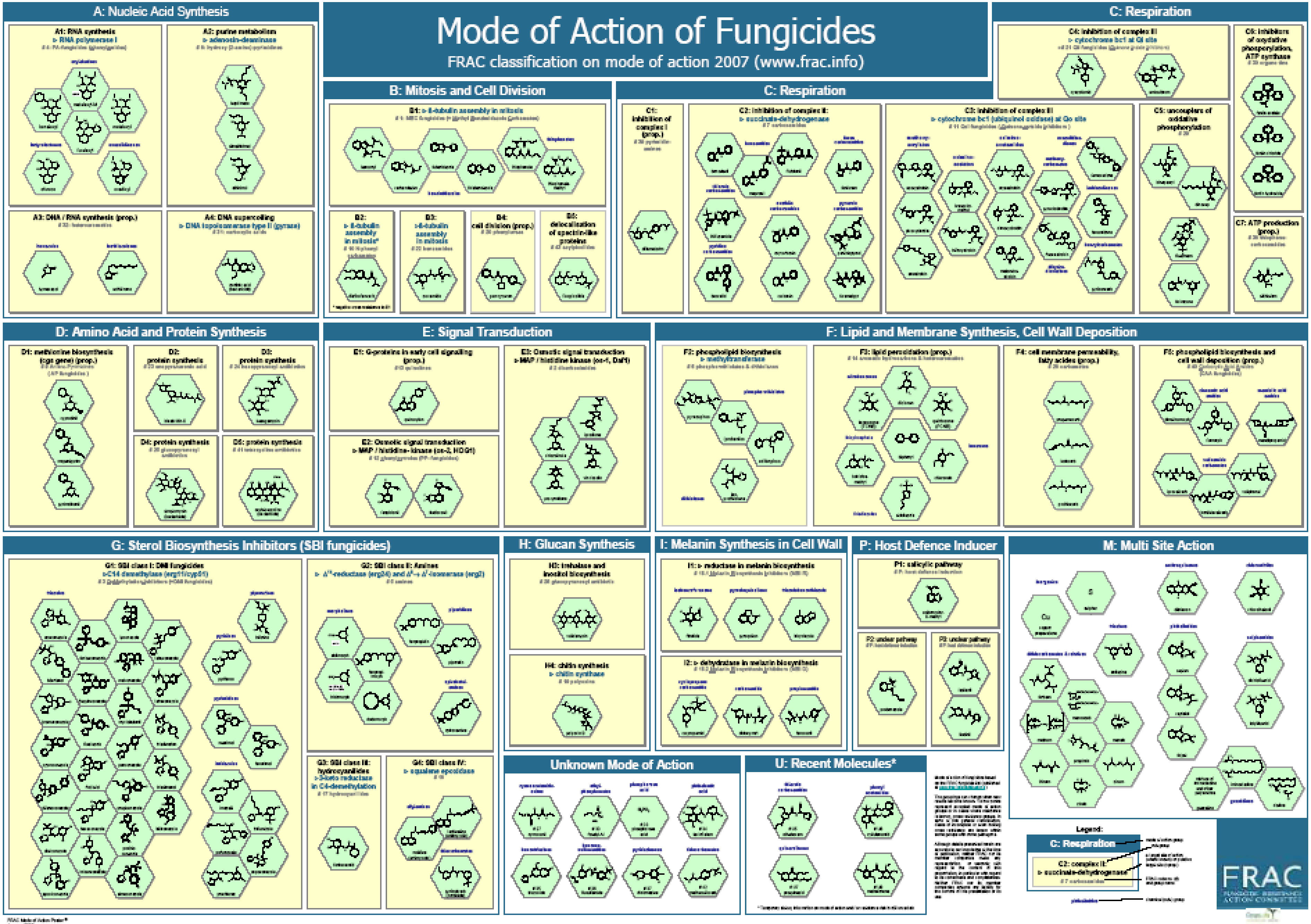


FUNGICIDE RESISTANCE MANAGEMENT

- Avoid excessive use
- Rotate/alternate MoA classes
- Use specialized mixtures or label instructions about resistance management strategies for different MoA groups
- Recommended dose rate
- Use thresholds, and IPM approaches

How many fungicide mode of action classes are there?

