Agriculture, insects and hormesis: evidence and considerations for study

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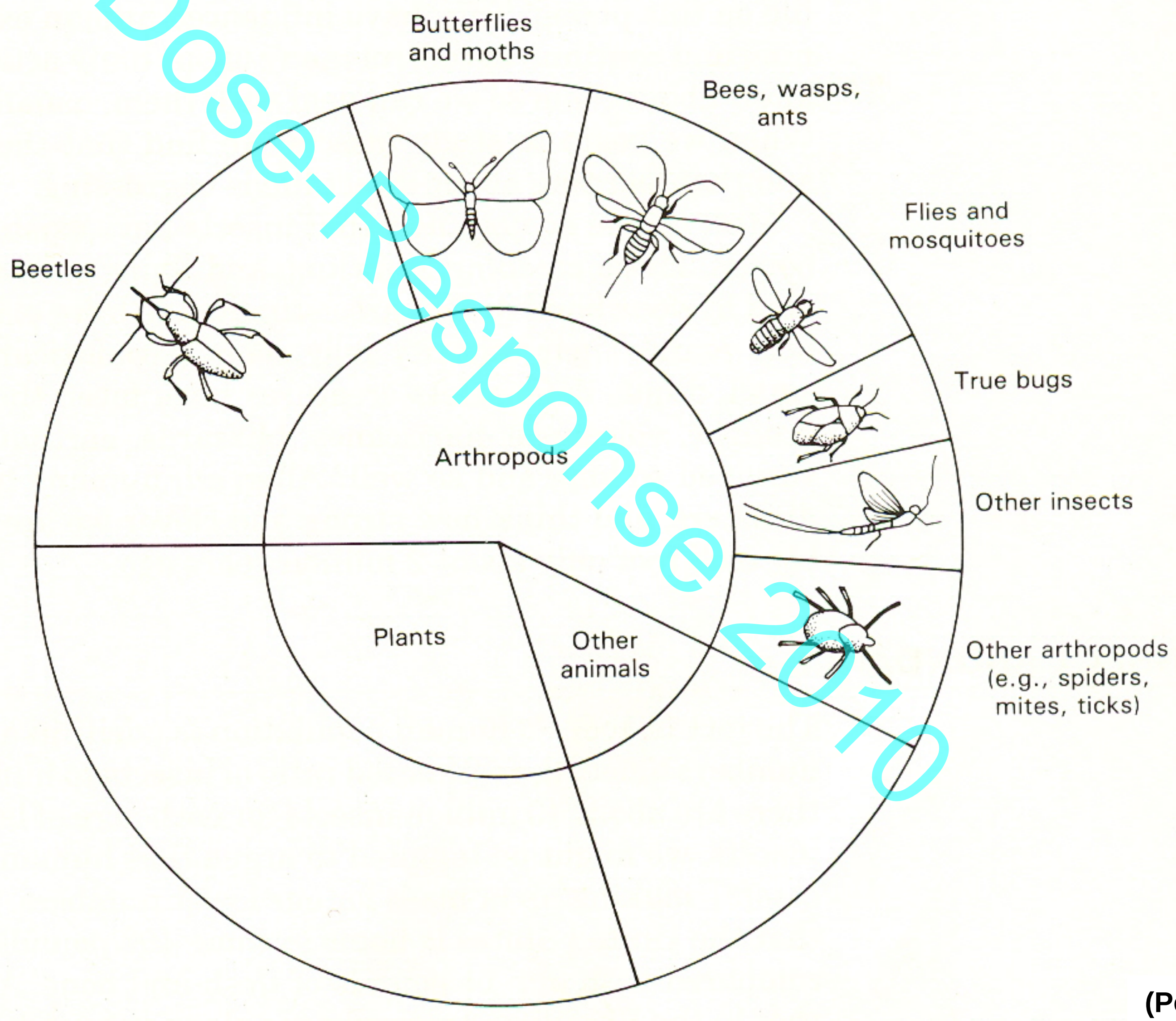
The insect world



(Pedigo 2006)

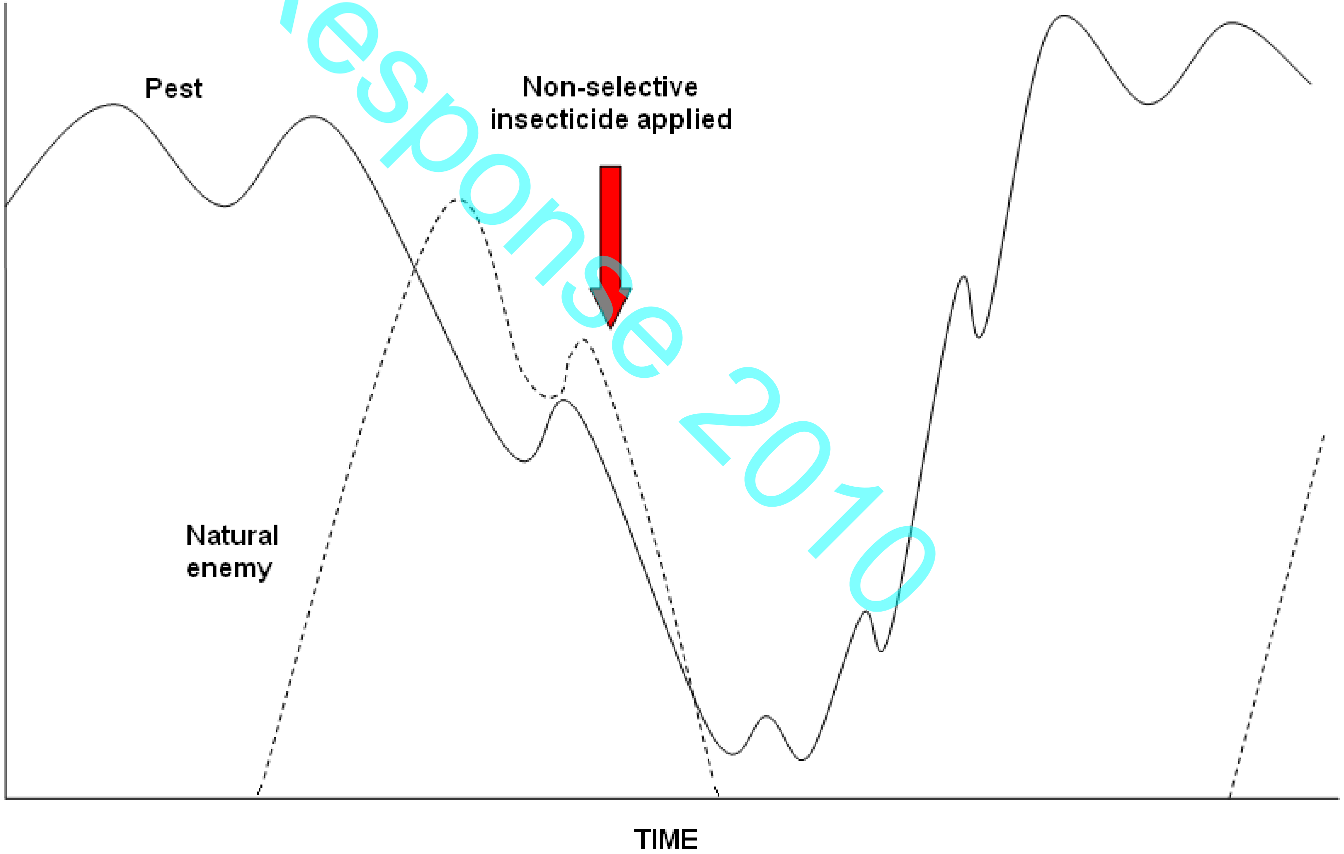
Insecticides in agriculture

- DDT 1939
- 560 million kg of insecticide used in 2001; 75% in agriculture

Pest population "explosions"

- Traditionally thought to be due to natural enemy (NE)/competition elimination
- Hormesis - an alternate/additional mechanism?

