PESTICIDES IN FOOD COMMODITIES AND HEALTH IMPACT

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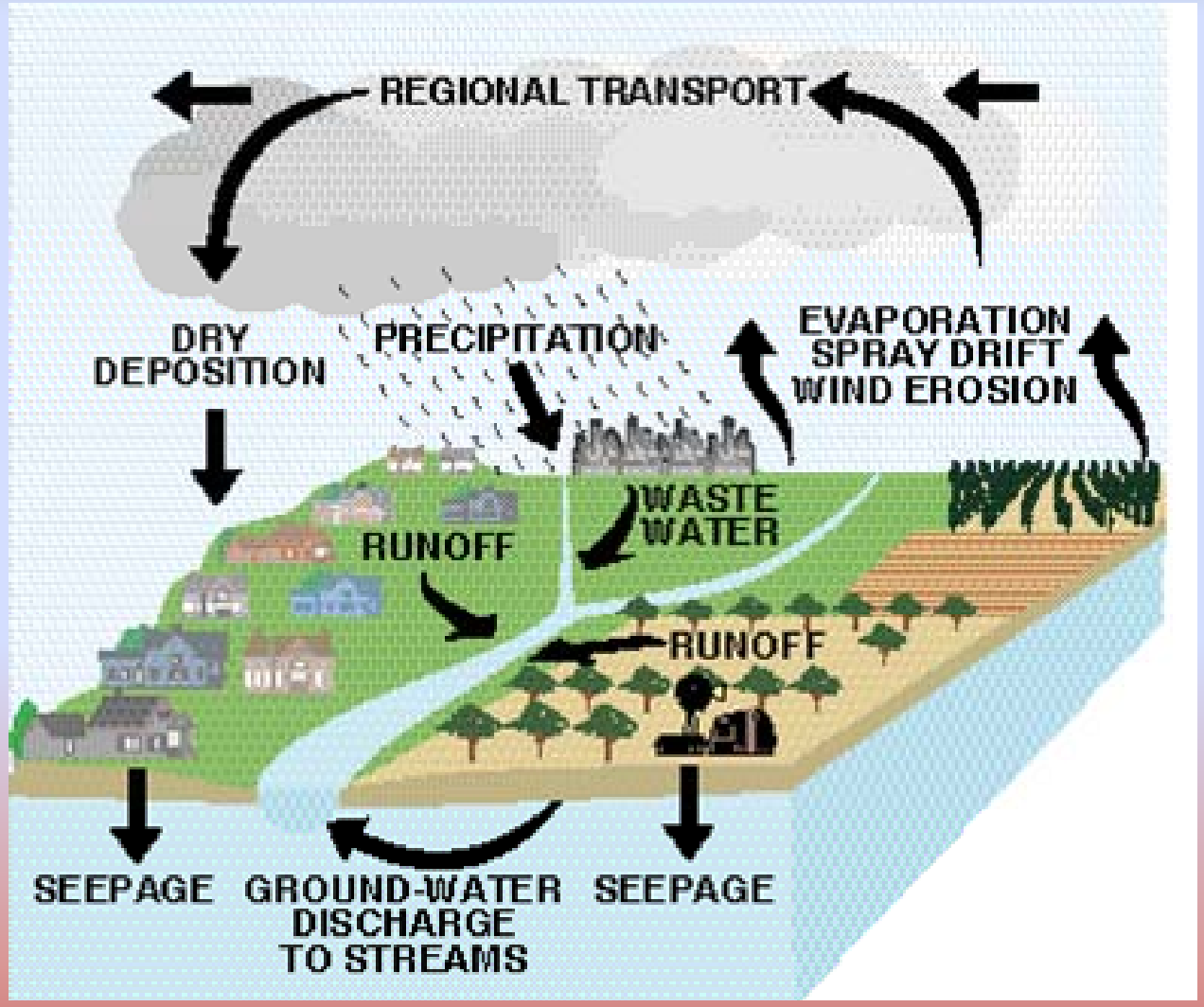
PESTICIDES IN THE ATMOSPHERE AND WATER

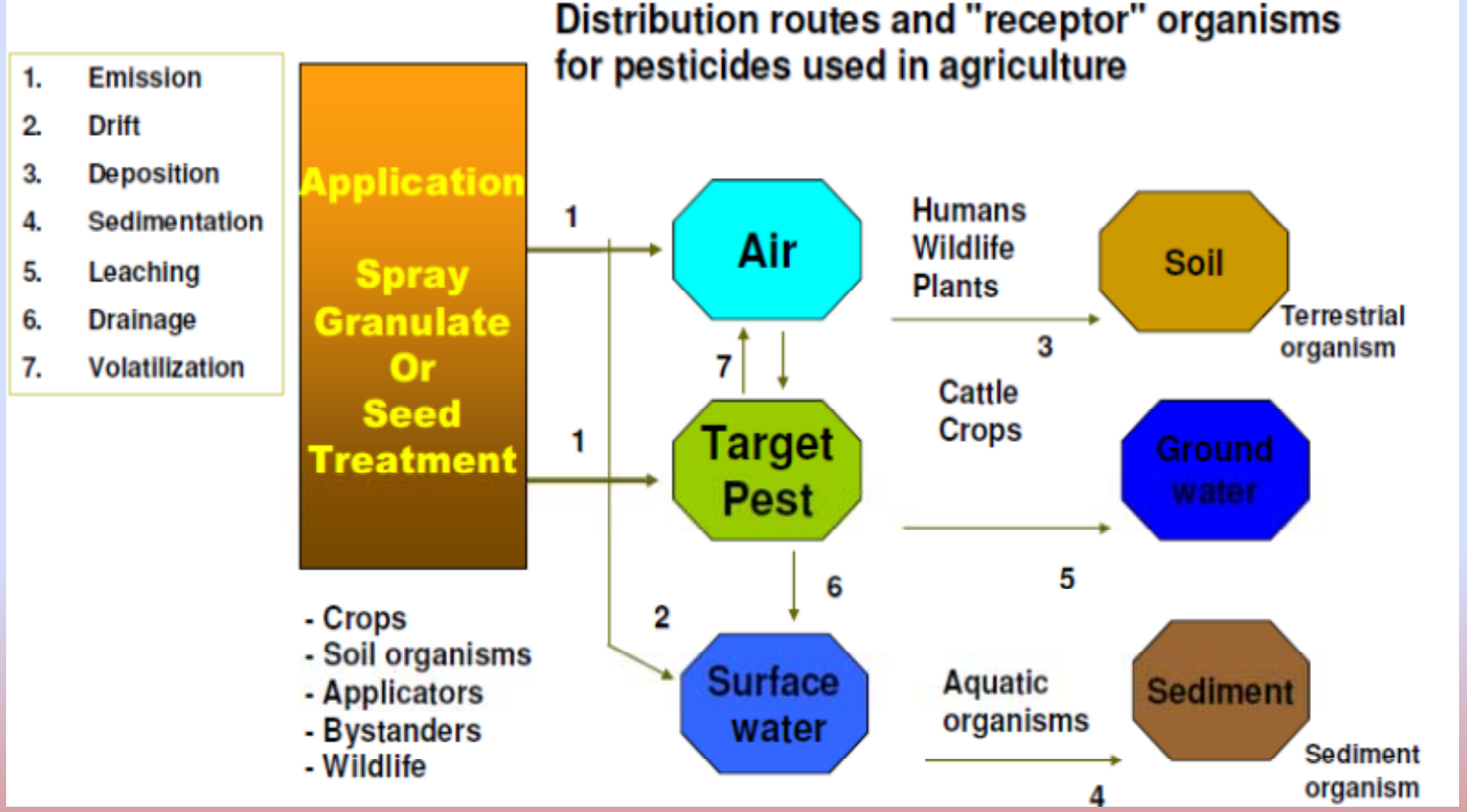


Reference:

•USGS: ca.water.usgs.gov/pnsp/index.html

A great potential for adverse effects of pesticides is through contamination of the hydrological system, which supports human life, aquatic life and related food-chains.