12, 22, 32, 42?


Mode of Action of Fungicides
FRAC classification on mode of action 2007 (www.frac.info)
A: Nucleic Acid Synthesis
B: Mitosis and Cell Division
C: Respiration
D: Amino Acid and Protein Synthesis
E: Signal Transduction
F: Lipid and Membrane Synthesis, Cell Wall Deposition
G: Sterol Biosynthesis Inhibitors (SBI fungicides)
H: Glucan Synthesis
I: Melanin Synthesis in Cell Wall
P: Host Defence Inducer
M: Multi Site Action
Unknown Mode of Action
U: Recent Molecules*
Legend:
FRAC

FUNGICIDE CLASSIFICATION

42 Mode of Action classes, plus unknowns

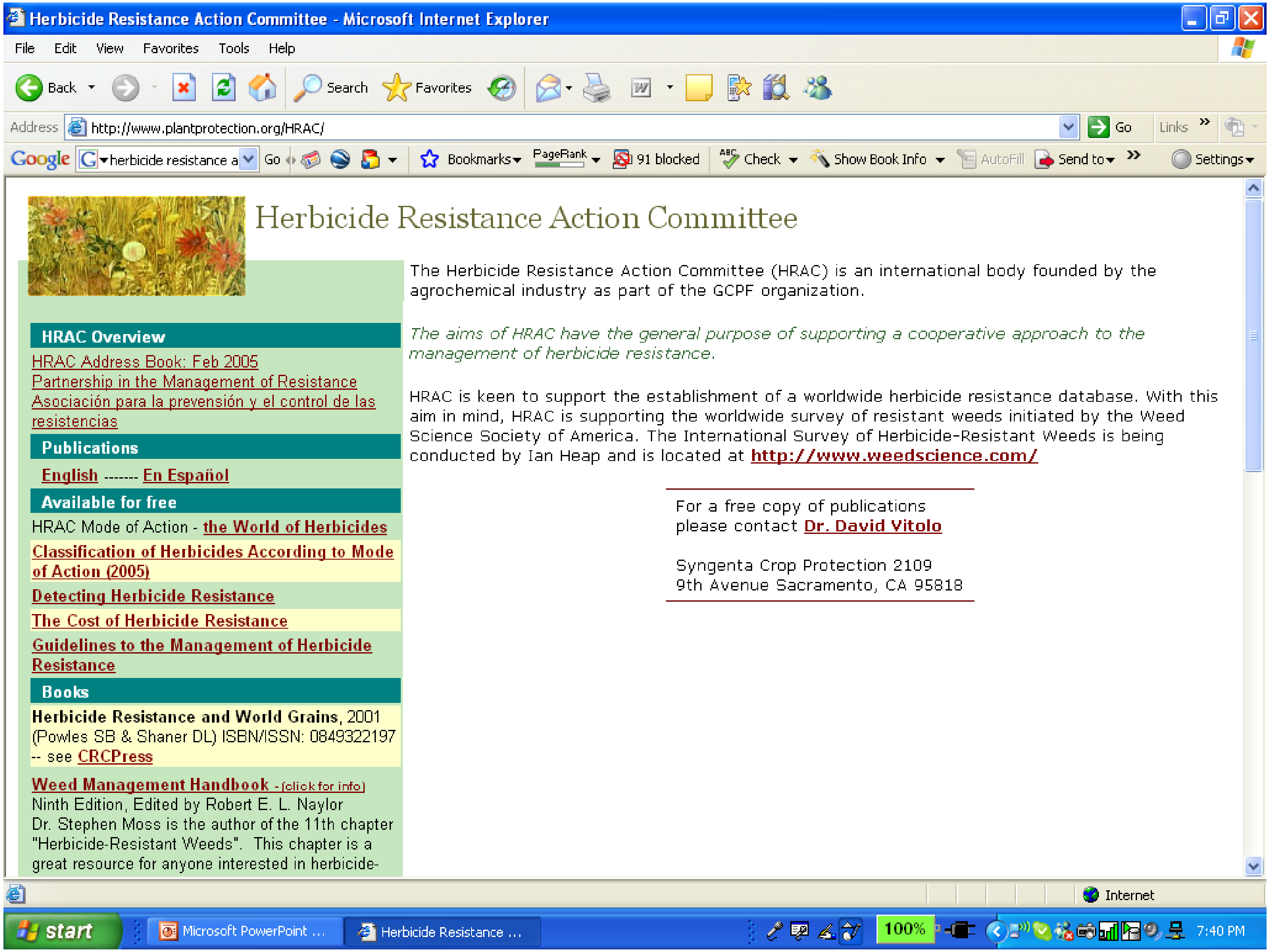
HIGH RISK EXAMPLES

1, Beta-tubuline assembly in mitosis. 'MBC fungicides', incl. benzimidazoles (benomyl)

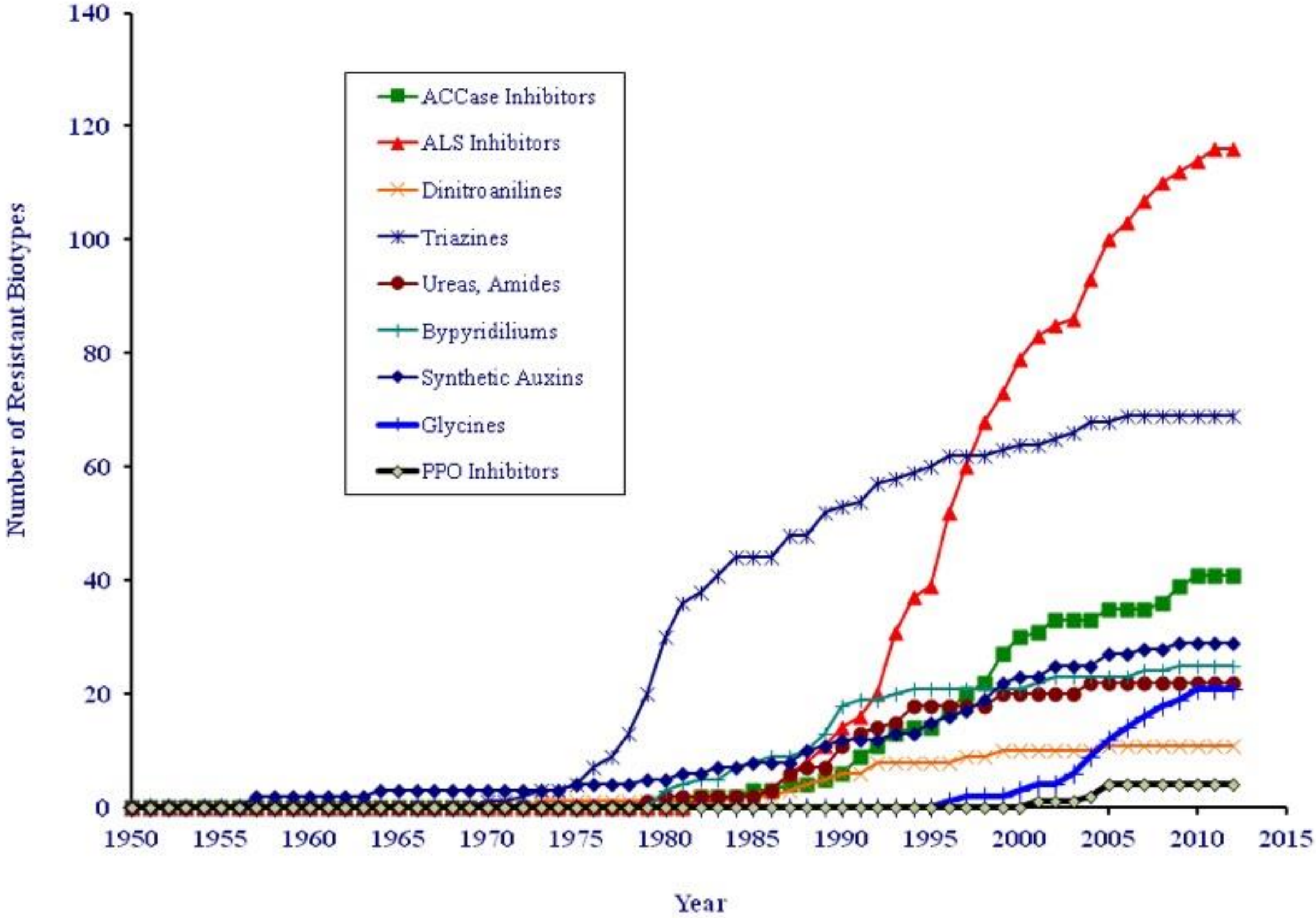
2, MAP/Histidine-Kinase in osmostic signal transduction. 'Dicarboximides', incl. vinclozin