Theory of NE elimination



Hormesis – relevance for insects

- Spatial and temporal shifts in exposure concentrations
  - Drift
  - Residue degradation
  - Plant growth, poor coverage

- Consequences of pest population stimulation:
  - increased crop/commodity damage
  - additional pesticide treatments → exacerbation of:
    - non-target impacts
    - insecticide resistance development
    - environmental contamination

Population stimulation in the field

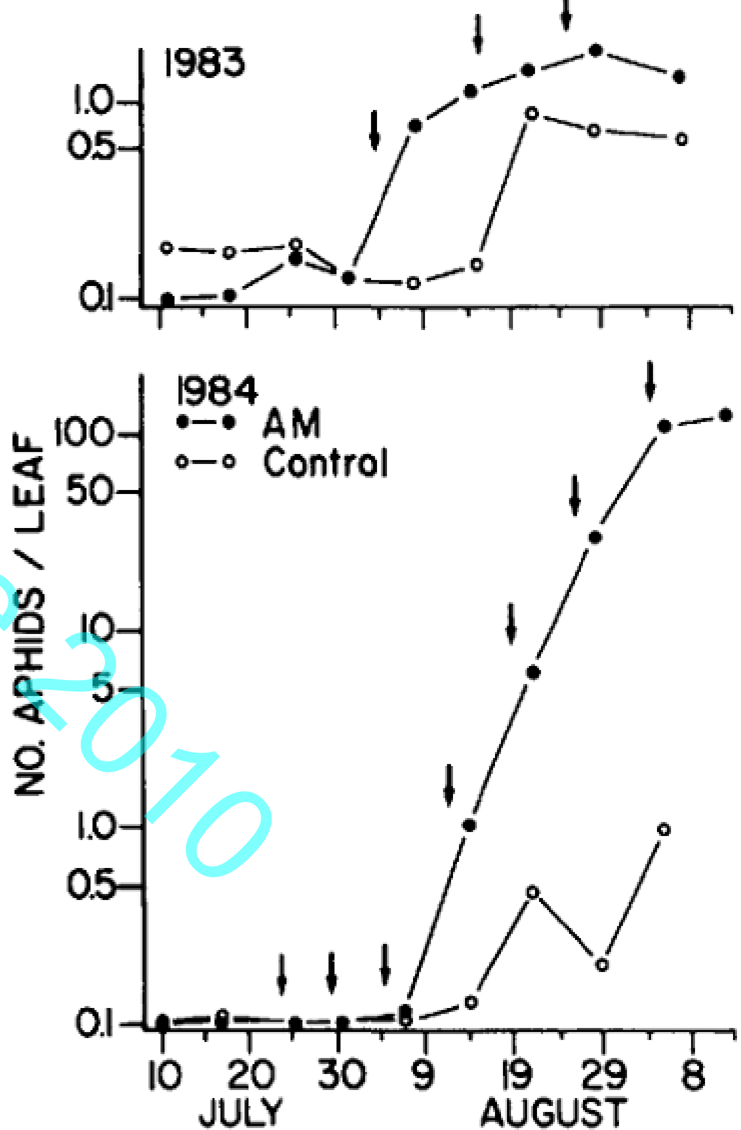
- Many examples with insects and mites
- E.g. Azinphosmethyl and *Myzus persciae* (Lowery ad Sears 1986)

Table 2. Average number of offspring produced per day for GPA collected from AM-treated or untreated potato plots and reared in the laboratory on potato leaf disks

Generation	Aphid treatment	No. ♀♀	$\bar{x}$ offspring per day	$\bar{x}$ generation
Parental A	AM	19	3.0a	2.6
	CK	19	2.1b	
Parental B	AM	22	3.8a	3.4
	CK	22	3.1b	
1st generation	AM	17	4.2a	4.3
	CK	15	4.3a	
2nd generation	AM	21	4.9a	4.7
	CK	22	4.5a	

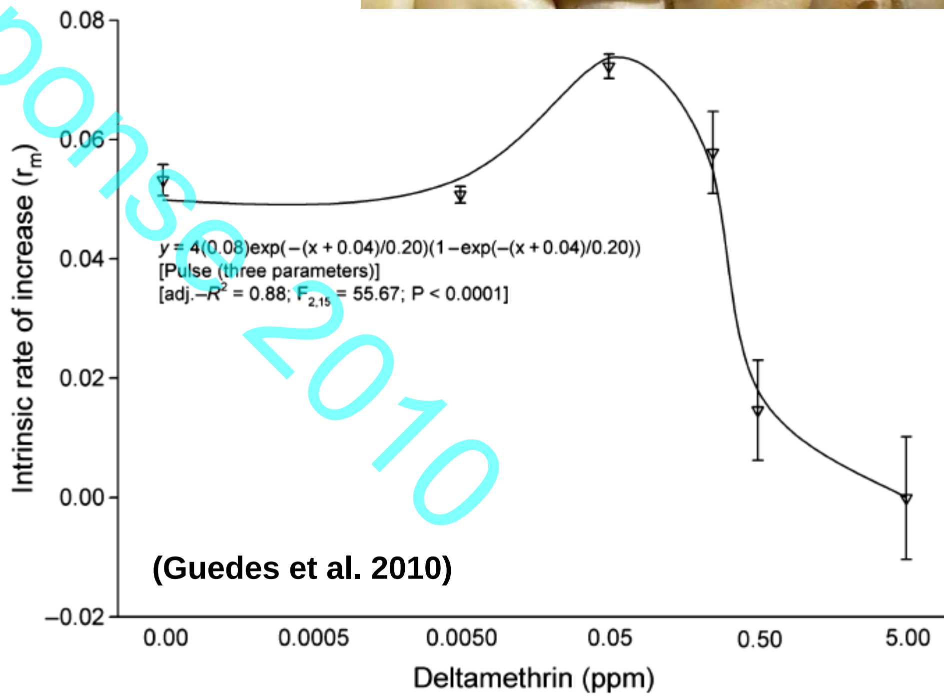
(Lowery and Sears 1986a)

(Lowery and Sears 1986b)