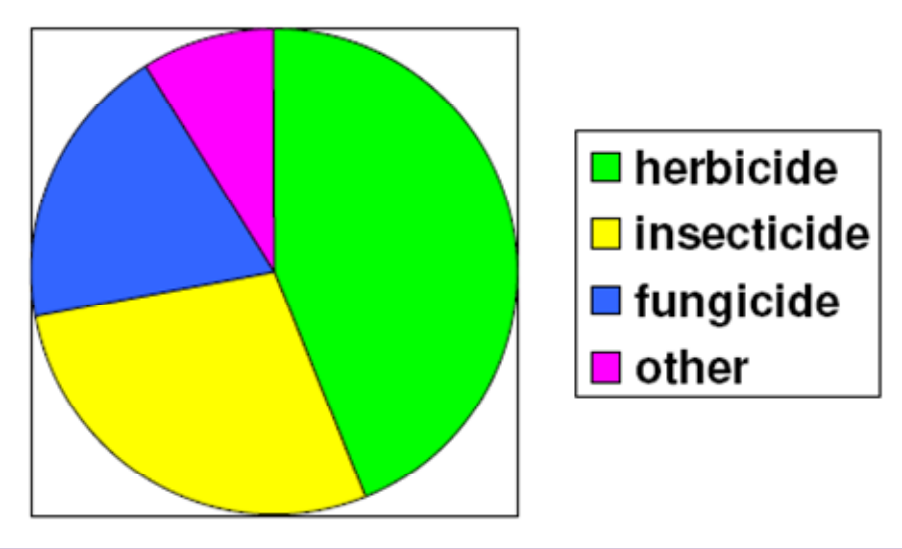
ORIGIN, TRANSPORT AND FATE



Reference:

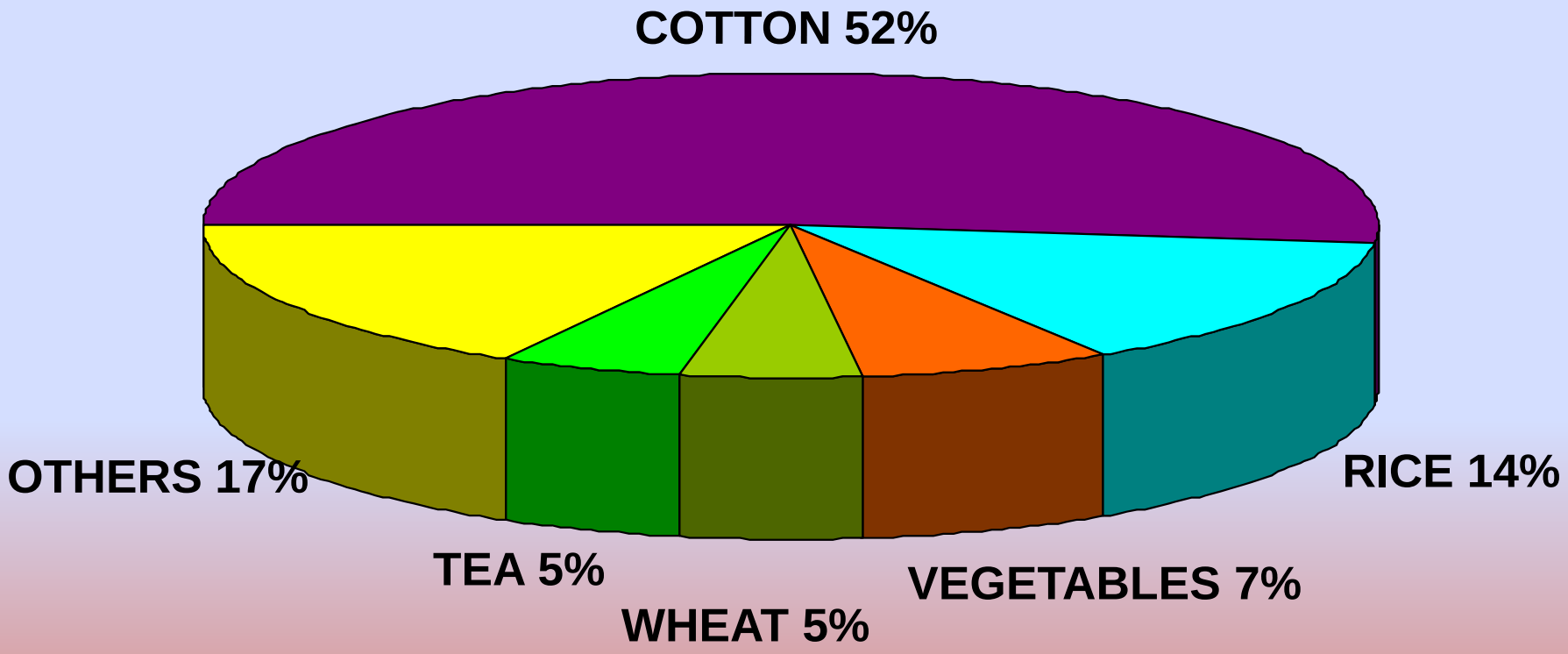
- *Children: a CICH profile, 3rd ed. Canadian Institute of Child Health, 2000.*
- *Health Canada. Health and the environment: handbook for health professionals. Ottawa, Ministry of Public Work and Government Services,1998.*

USE OF PESTICIDES
Global Pesticide Use (%)



(www.epa.gov/oppbead1/pestsales/01pestsales/market_estimates2001.pdf)

Crop wise segmentation of Utilization of pesticides



ROUTES OF EXPOSURE

Ingestion- Breastfeeding Accidental ingestion Residues in food Mouthing

Inhalation- Indoor and outdoor spraying Occupational exposure

Dermal absorption- Accidental contact Occupational Residues on surfaces Contaminated clothing, Multiple/simultaneous routes of exposure

Transplacental- Pesticides can cross the epithelium of the skin and mucous membranes that exchange gases (alveoli) or nutrients (gastrointestinal mucosa).

(Reference:
• Health Council of the Netherlands. Pesticides in food: assessing the risk to children. The Hague: Health Council of the Netherlands, 2004.)

ROUTES OF PERINATAL EXPOSURE

Mother's intake and body burden is transferred across the placenta

Breast milk may be contaminated
"The very top of the food chain"

- They have been detected in the amniotic fluid and body tissues of human foetuses even during early stages of prenatal life.
- Pesticides have also been found in the meconium.