11, Complex III: cytochrome bc1 at Qo site. 'QoI fungicides', incl. methoxy acrylates (azoxystrobin)

HERBICIDES



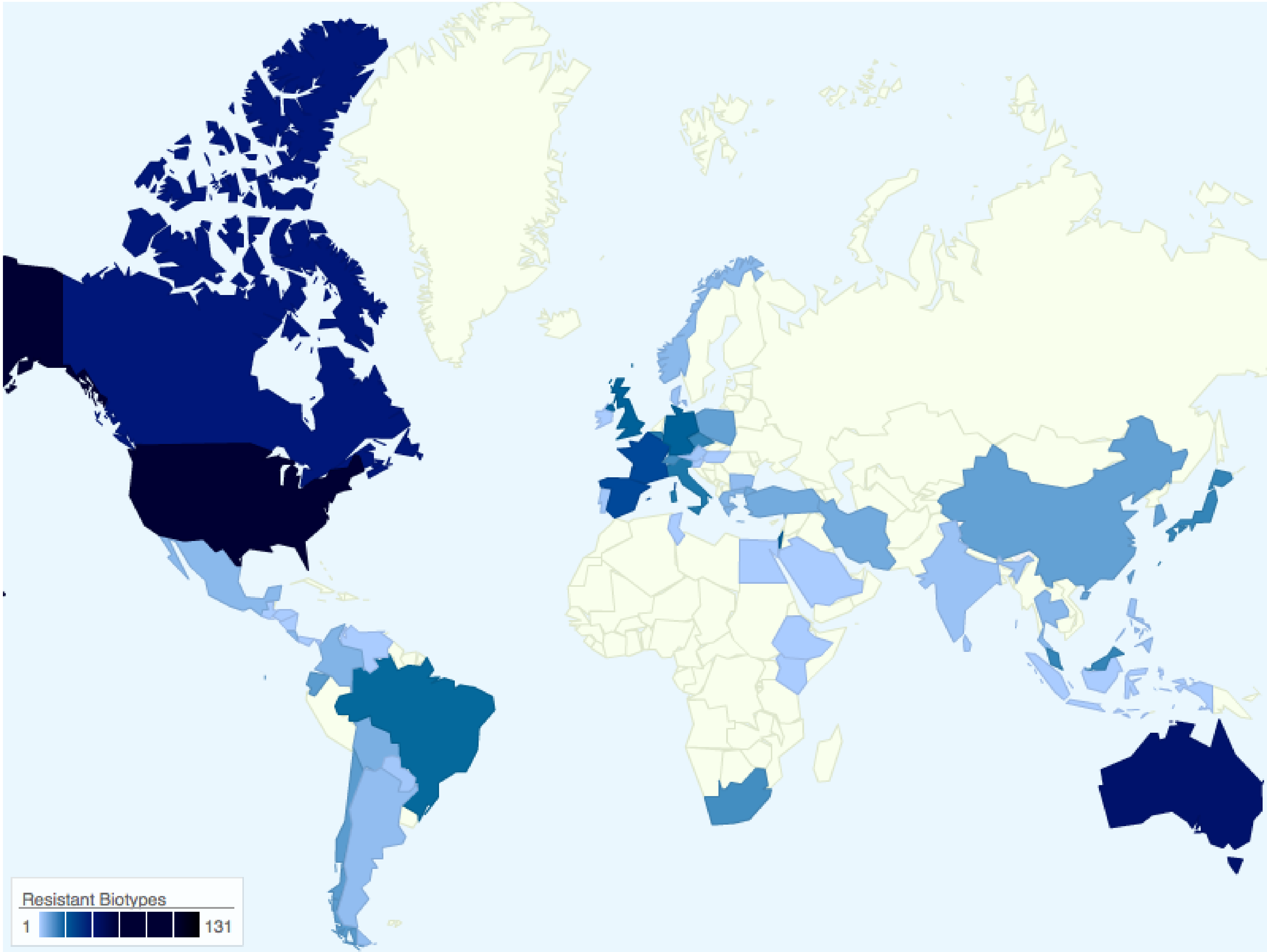