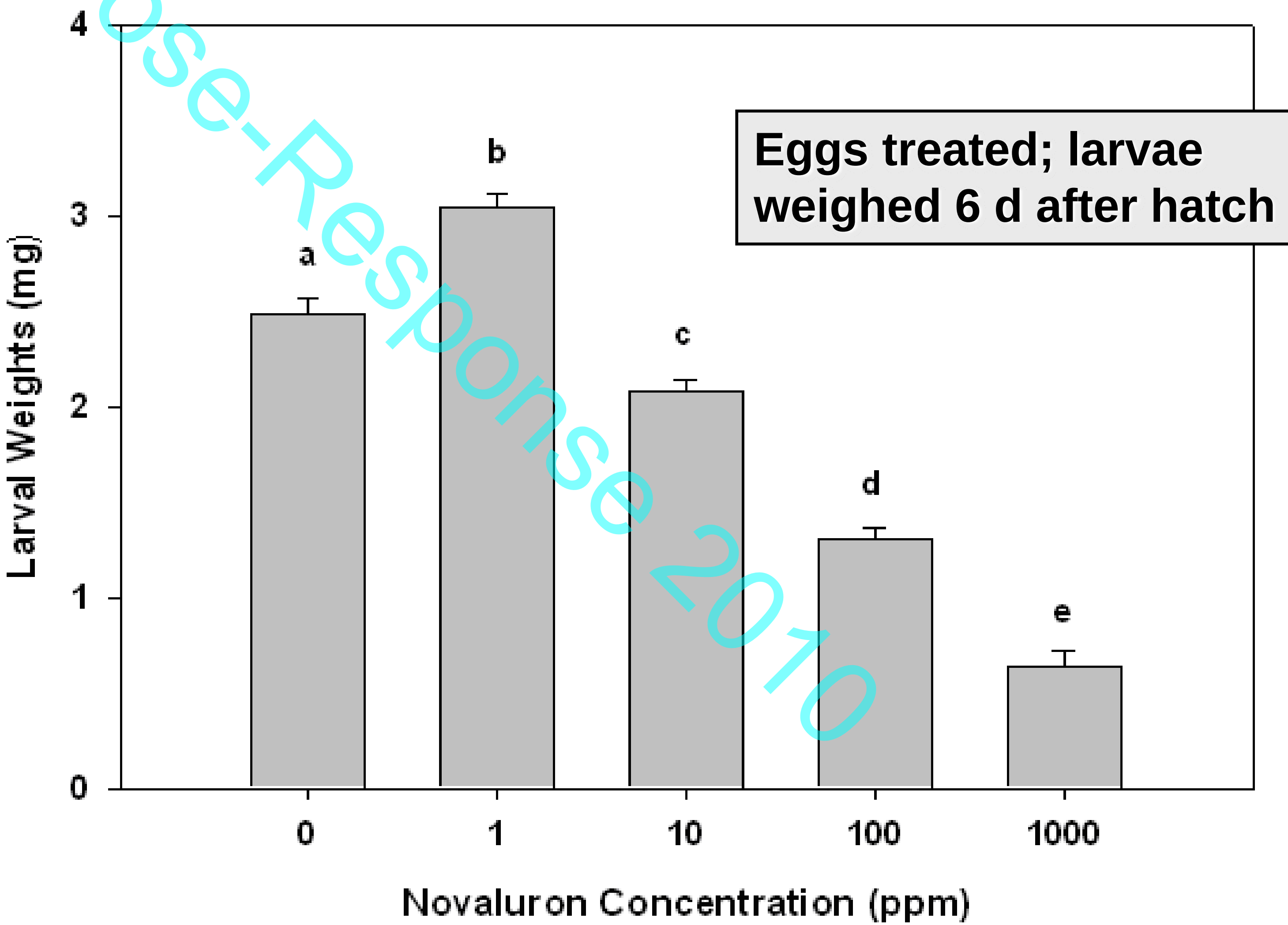


Insecticide resistance and hormesis

- > 100-fold reduced susceptibility not uncommon
- High-dose to a susceptible population may be a low-dose to resistant populations
- Hormetic response may boost resistant populations and increase frequency of the resistance alleles

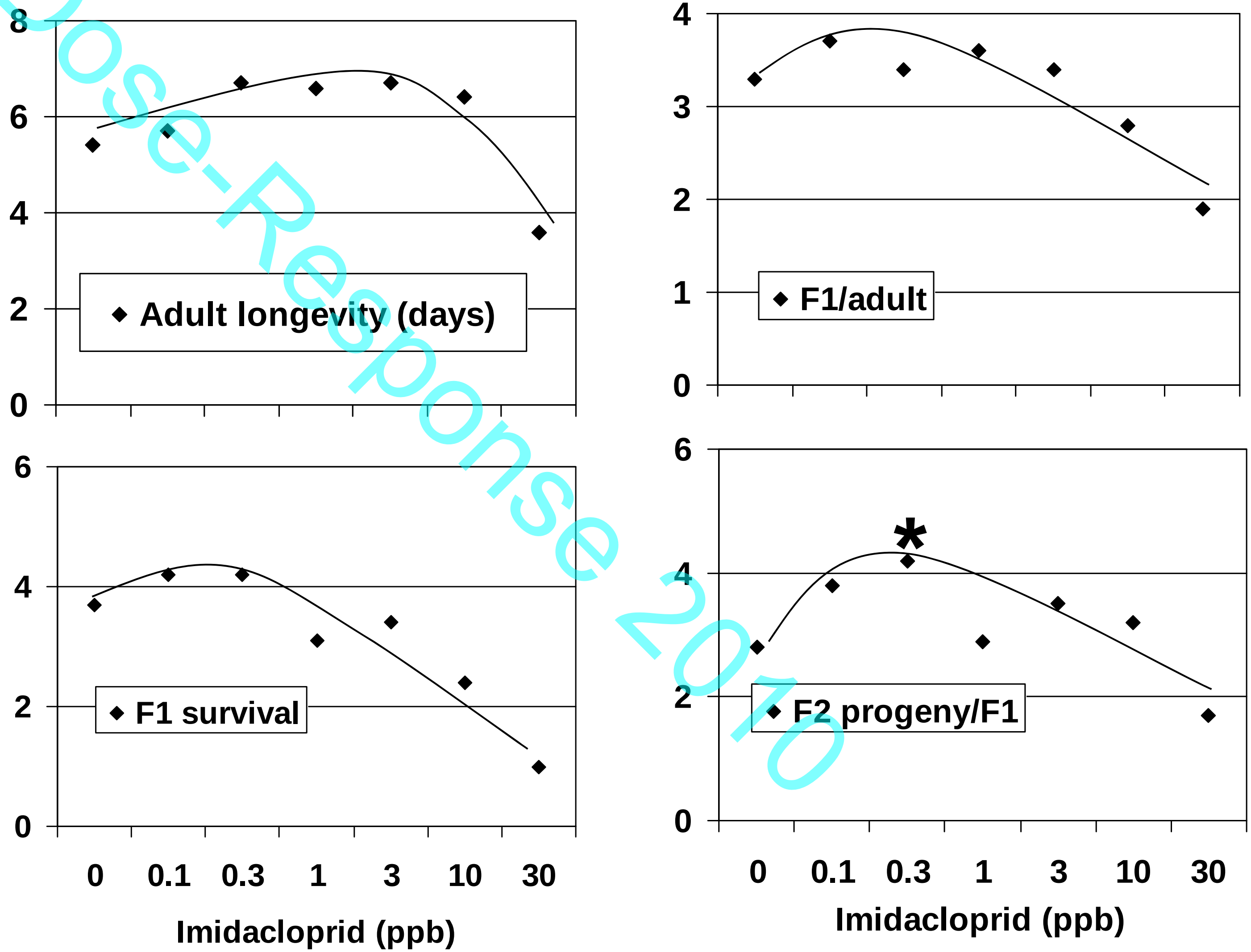


Novaluron and Colorado potato beetle



(Cutler et al. 2005)

Green peach aphid and imidacloprid



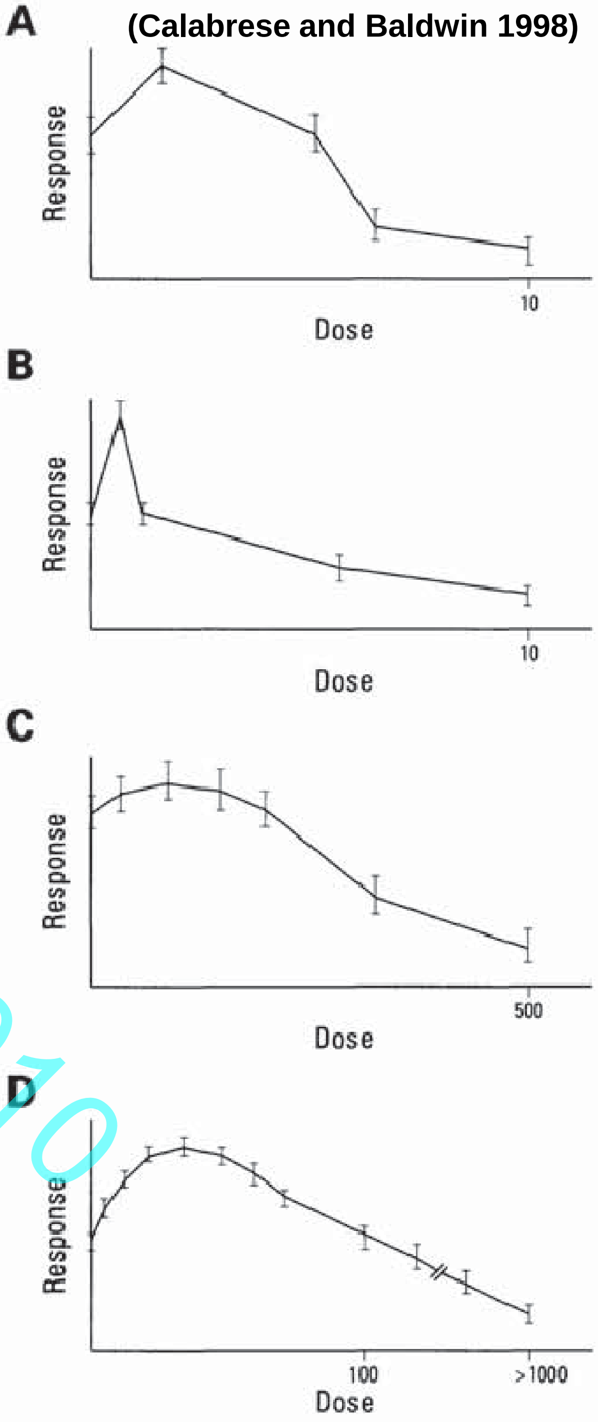
(Cutler et al. 2009)