(References:

• Pronczuk J et al. Global perspectives in breast milk contamination infectious and toxic hazards. *Environ Health Perspect,* 2002, 110:A349.

• Whyatt RM et al. Measurement of organophosphate metabolites in postpartum meconium as a potential biomarker of prenatal exposure. A validation study. *Env Health Perspect,* 2001, 109:417.

Picture: WHO, P. Virot. Portrait of a young baby breastfeeding, Delhi, India, Asia, November 2002.)

SOURCES AND SETTINGS OF EXPOSURE...

HOME, SCHOOL, DAY-CARE, INSTITUTIONS, …

Indoor and outdoor application

- Mosquito control
- Professional/domestic application

Health uses

- Medical use: scabies, head lice
- Fleas or ticks on pets

Pesticide residues

- Dust, soil, furniture, carpets, toys, food…
- Playgrounds, playing fields, lawns, gardens, farms, agricultural areas (rural setting)
- Wood preservatives in play structures (e.g. PCP: pentachlorophenol)
- Long range transport of POPs (e.g. DDT)

Different scenarios:

- ACUTE - high-level exposure, overt poisoning
- CHRONIC - low-level, chronic exposure, various effects

(Reference:
•www.epa.gov/pesticides/factsheets/childsaf.htm)

MECHANISMS OF ACUTE TOXICITY

- **Irritation (most of the pesticides)**
- **Allergic sensitization (e.g. fungicides)**
- **Enzyme inhibition (e.g. cholinesterase and OPs & carbamates)**
- **Oxidative damage (e.g. paraquat)**
- **Inhibition of neurotransmission (e.g. organochlorines)**
  - **Calcium (Ca2+) homeostasis alteration**
  - **GABA inhibition**
- **Uncoupling of oxidative phosphorylation (e.g. glyphosate)**

  - The effects are usually redness and pain.
  - Respiratory irritation can produce nasal, laryngeal or pulmonary effects.
  - Most herbicides and fungicides are strong irritants.
  - Allergic sensitization is a common effect of pesticides, especially fungicides.
  - Enzyme inhibition (e.g. cholinesterase activity is decreased by exposure to OP compounds and carbamates).
  - Oxidative damage (e.g. paraquat is a promoter of superoxide radicals).

Poisoning due to Pesticides

- 200,000 People are killed worldwide every year.
- 68,000 Farmers and Workers are poisoned yearly.
- 25 million workers suffer pesticide poisoning (occupational exposure in industries and agriculture fields)

Study 1:

INTRODUCTION:

- Food has been recognized as main source of exposure to pesticide (*Adachi and Okano, 2006*).
- Total Diet Studies (TDS) are conducted World-wide.
- TDS, measures the intakes of chemicals by various physiological age groups. Further, the samples are processed as consumed before they are extracted and analysed.
- Thus, they take into consideration the reduction of those chemicals that degrade during processing at home and present the best available data to estimate dietary pesticides intakes for a given population.

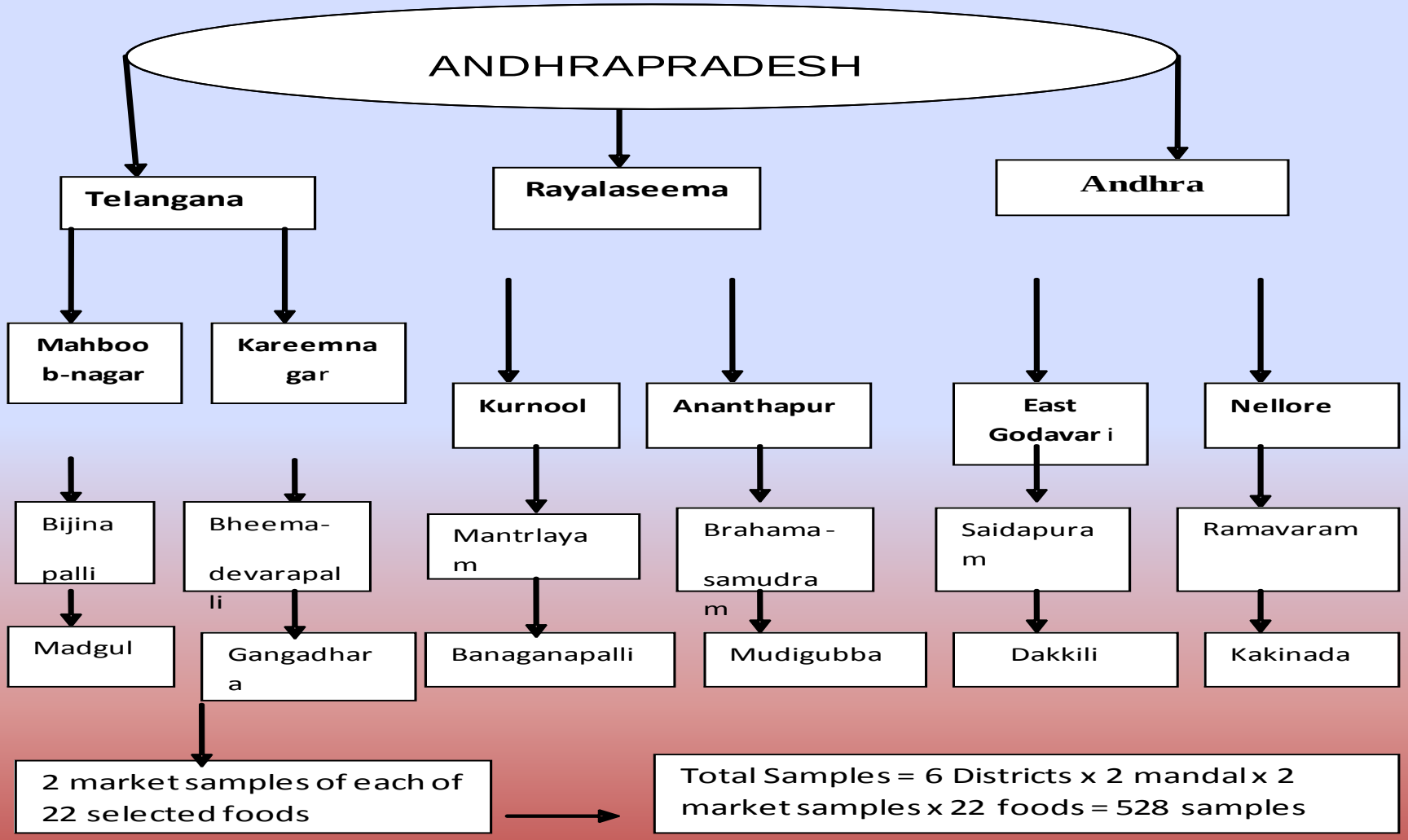
- The pesticide Production in India has remained stable at 82,000 - 85,000 MT in financial year 2009-2010.
- However, in India the pesticide industry is dominated by insecticides, whereas globally herbicides and fungicides are the key segments (*Indian Pesticides Industry, 2011*).
- Andhra Pradesh is the second highest pesticide consuming State in India (*Andhra Pradesh State Agricultural Department, 2010*).
- There is a dearth of information on exposure assessment through pesticide residues among various physiological groups in India (*Battu et al. 2004*).