http://www.plantprotection.org/HRAC/



Source: Ian Heap
http://www.weedscience.com

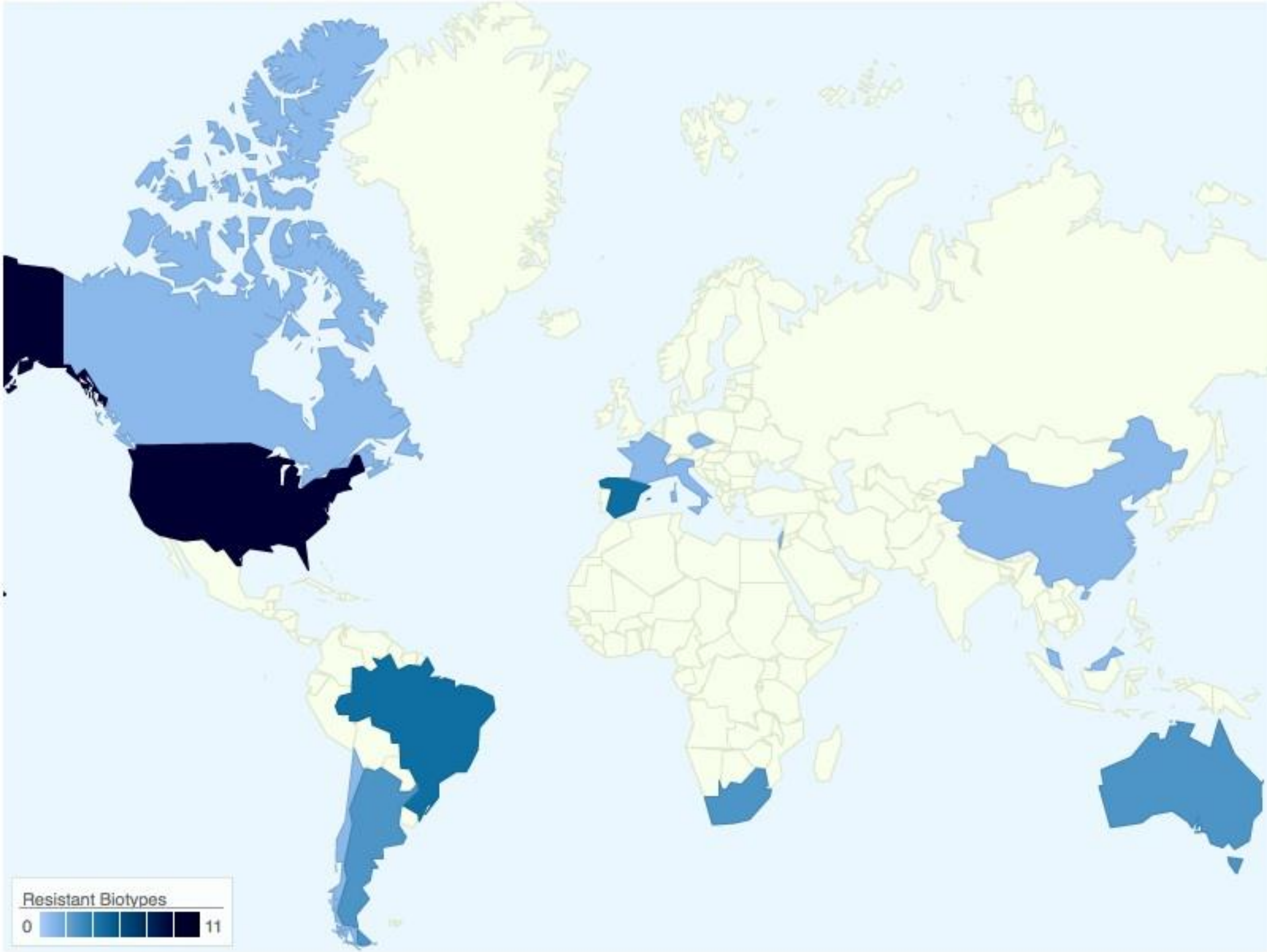
Herbicide Resistant Weeds Globally - 2010 - www.weedscience.com