Experiment considerations

- Stimulatory effects of insecticides often reported, e.g. reproduction, longevity, weight, population growth (see Cohen 2006)

- Most experiments with insects have experimental shortcoming precluding “true” designation of hormesis
  - Too few doses
  - No or few sub-NO(A)EC
  - Inadequate replication
  - No time component

- Use “hormesis” loosely in this talk



(Calabrese and Baldwin 1998)

Curiosities and Opportunities for Study

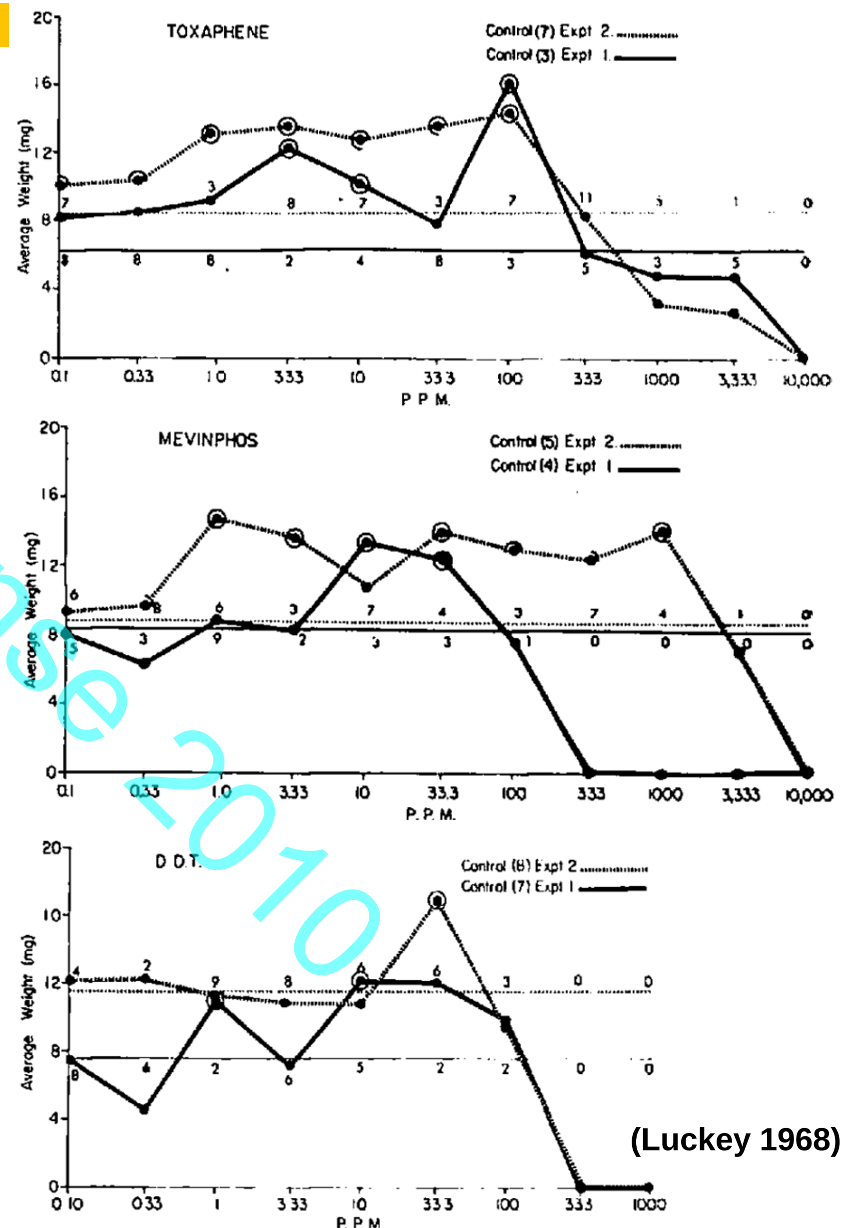
Insect hormesis semantics

- “Hormesis”
- “Hormoligosis” (Luckey 1963, 1968)
- “Pesticide-mediated homeostatic modulation (PMHM)” (Cohen 2006)

Hormoligosis

- “…. *minute quantities of any stressing agent (chemical, physical, psychological or social) would be stimulatory…under a wide variety of conditions, whereas larger quantities of stressing agent would be harmful to the same organism.*” (Luckey 1963)

- “*…subharmful quantities of many stress agents may be helpful when presented to organisms in* ***suboptimal environments***” (Luckey 1968)



(Luckey 1968)

Pesticide-mediated homeostatic modulation

- Cohen 2006
  - "Hormesis, however, cannot be claimed for cases in which the observed stimulatory effects were due to exposure of non-target pests (i.e., mites) to pesticides (DDT, carbaryl, insecticidal pyrethroids or imidacloprid). *Pesticides applied to **non-target** pests cannot be regarded as stressors since inhibition or mortality at very high doses can hardly be observed and measured.*" (emphasis is mine)
- E.g. mites – DDT, methyl parathion

Pesticide-mediated homeostatic modulation

- DDT **<u>is</u>** toxic to *T. urticae* (e.g. (Attiah and Boudreaux 1964)

Table 1.—Summary of oviposition by mites exposed to DDT under various conditions.

Species used and type of treatment	Average eggs laid/female: Parent generation	Average eggs laid/female: F_1 generation
1. *T. urticae*, exposed on treated paper		
0.1% E.C. for 2 minutes	93.8 (10)[1]	110.6 (16)
1.0% E.C. for 2 minutes	100.0 (8)	111.5 (11)
Control	126.7 (10)	102.8 (15)
1.0% E.C. for 10 minutes	76.9 (10)	97.3 (7)
0.1% E.C. for 10 minutes	98.3 (11)	77.3 (7)
Control	101.5 (12)	79.9 (7)
2. *T. urticae*, untreated on treated cut plants		
0.1% E.C.	74.1 (16)	86.9 (17)
1.0% E.C.	83.0 (9)	79.5 (15)
Control	112.8 (17)	93.6 (16)
3. *T. urticae*, treated on treated cut plants		
0.05% W.P.	106.8 (16)	94.7 (14)
0.1% W.P.	104.8 (15)	99.3 (16)
1.0% W.P.	Killed or lost (34)	91.7 (8)
Control	111.1 (13)	98.5 (16)
4. *T. urticae*, treated, on untreated cut plants[2]		
1.0% W.P.	40.7 (14)	
0.25% E.C.	34.5 (8)	
0.5% E.C.	Killed or lost (20)	
1.0% E.C.	Killed or lost (20)	
Control	35.4 (15)	
5. *T. urticae*, treated, held outside[2]		
0.05% E.C. on untreated plants	28.0 (6)	
0.1% E.C. on untreated plants	25.8 (6)	
0.25% E.C. on untreated plants	34.0 (3)	
Control	23.5 (8)	
0.05% on treated plants	Killed or lost (8)	
0.1% on treated plants	Killed or lost (8)	
0.25% on treated plants	Killed or lost (8)	

Pesticide-mediated homeostatic modulation

- Methyl parathion and permethrin **<u>are</u>** toxic to spider mites
- The dose makes the poison, not the name or the target organism
    - "High dose", "non-target", etc. are relative terms
    - Designation of "hormesis" should be based on the nature of response

(adapted from Ayyappath et al. 1997)

Pesticide	n	Slope (SEM)	LC (95% CL) mg AI/ml			
			LC_{05}	LC_{10}	LC_{25}	LC_{50}
Permethrin	1233	1.6 (0.16)	0.01 (0.003-0.03)	0.02 (0.01-0.04)	0.06 (0.03-0.08)	0.14 (0.1-0.2)
Methyl parathion	1198	2.3 (0.29)	9.8 (0.39-21.3)	14.05 (1.01-26.79)	25.50 (4.82-39.90)	49.48 (24.95-68.46)

NOAEC Doses?

- Do all chemical stressors induce hormesis?
- Stimulation observed at doses well above the NOAEC → different than hormesis?

Table 2.—Effect of lethal doses of insecticide on the number of progeny of *N. lugens*.