- A total diet study approach was used to assess the dietary exposure to nineteen pesticide residues in six districts of Andhra Pradesh state of southern India.
- Twelve commonly consumed foods which were representative of the diet, were collected, processed as table ready and analysed for the presence of various organochlorines, organophosphates and synthetic pyrethroids.

- The foods which were most commonly consumed in the state of Andhra Pradesh were listed in the order of consumption.
- The consumption data was obtained from National Nutrition Monitoring Bureau (*NNMB*).
- NNMB has been conducting surveys in rural areas of ten Indian states since 1975 (*NNMB Report, 2000*). The amounts of consumption of various foods were obtained from the NNMB consumption data and among those, the 22 most commonly consumed foods were selected from various food groups along with drinking water samples.
- Water samples were collected to estimate the exposure through water which is consumed and also used for the cooking of various foods.

METHODOLOGY

- Collection of samples- from September 2008 to May 2009.
- Study Design : Multistage random sampling procedure



- Information on the areas of foods cultivation/ local/imported, transportation/storage - a questionnaire.
- The items which were not found in the selected procurement points were collected from the next nearest market place.
- Semi perishable food samples were transported to the lab under ambient temp and perishable food samples under frozen conditions.
- Processing of the perishable samples was done the very next following day after they were transported to the lab and subsequent processing for the rest.
- Individual foods were subjected to basic household cooking using de-ionized water, stainless steel vessels, lids, ladles, knives and PTFE chopping boards and the foods were prepared as "*table ready*" without adding other ingredients such as salt oil and spices as per the SOPs.

- No detergent was used.
- Hot running water used for processing was tested for the Pesticide Residues.
- Cooked foods were cooled, weighed, homogenized and were stored in sterile wide mouthed PTFE containers and stored at -80°c until analyzed.
- Specific pesticide residue incidences reported in each of the foods were selected and analyzed.
- Each food was analysed individually using - multi residue QuEChERS extraction method finally quantified by *gas chromatograph (GC)* equipped with an *Electron Capture Detector (ECD).*
- The peaks eluted were confirmed by comparing the results of mass spectra of substances from food sample spectra to those of standard compounds selecting specific ions obtained from an ion trap detector using Turbomass GC/MS.
- Recovery studies were performed on 10 spiked replicates of blank food samples at concentrations 100, 75, 50 and 25 µg/ kg. Recoveries ranged from 80-85% using GC, LLOD for selected pesticides studied on GC ECD was 1.0 µg/ kg.

Table - Methods adopted for processing the foods

S. No	Food	Method of Preparation
1.	Rice, raw, milled	Boiled in distilled water
2.	Red gram dhal	Boiled in distilled water
3.	Groundnut oil	Analysed as it was
4.	Buffalo milk	Boiled and analysed
5.	Butter milk	Analysed as it was
6.	Tomato	Washed with distilled water, chopped and boiled in water
7.	Brinjal	Washed with distilled water, sliced and boiled in water
8.	Potato	Washed with distilled water, boiled in distilled water and peeled off the skin
9.	Mango	Washed with distilled water, peeled, sliced and mashed and used
10.	Amaranth	Washed with distilled water dried overnight, chopped and boiled
11.	Spinach	Washed with distilled water dried overnight, chopped and boiled

DIETARY EXPOSURE

- Dietary exposure was calculated based on the consumption data, contaminant data.
- Exposures were calculated by multiplying concentration of the pesticide residues and amount of that food consumed.
- Mean Conc. of pesticide residues were considered for calculating the exposures at mean and 95th percentile ingestion levels.

RESULTS

- All samples were contaminated with one or the other of the 19 pesticide residues.
- Number of residues detected varied from sample to sample.
- *Cypermethrin* was detected in highest Conc. (13.7µg/kg) in all the samples and samples of mango, milk and spinach (>60%) *(Table 2).*
- Spinach had the highest mean concentration of *Cypermethrin* among all the foods.
- Highest concentrations of *g-HCH*, *chlorpyriphos*, *2'4DDE* and *endosulfan sulphate* were detected in water than in food samples *(Table 2).*

- Mean Conc. of total *DDT* was highest in milk (1.778 µg/kg fresh weight) followed by brinjal (1.76 µg/kg fresh weight) & in potato (0.061 µg/kg fresh weight) lowest of mean conc. was detected *(Table 2).*
- *Alpha Chlordane* & *Endosulfan Sulphate* were Below the Detection Limit (BDL) in many of the foods *(Table 2).*
- *HCH & its derivatives (α, β, γ and δ)* were found above the detection levels in all the foods *(Table 2).*
- None of the samples tested without processing (mango and water), exceeded the MRL values given by *FSSAI, 2011*.

- Exposure assessments were carried out for different age, sex and physiological groups. The results of the estimated dietary exposure analysis revealed that exposures to all the physiological groups were within the safe limits.
- The children of 7-9 and 10-12 years however, were more at risk to the persistent organochlorine pesticide residues.

DIETARY EXPOSURE ASSESSMENT

- Dietary exposure to a specific contaminant is dependent on the quantity of food consumed, which varies with age.
- The estimates were calculated both at mean intakes levels and at "upper bound" intake levels, i.e. at 95th percentile intakes (*As per the NNMB report*).
- Dietary intake of all the pesticide residues detected was well below their respective *Acceptable Daily Intakes* (ADI).
- There has been a concern regarding the application of the ADIs generated based on adult body weight of 60 Kg to children.
- Extrapolating the ADIs to children, it was further reduced by ten times.
- The exposures did not exceed the ADIs for any of the pesticide residues in any of the age/physiological groups at both mean/95th percentile intakes.

DISCUSSION

- Total diet studies are recommended internationally as the most effective method for National exposure assessments.
- Total Diet Study in Andhra Pradesh (APTDS) is first of its kind to have been conducted in India.
- None of the samples were free from pesticide residual contamination.
- pesticide residues did not exceed the reference toxicological values in any age/physiological groups either due to dietary exposure or in their concentrations in cooked/raw foods (MRLs).
- Major drawback of TDS is use of composites which do not indicate which food is the actual source of exposure to what level.
- Milk was the major contributor for *γ-HCH* in 4-6 yrs (33%), 7-9 yrs (34%), 13-15yrs (27%), 16-17 yrs (30%) and pregnant women (26%) *(Table 3,4)*.

- The total *DDT* is far less than the ADIs in all the age groups ranging from 0.01-0.03% of ADIs which are far less than the average value of 0.4% of ADI found in Canadian TDS during 1998 (*Rawn et al, 2004*).
- Further, the values of *γ-HCH* and *chlorpyriphos* were 0.13% and 0.08% of ADI, respectively; in Canadian population during the same period while these values in Indian populations were lower ranging from 0.003-0.1% and 0.02% of ADI, respectively *(Table 3,4)*.
- Mango and amaranth were contaminated with a large number of pesticides, some even at high levels, the exposure is lower than the other foods as they are consumed in smaller quantities.
- The detection of banned pesticide residues like *DDT* and its isomers/derivatives was not surprising and it hypothesised that they, being organochlorines have a longer sustenance in the environment.

(*http://het.sagepub.com/content/early/2011/01/18/0960327110396535.full.pdf*).