Mouse over countries to see numbers of resistant weeds.



Glyphosate-Resistant Weeds Globally - 2010 - www.weedscience.com

Mouse over countries to see numbers of resistant weeds.



HERBICIDE RISK FACTORS

	LOW	MODERATE	HIGH
AI mix or rotation	*>2 MoA*	*2*	*1*
Weed control	*Cultural, mechanical + chem*	*Cultural and chem*	*Chem only*
Use of same MoA/season	*Once*	*>1*	*Many*
Cropping system	*Full rotation*	*Limited*	*None*
Resistance to MoA	*Unknown*	*Limited*	*Common*
Weed infestation	*Low*	*Moderate*	*High*
Control last 3 years	*Good*	*Declining*	*Poor*

HERBICIDE RESISTANCE MANAGEMENT

- Crop rotation
  - Enables herbicide rotation
  - Disrupts weed growing season
  - Different cultural measures
  - Variable competition with weeds
- Cultural methods
  - Bury non-germinated seeds
  - Delay planting to enable non-selective herbicide use
  - Weed free seed
  - Seed predators
- Herbicide rotation (NB some weeds resistant to several classes)

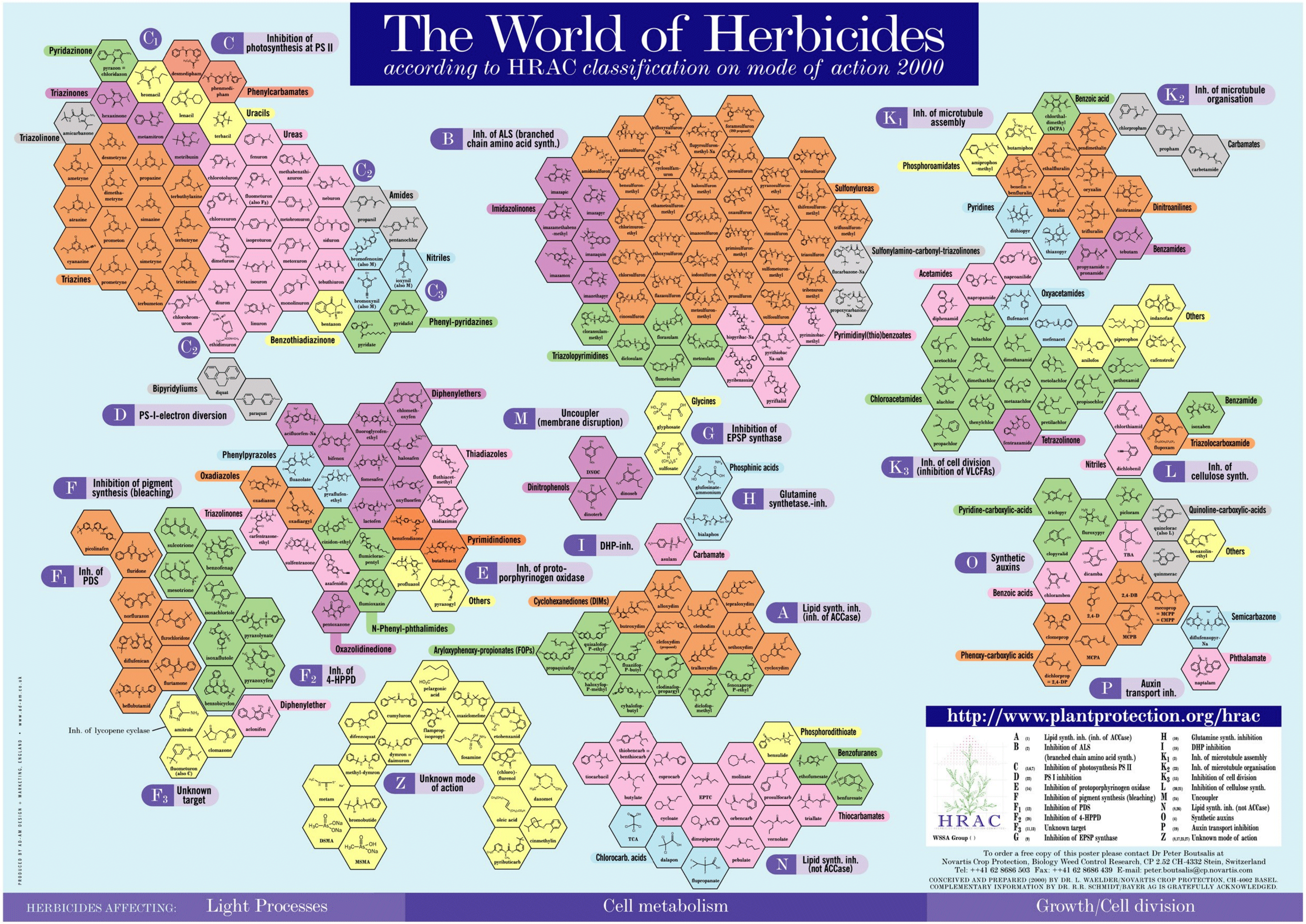
E.g. Selected resistant weeds in OR

- Kochia, prickly lettuce, Russian thistle, annual bluegrass (Group 2 sulfonylurea: e.g. Glean, Amber, Ally)
- Wild oat and Italian Ryegrass (Group 1 ACCase inhibitors: e.g. Discover; G 9)
- Powell amaranth and other pigweeds (Group 5, P. system II inhibitors: traizines: e.g. Atrazine)
- Yellow starthistle (Group 4 Synthetic auxins: e.g. Tordon)
- Wild oat (Far-Go (Group 8), Avenge (Group 26)

http://weedscience.org/Summary/Country.aspx

How many herbicide mode of action classes are there?

8, 18, 28, 38?