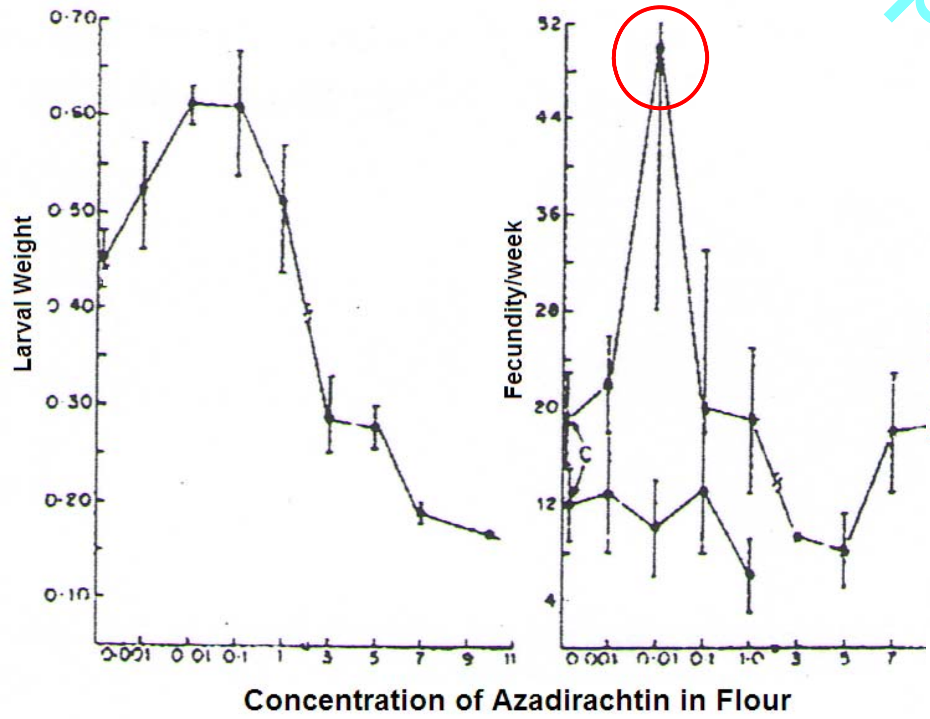
(Chelliah et al. 1980)

Lethal dose [1] (LD)	No. of nymphs hatched [2]		
	Decamethrin	Methyl parathion	Perthane
5	232.8b	147.3b	160.0a
10	198.0bc	147.5b	126.5a
25	214.0bc	247.3a	111.3a
50	287.5a	180.8b	159.5a
Control	163.8c	137.0b	134.0a

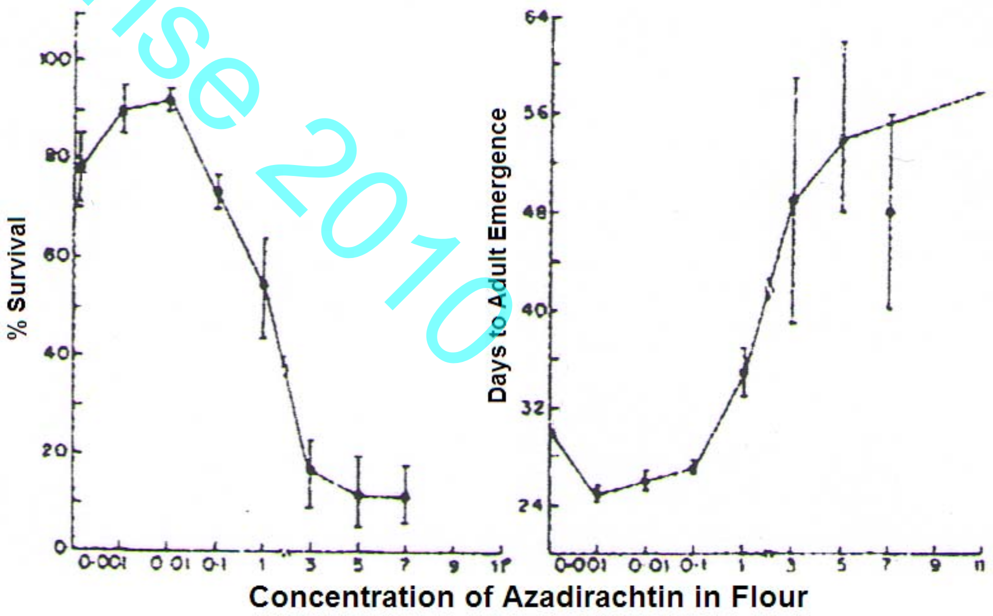
Magnitude of response

- Rarely is the magnitude of response greater than two-fold the control; generally 30%–60% greater than control (Calabrese and Baldwin 1988)

- Much greater stimulation may occur

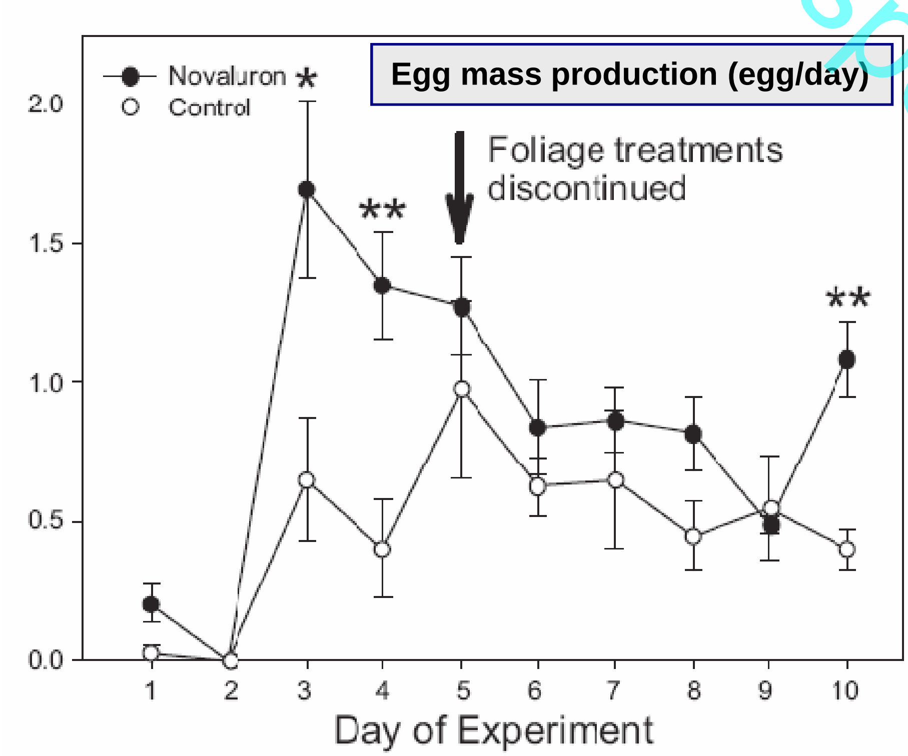


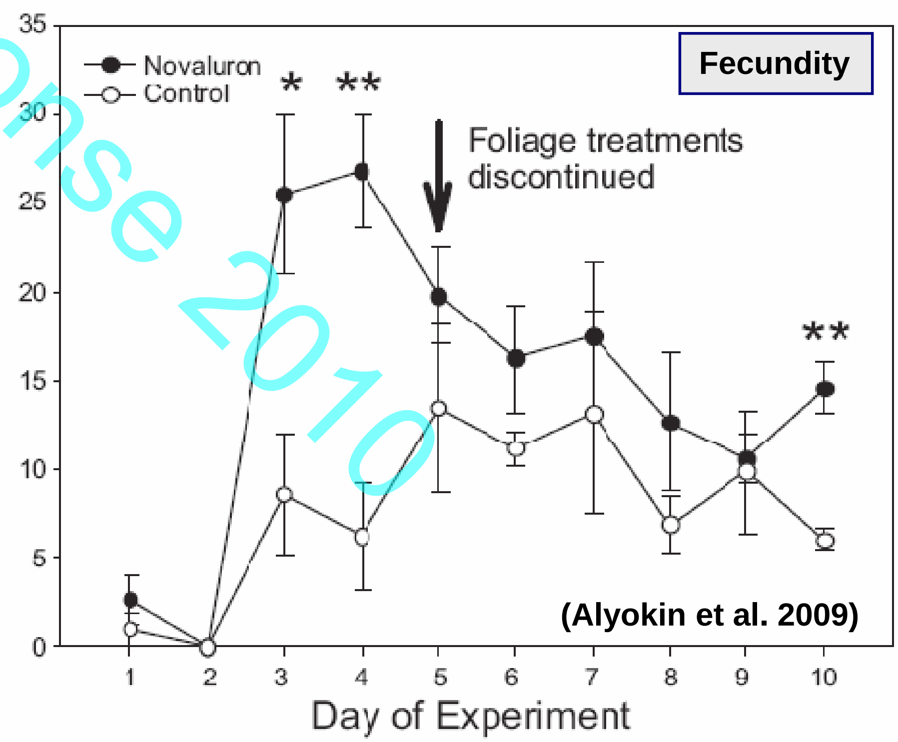
(Ramachandran et al. 1988)



Magnitude of response

- Greater than 30-60% stimulation → different than hormesis?
- Questions – which endpoints? Consistency among groups? Mechanisms?