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Table 1. Intakes of various foods (g) at mean and 95th percentile levels in different age and physiological groups in Andhra Pradesh in 2006.

Age Group	1-3 yrs (N=176)		4-6 yrs (N=178)		7-9 yrs (N=215)		10-12 yrs (N=102)		13-15 yrs (N=97)		16-17 yrs (N=47)		Sedentary Worker (N=343)		Pregnant Women (N=21)	
Foods	Mean	95th	Mean	95th	Mean	95th	Mean	95th	Mean	95th	Mean	95th	Mean	95th	Mean	95th
Cereals	172 ± 92	340	243± 101	455.5	279± 120	540.0	347± 131	565.45 (B) 589.95 (G)	440± 164	745.00(B) 543.00(G)	510± 164	836(B) 644.60 (G)	426± 155	689.6(M) 600.8 (F)	401± 149	659.3
Pulses	12± 15	37.2	18± 18	55.25	22± 24	75.2	22± 25	75.85 (B) 71.45(G)	23± 25	68.08 (B) 70.00(G)	37± 55	123.7 (B) 95.08 (G)	32± 34	92.46 (M) 86.4(F)	14± 29	97.70
Spinach and Amaranth	1± 6	5.2	3± 10	25.25	4± 14	27.28	6± 20	45.70(B) 29.37(G)	9± 28	68.44(B) 28.05(G)	7± 16	39.18 (B) 40.07 (G)	7± 19	40.52 (M) 32.34 (F)	14± 27	93.50
Tomato and Brinjal	11± 19	47.4	19± 29	88.05	22± 33	99.20	27± 41	116.11 (B) 114.55(G)	30± 36	101.00 (B) 114.50(G)	48± 54	159(B) 119.4(G)	41± 57	161.40.(M) 142.3 (F)	47± 44	125.7
Potato	12± 19	50.0	17± 22	69.35	21± 25	78.64	24± 26	76.80(B) 88.22(G)	30± 28	97.67(B) 61.95(G)	44± 52	182.2 (B) 91.26 (G)	39± 45	146.40(M) 111.2 (F)	33± 43	161.3
Mango	33± 60	166. 5	34± 59	194.25	39± 62	207	47± 88	186.35 (B) 235(G)	38±53	161.50 (B) 254.25(G)	45± 55	186.4 (B) 175.5 (G)	44± 58	173.60(M) 205.6 (F)	58± 80	260.9
Milk and Buttermilk	81± 105	298. 5	77± 91	255.25	78± 106	356.2	82± 112	364.15 (B) 274(G)	71± 96	210.00 (B) 263.00(G)	106± 94	284.8(B) 308.1(G)	139± 130	388.2(M) 392 (F)	141± 198	652.2
Cooking Oil	7± 8	19.1 5	9± 6	22.02	12± 11	27.12	13± 11	30.87(B) 31.3(G)	14± 8	29.2(B) 34.10(G)	23± 22	57.40 (B) 36.89 (G)	20± 16	52.60 (M) 43.4 (F)	14± 8	34.1

Abbreviations: yrs, years; 95th, 95th percentile intake levels; B, boys; G, girls; Source: *NNMB Report, 2006*

Table 2. Concentration of pesticide residues in foods (µg/kg raw weight)

Pesticide	Amaranth	Brinjal	Buttermilk	Mango	Milk	Potato	RGD	Rice	Spinach	Tomato	Water	G oil
a-HCH	0.04 (0.12)	0.21 (0.49)	0.023 (0.08)	0.57 (0.29)	0.83 (0.67)	0.03 (0.16)	BDL	1.42 (6.4)	0.007 (0.03)	0.28 (0.47)	BDL	0.98 (0.63)
b- HCH	0.11 (0.24)	0.22 (0.57)	0.18 (0.54)	2.32 (1.83)	BDL.	0.01 (0.040	0.043 (0.21)	0.13 (0.64)	0.064 (0.17)	0.27 (0.91)	BDL	0.44 (0.59)
g- HCH	0.5 (0.67)	0.16 (0.59)	0.30 (0.39)	0.35 (0.28)	0.65 (0.71)	0.13 (0.41)	0.093 (0.29)	0.029 (0.11)	0.048 (0.08)	0.048 (0.20)	0.89 (1.81)	0.55 (0.39)
d- HCH	0.14 (0.26)	0.04 (0.20)	0.018 (0.08)	0.75 (0.48)	0.5 (0.28)	BDL	0.048 (0.13)	0.25 (0.77)	0.303 (0.53)	0.063 (0.23)	0.1 (0.27)	0.36 (0.34)
Chlorpyriphos	0.29 (0.81)	0.21 (0.54)	0.12 (0.35)	1.01 (1.14)	1.55 (0.7)	0.25 (0.50)	0.32 (0.63)	0.27 (0.63)	BDL	0.17 (0.36)	1.027 (2.16)	0.83 (0.32)
2`4 DDE	0.13 (0.33)	0.11 (0.17)	0.25 (0.14)	BDL	0.1 (0.06)	0.013 (0.06)	0.28 (0.66)	0.033 (0.16)	0.13 (0.23)	0.046 (0.11)	0.338 (0.33)	BDL
a-Endosulfan	0.38 (1.51)	0.37 (1.62)	0.214 (0.25)	0.42 (0.49)	0.3 (0.14)	0.055 (0.16)	0.32 (0.98)	0.19 (0.59)	BDL	0.052 (0.24)	0.016 (0.06)	0.12 (0.18)
a-Chlordane	BDL	BDL	BDL	BDL	BDL	BDL	BDL	0.109 (0.33)	BDL	BDL	BDL	BDL
Dieldrin	0.04 (0.12)	0.06 (0.21)	0.15 (0.3)	BDL	0.1	0.025 (0.12)	0.016 (0.05)	0.038 (0.14)	BDL	0.005 (0.02)	BDL	0.008 (0.04)
2'4 DDD	0.04 (0.06)	0.02 (0.06)	0.05 (0.09)	BDL	BDL	0.015 (0.04)	BDL	0.056 (0.23)	BDL	0.017 (0.04)	0.029 (0.11)	BDL
b-Endosulfan	BDL	0.27 (1.19)	0.068 (0.080	0.467 (0.31)	0.157 (0.11)	0.004 (0.02)	0.007 (0.03)	0.239 (0.46)	BDL	0.013 (0.04)	0.143 (0.32)	0.133 (0.24)
4'4 DDD	0.17 (0.69)	0.12 (419)	BDL	0.5 (0.14)	BDL	BDL	0.01 (0.04)	0.082 (0.27)	BDL	0.186 (0.64)	0.173 (0.25)	0.404 (1.8)
2'4 DDT	0.02 (0.09)	0.12 (0.407)	0.075 (0.15)	BDL	0.9 (0.71)	BDL	BDL	0.015 (0.06)	BDL	BDL	0.071 (0.24)	BDL
Endosulfan Sulphate	BDL	BDL	BDL	BDL	BDL	BDL	BDL	0.23 (0.69)	BDL	0.021 (0.07)	0.439 (1.19)	BDL
4'4 DDT	0.01 (0.05)	1.09 (0.13)	1.2 (2.4)	1.043 (0.34)	0.778 (0.32)	0.028 (0.09)	0.064 (0.18)	0.204 (0.36)	0.128 (0.17)	0.047 (0.09)	0.322 (0.41)	0.083 (0.19)
4'4 DDE	BDL	0.3 (0.02)	BDL	BDL	BDL	0.005 (0.02)	BDL	0.419 (1.68)	BDL	0.011 (0.03)	BDL	BDL
Cypermethrin	3.96 (3.02)	1.25 (1.68)	BDL	3.604 (2.37)	2.967 (2.01)	1.028 (1.02)	0.514 (0.72)	2.737 (6.12)	5.1 (3.05)	BDL	2.973 (2.73)	3.63 (3.29)