The World of Herbicides
according to HRAC classification on mode of action 2000
C Inhibition of photosynthesis at PS II
C1
C2
C3
Pyridazinone
Triazinones
Triazolinone
Triazines
Phenylcarbamates
Uracils
Ureas
Amides
Nitriles
Benzothiadiazinone
Phenyl-pyridazines
B Inh. of ALS (branched chain amino acid synth.)
Imidazolinones
Sulfonylureas
Sulfonylamino-carbonyl-triazolinones
Triazolopyrimidines
Pyrimidinyl(thio)benzoates
K1 Inh. of microtubule assembly
K2 Inh. of microtubule organisation
Benzoic acid
Carbamates
Phosphoroamidates
Pyridines
Dinitroanilines
Benzamides
Acetamides
Oxyacetamides
Others
Chloroacetamides
Tetrazolinone
K3 Inh. of cell division (inhibition of VLCFAs)
Benzamide
Triazolocarboxamide
Nitriles
L Inh. of cellulose synth.
D PS-I-electron diversion
Bipyridyliums
Diphenylethers
Phenylpyrazoles
Thiadiazoles
Oxadiazoles
Triazolinones
Pyrimidindiones
E Inh. of proto-porphyrinogen oxidase
Others
N-Phenyl-phthalimides
Oxazolidinedione
F Inhibition of pigment synthesis (bleaching)
F1 Inh. of PDS
F2 Inh. of 4-HPPD
F3 Unknown target
Diphenylether
Inh. of lycopene cyclase
M Uncoupler (membrane disruption)
Dinitrophenols
Glycines
G Inhibition of EPSP synthase
Phosphinic acids
H Glutamine synthetase.-inh.
I DHP-inh.
Carbamate
Cyclohexanediones (DIMs)
A Lipid synth. inh. (inh. of ACCase)
Aryloxyphenoxy-propionates (FOPs)
Z Unknown mode of action
Phosphorodithioate
Benzofuranes
Thiocarbamates
Chlorocarb. acids
N Lipid synth. inh. (not ACCase)
Pyridine-carboxylic-acids
Quinoline-carboxylic-acids
O Synthetic auxins
Others
Benzoic acids
Semicarbazone
Phenoxy-carboxylic acids
Phthalamate
P Auxin transport inh.
http://www.plantprotection.org/hrac
A (1) Lipid synth. inh. (inh. of ACCase)
B (2) Inhibition of ALS (branched chain amino acid synth.)
C (5,6,7) Inhibition of photosynthesis PS II
D (22) PS I inhibition
E (14) Inhibition of protoporphyrinogen oxidase
F Inhibition of pigment synthesis (bleaching)
F1 (12) Inhibition of PDS
F2 (28) Inhibition of 4-HPPD
F3 (11,13) Unknown target
G (9) Inhibition of EPSP synthase
H (10) Glutamine synth. inhibition
I (18) DHP inhibition
K1 (3) Inh. of microtubule assembly
K2 (23) Inh. of microtubule organisation
K3 (15) Inhibition of cell division
L (20,21) Inhibition of cellulose synth.
M (24) Uncoupler
N (8,26) Lipid synth. inh. (not ACCase)
O (4) Synthetic auxins
P (19) Auxin transport inhibition
Z (8,17,25,27) Unknown mode of action
HRAC
WSSA Group ( )
To order a free copy of this poster please contact Dr Peter Boutsalis at
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CONCEIVED AND PREPARED (2000) BY DR. L. WAELDER/NOVARTIS CROP PROTECTION, CH-4002 BASEL.
COMPLEMENTARY INFORMATION BY DR. R.R. SCHMIDT/BAYER AG IS GRATEFULLY ACKNOWLEDGED.
HERBICIDES AFFECTING: Light Processes
Cell metabolism
Growth/Cell division

HERBICIDE CLASSIFICATION

28 MoA classes, plus unknowns

- Group 2, Inhibition of acetolacetate synthase, incl. sulfonylureas (chlorsulfuron)
- Group 5, Inhibition of photosynthesis at photosystem II, incl. triazines (atrazine)

PNW 437: Herbicide resistant weeds and their management (Hulting et al)

IF CONTROL FAILURE IS CONFIRMED WITH A HERBICIDE

- **Eradicate remaining weed population to limit build up in soil**
- **Limit field to field movement**
- **Avoid the herbicide to which resistance confirmed**
- **Consider grazing or cutting for feed (avoid spreading manure)**
- **Select field for rotation or set aside**
- **Develop a long-term plan for weed management**

CONCLUSIONS

- Good IPM practices extend chemical life in the market
- Knowledge of MoA class is useful
- Maintain records and develop a rotation strategy
- Heed local advice

IPM that limits selection pressure for resistance	Impact on resistance	Impact on efficacy	

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SOURCE REDUCTION (only use pesticides when needed)			

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