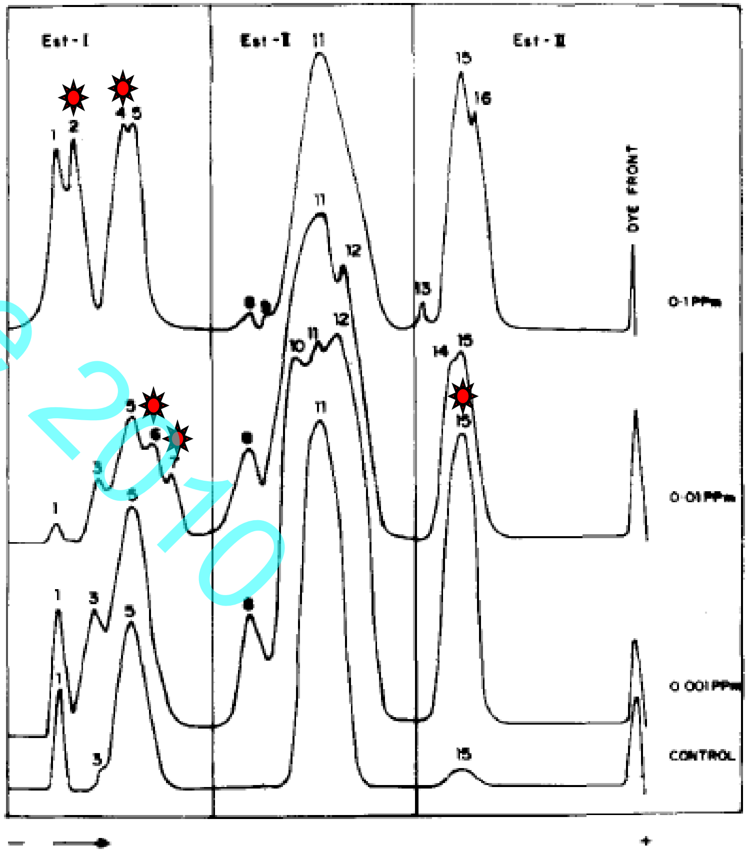




Avenues to study mechanisms

- Solid foundations in insect/insecticide toxicology, biochemistry and molecular biology
- Enzyme induction, e.g. esterase
  - Reproductive behavior
  - Pheromone, hormone metabolism
  - Digestion
  - Neurotransmission
  - Insecticide resistance
- Dose – time – response
  - Induction vary with time and dose?

(Mukherjee et al 1993)



Avenues to study mechanisms

- Many genes/factors involved in insect reproduction, endocrinology, metabolism, etc. now identified
  - Link dose-response measures to gene expression
- e.g. genes in *Myzus persicae*
  - Pesticide metabolism (AChE)
  - Mitochondrial carrier proteins (Adenine nucleotide translocase)
  - JH binding proteins (Mp TOL); locomotor activity
  - Wing dimorphism (OS-D gene)
  - JH precursor (Farnesyl diphosphate synthase (*Mp*FPS1/2)
- Much work in this area is needed

Behavioral and Plant Effects

- Insecticides may stimulate feeding, modify behavior
- Insecticides may affect plant growth

Table 2.—Feeding rate of *N. lugens* as influenced by insecticide treatment in rice.

Treatment	Counts/5 sec/ insect [1]	Increase/ decrease over control (%)
Decamethrin	6308a	+61
Methyl parathion	5587ab	+43
Diazinon	5185b	+33
Perthane	2955d	−24
Control	3912c	—

(Chelliah et al 1980)

Table 1.—Effect of spray of insecticides on plant growth and on the orientational response of brown planthopper, *Nilaparvata lugens* (Stål) as influenced by odor stimulus and plant growth.

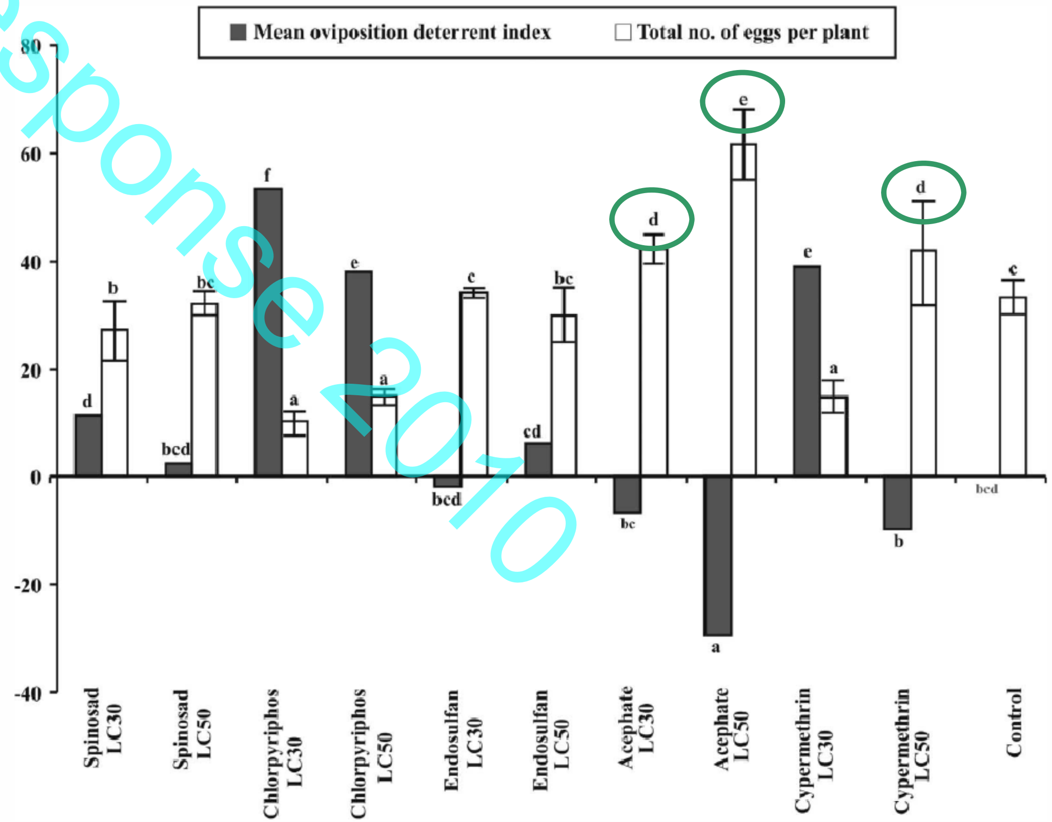
Treatment	Orientational response as influenced by [1]		Changes in plant growth		
	Odor stimulus (% adults alighted)	Plant growth (% adults alighted)	Tiller (no.)	Leaves (no.)	Height (no.)
Methyl parathion	24.3a	31.5a	9.8a	32.4a	75.3a
Decamethrin	27.4a	28.6b	7.6b	27.4ab	75.4a
Diazinon	25.9a	23.2c	6.8b	23.5b	71.6ab
Perthane	27.2a	23.4c	7.2b	23.5b	69.6b
Control	26.0a	24.3c	7.2b	23.5b	74.7a

Insecticide induced plant changes

- Cotton with less spread and reduced upper canopy leaf area were preferred for oviposition by cotton bollworm (Hari and Mahal 2008)

Table 2. Sub-lethal influences of different insecticides on various phenological characteristics of cotton plant.