Abbreviations: µg/kg, micrograms per kilogram of the food; BDL, Below Detection Limit, RGD, Red gram dhal, G Oil, groundnut oil; Values are represented as mean and numbers in the parenthesis are Standard Deviations (SD), LOD (GC-ECD) =1ng/g; LOD (GC/MS) = 5ng/g

Table 3. Estimated daily intakes of the target pesticide residues at 95th percentile food intake levels (µg/kg bw/day)

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Contaminant	1-3 yrs		4-6yrs		7-9yrs		10-12yrs B		10-12yrs G		13-15yrs B	
	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI
a- HCH	0.154	0.307	0.124	0.248	0.105	0.210	0.083	0.166	0.084	0.169	0.047	0.094
b- HCH	0.048	0.095	0.037	0.075	0.029	0.059	0.022	0.043	0.025	0.050	0.013	0.027
g-HCH	0.029	0.059	0.020	0.039	0.018	0.035	0.014	0.028	0.013	0.025	0.008	0.015
d- HCH	0.045	0.090	0.034	0.068	0.029	0.058	0.022	0.045	0.022	0.045	0.013	0.025
Chlorpyriphos	0.083	0.829	0.058	0.582	0.051	0.513	0.040	0.398	0.037	0.374	0.021	0.208
2`4 DDE	0.011	0.109	0.009	0.085	0.008	0.078	0.006	0.062	0.006	0.056	0.003	0.029
a-Endosulfan	0.036	0.061	0.029	0.048	0.025	0.041	0.020	0.033	0.019	0.032	0.011	0.018
a-Chlordane	0.009	0.932	0.008	0.789	0.007	0.661	0.005	0.526	0.005	0.548	0.003	0.303
Dieldrin	0.008	0.000	0.006	0.000	0.005	0.000	0.004	0.000	0.004	0.000	0.002	0.000
2’4 DDD	0.005	0.055	0.005	0.045	0.004	0.038	0.003	0.031	0.003	0.031	0.002	0.018
b-Endosulfan	0.033	0.055	0.026	0.044	0.022	0.037	0.017	0.029	0.018	0.030	0.010	0.016
4’4 DDD	0.016	0.157	0.013	0.131	0.011	0.105	0.008	0.082	0.009	0.089	0.007	0.067
2’4 DDT	0.025	0.245	0.014	0.142	0.014	0.137	0.011	0.107	0.008	0.084	0.005	0.051
Endosulfan Sulphate	0.020	0.331	0.017	0.280	0.014	0.235	0.011	0.187	0.012	0.195	0.011	0.181
4’4 DDT	0.064	0.639	0.044	0.440	0.038	0.385	0.030	0.295	0.028	0.281	0.019	0.194
4’4 DDE	0.036	0.361	0.031	0.306	0.026	0.256	0.020	0.204	0.021	0.212	0.020	0.197
Cypermethrin	0.378	0.756	0.304	0.608	0.256	0.513	0.205	0.410	0.204	0.409	0.175	0.350

Abbreviations: yrs, years; B, boys; G, girls; µg/kg bw/day, micrograms per kilogram body weight per day; ADI, Acceptable Daily Intakes (*FAO, 1996*)

CSE, Feb 2013

Table 4. Estimated daily intakes of the target pesticide residues at 95th percentile food intake levels (µg/kg bw/day)

Contaminant	13-15yrs G		16-17yrs B		16-17yrs G		SW (M)		SW (F)		PW	
	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI	µg/kg bw/day	%ADI
a- HCH	0.058	0.117	0.072	0.143	0.057	0.113	0.059	0.118	0.057	0.114	0.059	0.117
b- HCH	0.019	0.038	0.016	0.032	0.014	0.027	0.014	0.027	0.015	0.031	0.016	0.033
g- HCH	0.009	0.018	0.009	0.018	0.008	0.016	0.009	0.018	0.010	0.020	0.013	0.026
d- HCH	0.016	0.032	0.017	0.034	0.014	0.029	0.015	0.030	0.015	0.031	0.018	0.035
Chlorpyriphos	0.027	0.268	0.028	0.282	0.024	0.245	0.027	0.267	0.028	0.279	0.034	0.336
2`4 DDE	0.004	0.039	0.005	0.048	0.004	0.041	0.004	0.043	0.004	0.043	0.005	0.052
a-Endosulfan	0.014	0.023	0.015	0.026	0.013	0.021	0.013	0.022	0.014	0.023	0.015	0.025
a-Chlordane	0.004	0.374	0.005	0.482	0.004	0.372	0.004	0.378	0.004	0.359	0.004	0.350
Dieldrin	0.002	0.000	0.003	0.000	0.002	0.000	0.003	0.000	0.003	0.000	0.003	0.000
2’4 DDD	0.002	0.021	0.003	0.027	0.002	0.021	0.002	0.022	0.002	0.021	0.002	0.022
b-Endosulfan	0.013	0.021	0.014	0.024	0.011	0.019	0.012	0.020	0.012	0.020	0.012	0.021
4’4 DDD	0.007	0.066	0.007	0.066	0.005	0.054	0.006	0.056	0.006	0.058	0.006	0.059
2’4 DDT	0.006	0.060	0.006	0.057	0.006	0.058	0.007	0.069	0.007	0.075	0.011	0.106
Endosulfan Sulphate	0.008	0.133	0.010	0.171	0.008	0.132	0.008	0.135	0.008	0.128	0.007	0.124
4’4 DDT	0.020	0.203	0.020	0.198	0.018	0.178	0.019	0.192	0.021	0.205	0.025	0.251
4’4 DDE	0.015	0.145	0.019	0.187	0.014	0.144	0.015	0.147	0.014	0.139	0.014	0.136
Cypermethrin	0.143	0.287	0.166	0.333	0.135	0.270	0.142	0.284	0.140	0.281	0.157	0.314

Abbreviations: yrs, years; B, boys; G, girls; SW (M), Sedentary worker (Male); SW (F); Sedentary Worker (Female); µg/kg bw/day, micrograms per kilogram body weight per day; ADI, Acceptable Daily Intakes (*FAO, 1996*).

Table 5. Estimated daily intakes of the target pesticide residues at mean levels of food intake (µg/kg bw/day)

CSE, Feb 2013

Pesticides	1-3yrs		4-6yrs		7 – 9 yrs		10-12yrs		Ref. ADI
	µg/kgbwt/day	% of ADI	µg/kgbw/day	% of ADI	µg/kgbw/day	% of ADI	µg/kgbw/day	% of ADI	
g- HCH	0.007	0.14	0.0421	0.84	0.0034	0.007	0.00291	0.006	0.05
Chlorpyriphos	0.022	0.2	0.0168	0.2	0.014	0.14	0.012	0.12	0.01
2'4' DDE	0.0042	0.04	0.0028	0.03	0.0023	0.02	0.0019	0.02	0.01
2'4' DDD	0.0027	0.03	0.00023	0.002	0.0019	0.02	0.0033	0.02	0.01
4'4' DDD	0.0062	0.06	0.0044	0.07	0.0037	0.04	0.0033	0.03	0.01
2'4 DDT	0.0043	0.008	0.0022	0.02	0.0017	0.02	0.0014	0.01	0.01
Endoslfan Sulphate	0.098	0.2	0.089	1.5	0.008	0.12	0.0068	0.1	0.006
4'4 DDT	0.018	0.2	0.0134	0.1	0.0109	0.1	0.0094	0.09	0.01
4'4 DDE	0.018	0.2	0.0162	0.2	0.139	1.4	0.0012	0.01	0.01
Cypermethrin	0.157	0.4	0.1253	0.2	0.107	0.21	0.094	0.2	0.05

Abbreviations: yrs, years; B, boys; G, girls; µg/kg bw/day, micrograms per kilogram body weight per day; ADI, Acceptable Daily Intakes (*FAO, 1996*)

Table 6. Estimated daily intakes of the target pesticides using mean measured residue concentration of at mean levels of food intake (µg/kg bw/day)

Pesticides	13-15yrs		16 - 17yrs		Sedentary Worker (Male)		Pregnant women		Ref.ADI
	µg/kgbw/day	% of ADI	µg/Kgbw/day	% of ADI	µg/kgbw/day	% of ADI	µg/kgbw/day	% of ADI	
g- HCH	0.0023	0.005	0.0025	0.005	0.0019	0.004	0.0028	0.006	0.05
Chlorpyriphos	0.010	0.1	0.011	0.11	0.0083	0.08	0.011	0.1	0.01
2'4 DDE	0.0017	0.02	0.0018	0.02	0.0018	0.02	0.0017	0.02	0.01
2'4 DDD	0.0016	0.02	0.0016	0.02	0.0014	0.01	0.0015	0.01	0.01
4'4' DDD	0.0029	0.03	0.0029	0.03	0.0032	0.03	0.0028	0.03	0.01
2'4 DDT	0.0011	0.01	0.0012	0.01	0.0004	0.004	0.0016	0.02	0.01
Endoslfan Sulphate	0.0064	0.1	0.0063	0.1	0.0068	0.11	0.0056	0.09	0.006
4'4 DDT	0.0079	0.08	0.0081	0.08	0.0054	0.05	0.0086	0.09	0.01
4'4 DDE	0.012	0.1	0.011	0.1	0.012	0.11	0.0010	0.1	0.01
Cypermethrin	0.087	0.2	0.086	0.2	0.0066	0.12	0.080	0.2	0.05

Abbreviations: yrs, years; B, boys; G, girls; µg/kg bw/day, micrograms per kilogram body weight per day; ADI, Acceptable Daily Intakes (*FAO, 1996*)

CONCLUSION

- The results of the study reveal that the dietary exposure to the contaminants investigated is much lower than the Acceptable Daily Intake in all the age groups that were computed.
- In specific cases, where the concentration of contaminants was high or where the consumption of a particular food was high, the exposures were also found to be higher.
- Risk assessment in vulnerable population like pregnant women should be done accurately as even the lowest concentration of the organochlorine pesticide residues, which were still found to be persisting in the diets may cause serious damage to the growing foetus which might lead to congenital deformities.

LIMITATIONS OF THE STUDY:

- The study is limited to rural areas.
- The samples were not collected in rainy seasons, therefore; the effect of rains on the occurrence of pesticide residues could not be assessed.
- Processed foods like jams, jellies etc. were not included for analyses and exposure assessment.

Study 2:

- Study area - Guntur Dist. Out of 3 revenue divisions- 2 were selected randomly
- Total no. of villages in 2 divisions- 571
- Total no. of inhabitants in each village-1000
- No. of villages selected- 24 Villages
- Cross sectional study
- Target population- Agricultural community (300no.)-Cotton cultivating farmers (exposed) and equal no. of controls (Un exposed- other than farmers)

METHODOLOGY

- Study design- stratified proportionate random sampling procedure.
- Biochemical parameters- RBC-AchE inhibition activity.
- Lipid peroxidation parameters- Catalase activity, Plasma TBARS.
- Antioxidant levels - reduced glutathione (GSH), Vit.E (α-tocopherol).
- Liver and Kidney functioning tests- AST, ALT, Creatinine and Urea.
- Statistical analysis- Two tailed student's 't' test was used to compare the mean difference between exposed and unexposed subjects for biochemical parameters.
- A post hoc analysis of Least Significant Difference (LSD) was used to assess the significance between the groups.

- Erythrocyte AchE, vitamin E and TBARS levels- determined using standard method-*Ellman et al (1961)*
- Lipid peroxidation quantified by plasma Thiobarbituric Acid Reactive Substances –spectrophotometric method (*Bhat, 1991*)
- Vitamin E was determined – HPLC method (*Allard et al. 1998*)
- Catalase activity -spectrophotometrically and Reduced Glutathione -fluorimetrically (*Aebi et al. 1974*)
- Liver function tests (serum ALT and AST) and kidney function tests (levels of creatinine and urea) - automated kits (supplied by M/s. Biosystems India Ltd)

RESULTS

70% (exposed subjects) were engaged in agriculture on leased land

-12.1% were agricultural labourers

-17.9% were other labourers

-Mean extent of land holding -5.9 acres

PROFILE OF THE STUDY SUBJECTS

Table 1. Profile of the study subjects.

Parameter	Exposed (n = 300)	Un- Exposed (n = 300)
Age (yrs)	37.8 (10.8)	37.3 (12.3)
Average (yrs) Smoking habit (any time)	42% (n = 100)	44% (n = 110)
Daily Smokers	60% (n = 100)	74%
Average smoking (per day)	7.0 (10.64)	9.1 (8.9)
Average tobacco chewing	6.9 (7.3)	4.7 (4.5)
Alcohol consumption (ml/ day)	195.8 (221.2)	196 (163.6)

Values are expressed as mean (standard deviation); $P > 0.05$.