Treatment (Conc.)		Plant height (cm)	Plant spread (cm)	Upper canopy leaf area (cm^2)*
Spinosad	LC_{30}	58.27 c	35.13 de	246.86cd
	LC_{50}	59.97 c	32.58 bcd	229.91cd
Chlorpyriphos	LC_{30}	54.34 c	34.13 cde	262.91d
	LC_{50}	57.77 c	27.27 abc	195.04bc
Endosulfan	LC_{30}	58.37 c	29.83 bcd	174.25b
	LC_{50}	50.68 bc	40.16 e	195.78bc
Acephate	LC_{30}	40.50 a	20.33 a	156.24ab
	LC_{50}	53.38 c	26.66 ab	169.51b
Cypermethrin	LC_{30}	52.92 bc	28.13 bcd	151.58ab
	LC_{50}	43.25 ab	20.64 a	105.05a
Control	–	57.80 c	34.41 cde	258.27d



Models – e.g. fitness trade-offs

- Are there trade-offs? What are they? How consistent across groups/stressors?
- Increased pupation of blow flies with cadmium spiked diet but reduced survival (Nascarella et al. 2003)

Treatment group	Cadmium conc. of larval diet (ppm)	(a) mean% pupation (S.E.M.)	(b) mean% emergence (S.E.M.)	(c) Pupae deaths (% of total larvae)	(d) Stage specific deaths (% of total pupae)
10	200.0002	0.0 (0)	0 (0)	100	0
9	20.0002	13.9 (12.3)	0.0 (0)	86	100
8	2.0002	70.9 (11.4)	20.8 (14.9)	29	69†
7	0.2002	80.7 (7.8)*	54.5 (12.2)	19	44
6	0.0202	86.7 (4.4)**	57.8 (17.0)	13	46†
5	0.0022	80.5 (6.2)*	77.0 (9.5)	19	17
4	0.0004	80.7 (6.0)*	55.3 (15.2)	19	45†
3	0.00022	88.1 (6.5)***	67.7 (11.2)	12	25†
2	0.0002	78.1 (5.3)*	64.7 (14.4)	22	36†
1 (Control)	> 0.0002	74.4 (4.2)	79.2 (9.5)	26	16

Models – fitness trade-offs

- Reduced duration of red cotton bug post-embryonic development with eucalyptus oil exposure but reduced survival (Srivastava et al. 1995)

Postembryonic developmental data of *Dysdercus koenigii* in relation to a single exposure of nymphs to eucalyptus oil volatiles

Age at exposure (days)	Duration of exposure (hours)	Nymphal condition	Nymphal mortality N = 100	Total PED time, Mean (± SE) (in days)		Number of surviving adults		Adult fresh weight Mean (± SE), (in mg.)	
				Male	Female	Male	Female	Male	Female
3	2	Control	16	25.5 ± 1.3	29.9 ± 1.4	42	42	137.0 ± 5.4	231.0 ± 11.9
		Treated	30	26.3 ± 1.4	27.2 ± 1.5	40	30	104.5 ± 2.5**	197.5 ± 4.7*
5	3	Control	23	19.4 ± 0.3	18.9 ± 0.3	46	31	145.5 ± 3.7	220.0 ± 4.4
		Treated	57*	19.9 ± 0.6	18.9 ± 0.4	26	17*	117.0 ± 3.0**	200.5 ± 6.8*
10	4	Control	21	24.9 ± 0.3	25.3 ± 0.2	50	29	106.4 ± 1.9	220.4 ± 8.9
		Treated	56*	22.8 ± 0.3**	22.5 ± 0.3**	30**	14*	99.6 ± 1.8**	195.2 ± 2.9*
15	5	Control	21	24.9 ± 0.3	25.3 ± 0.2	50	29	106.4 ± 1.9	220.4 ± 8.9
		Treated	33	21.6 ± 0.2**	23.2 ± 0.4**	45	22	101.4 ± 2.3	190.9 ± 2.9*

Models – fitness tradeoffs

- Sublethal imidacloprid and dinotefuran doses reduce reproduction but stimulate production of wing forms (Bao et al. 2008

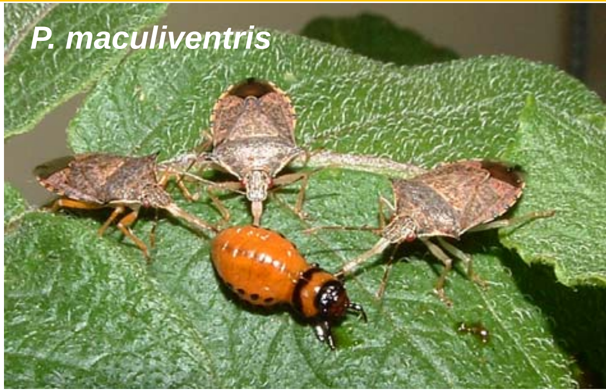
Table 2. Effects on reproduction in macropterous families caused by sublethal doses (LD_{20}) of the four insecticides[a] (Bao et al. 2008)

Treatment	Copulation rate (%)	Fecundity (eggs per female)	Viability (%)	Number of offspring per female
Control	82.31 (±4.56)a	333.65 (±52.77)b	88.20 (±4.07)a	242.22 (±34.22)b
Imidacloprid	76.44 (±5.09)ab	229.41 (±34.88)c	90.07 (±5.42)a	157.95 (±26.01)c
Dinotefuran	70.88 (±5.42)b	174.90 (±31.06)d	86.56 (±5.30)a	107.31 (±18.39)d
Triazophos	84.42 (±6.13)a	488.63 (±43.10)a	89.46 (±4.75)a	369.02 (±45.79)a
Fenvalerate	83.61 (±4.32)a	526.22 (±64.22)a	91.00 (±5.29)a	400.38 (±76.67)a

Table 4. Percentages of macropterous females and males in macropterous and brachypterous families treated with sublethal doses of four insecticides[a]

Treatment	Females in macropterous families	Males in macropterous families	Females in brachypterous families	Males in brachypterous families
Control	43.53 (±3.26)a	52.56 (±3.57)a	13.68 (±1.47)a	21.75 (±2.42)a
Imidacloprid	65.27 (±4.22)b	66.23 (±3.29)b	35.77 (±4.02)c	33.28 (±2.57)c
Dinotefuran	74.19 (±5.37)c	72.01 (±3.32)c	43.19 (±3.21)d	38.72 (±2.79)d
Triazophos	46.24 (±4.70)a	48.88 (±2.95)a	15.23 (±2.18)a	23.06 (±3.39)a
Fenvalerate	48.84 (±6.79)a	55.71 (±4.34)a	22.49 (±4.76)b	28.49 (±3.15)b

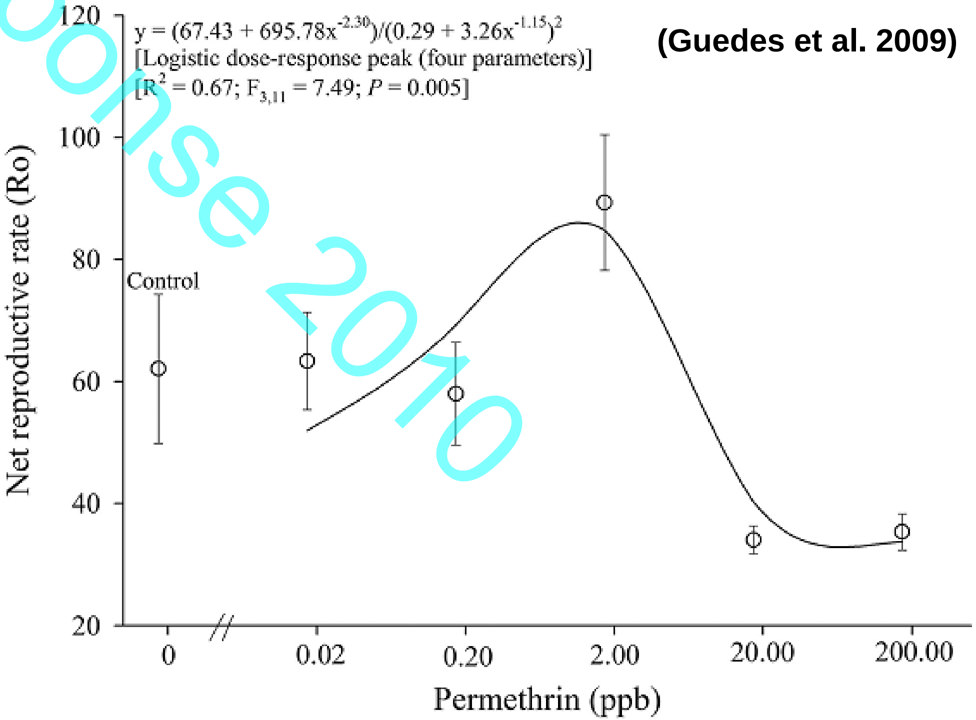
Hormesis in beneficial insects



- Could hormesis be utilized in biological control?

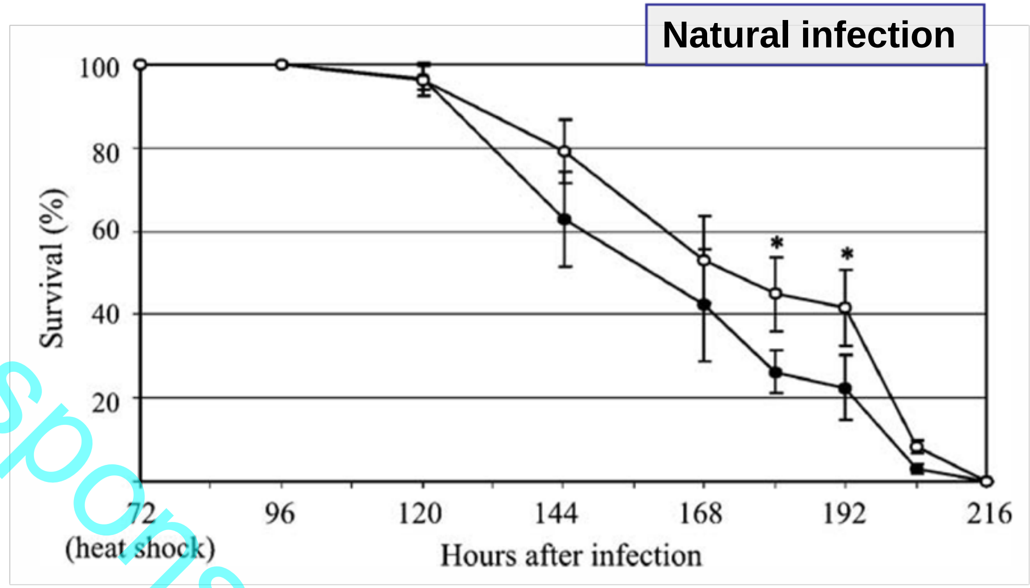
- Increase in reproductive rate of *Podisus distinctus* following single topical application of permethrin (Guedes et al. 2009)

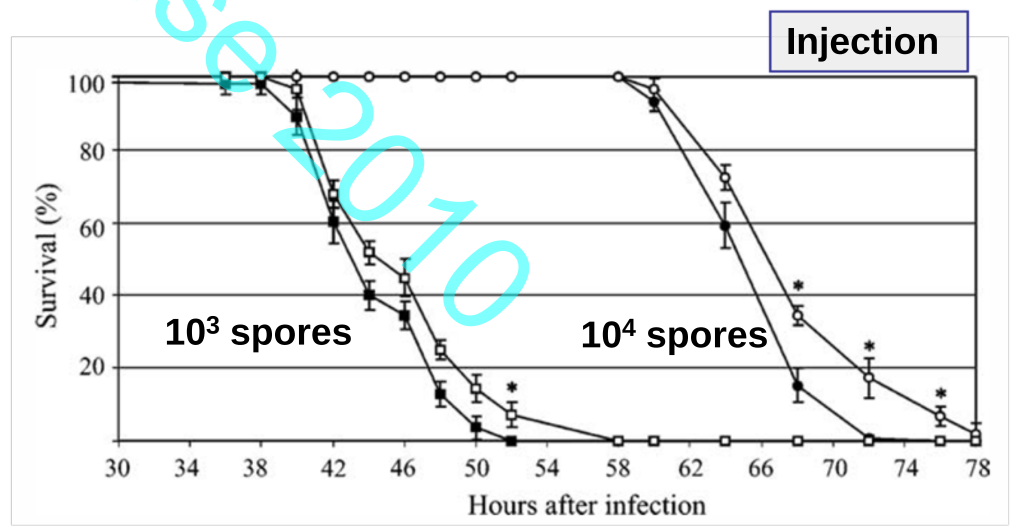
(Guedes et al. 2009)



Hormesis in beneficial insects

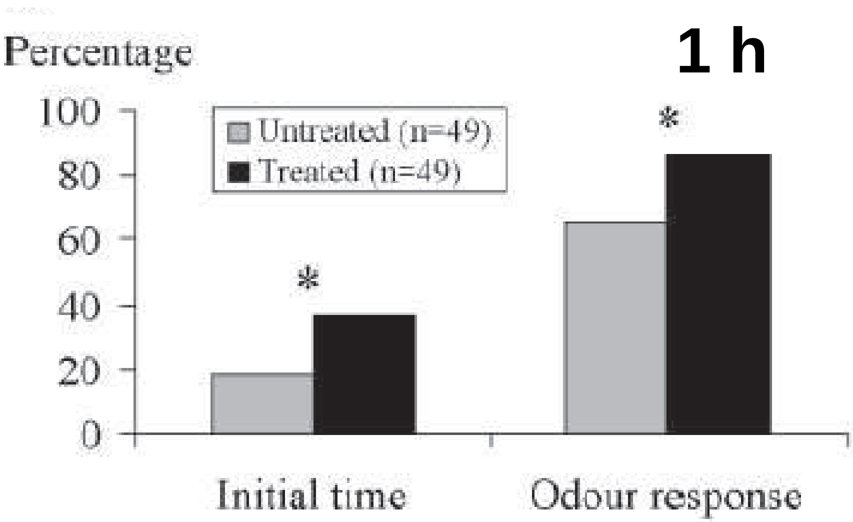
- Short-term heat shock increased survival of *G. mellonella* larvae infected with entomopathogenic fungus *B. bassiana* (Wojda et al.2009)

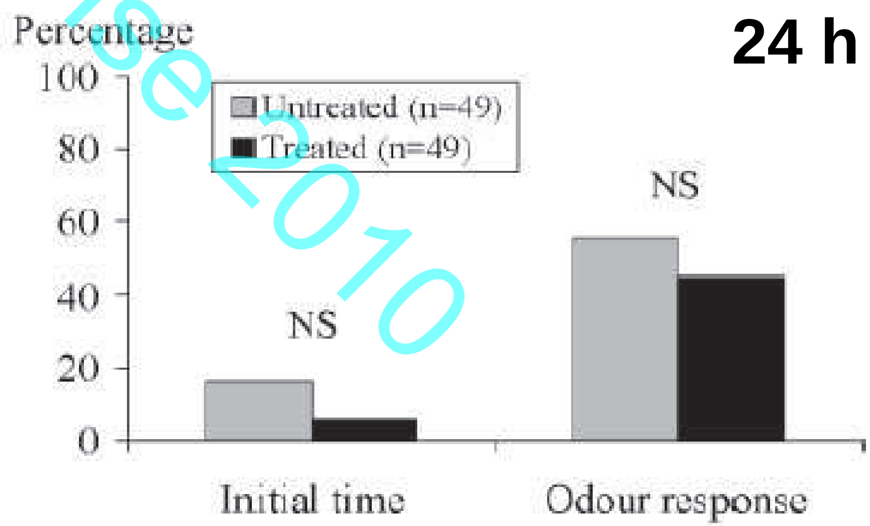




Hormesis in beneficial insects

- Treatment of chlorpyrifos LC_{20} increased *Leptopilina heterotoma* (parasitoid of *Drosophila*) probing with or with banana odor at 1 h after conditioning but not 24 h after conditioning (Rafalimanan et al. 2002)





Summary – Insects and Hormesis

- Practical and basic importance
  - Insecticides and pest management → pest resurgence, resistance, biological control, etc.
    - Tease apart hormesis from other factors causing stimulation
  - Useful models to study the phenomenon
    - Questions – Doses that induce stimulation, magnitude of response; consistency across groups; mechanisms

Thank-you

Questions?