MUSCARINIC SIGNS AND SYMPTOMS

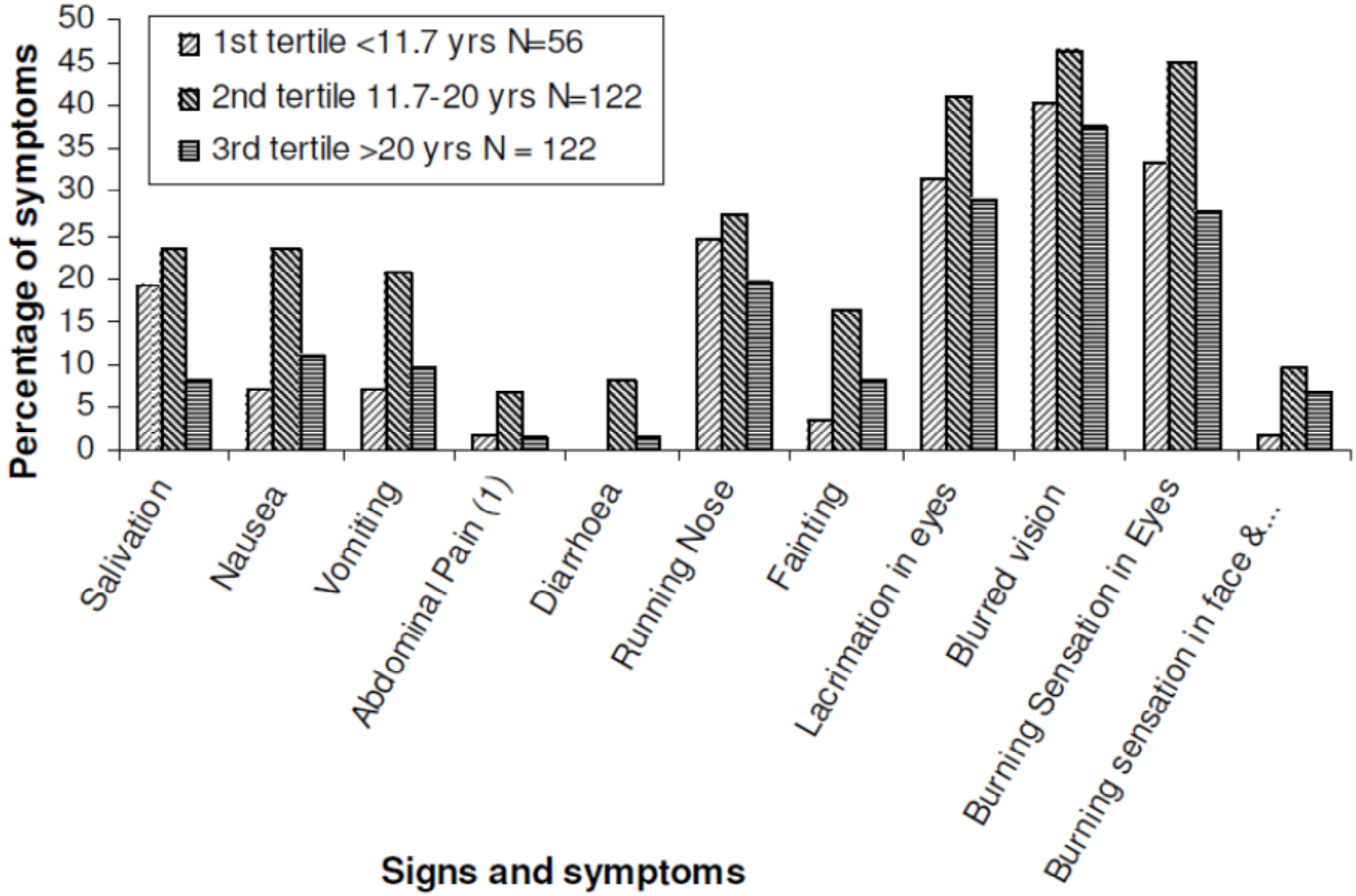

Figure 1. Muscarinic signs and symptoms (%) of the exposed subjects.

SIGNS AND SYMPTOMS OF CENTRAL NERVOUS SYSTEM

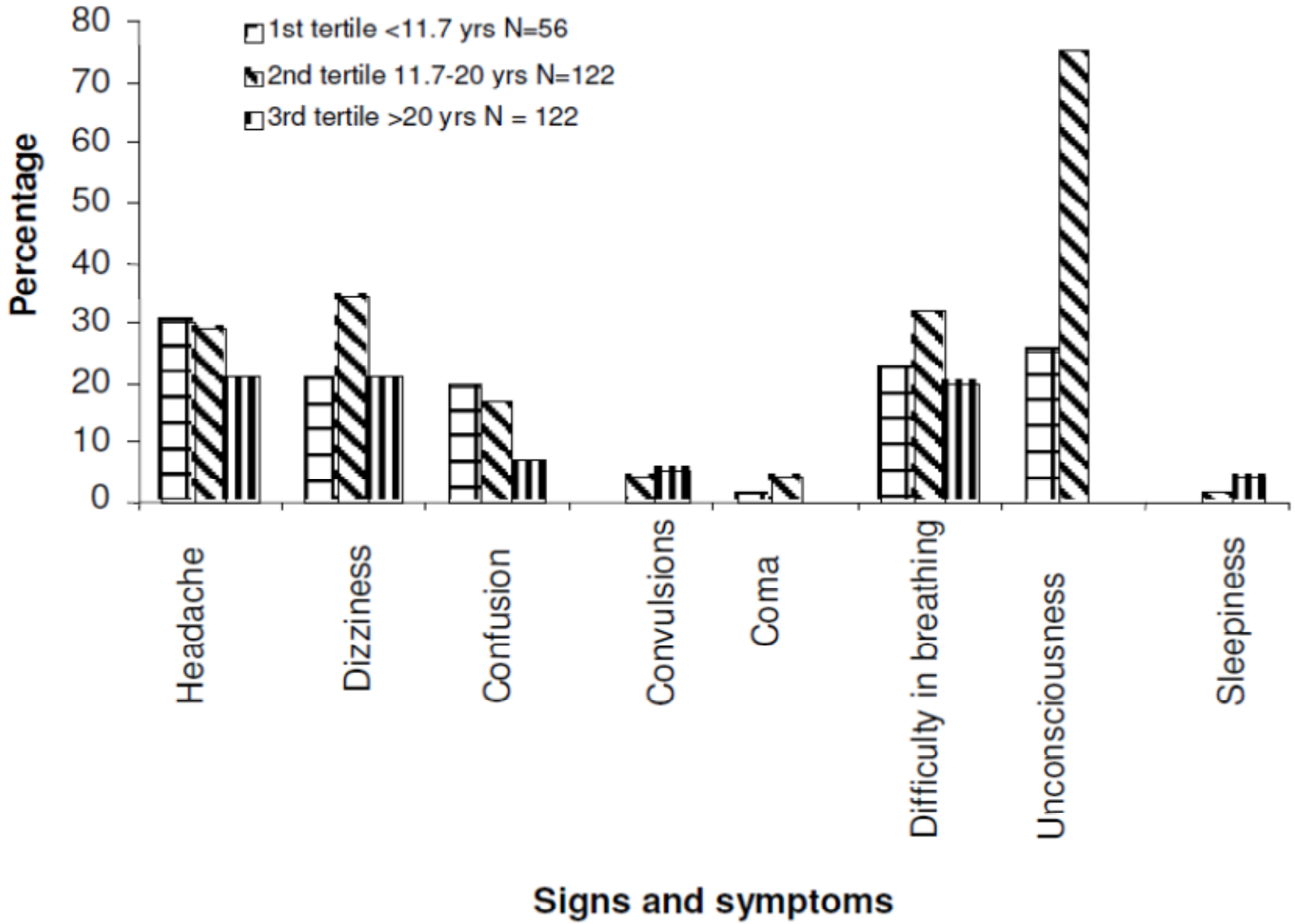

Figure 2. Signs and symptoms (%) of the central nervous system.

NICOTINIC SIGNS AND SYMPTOMS

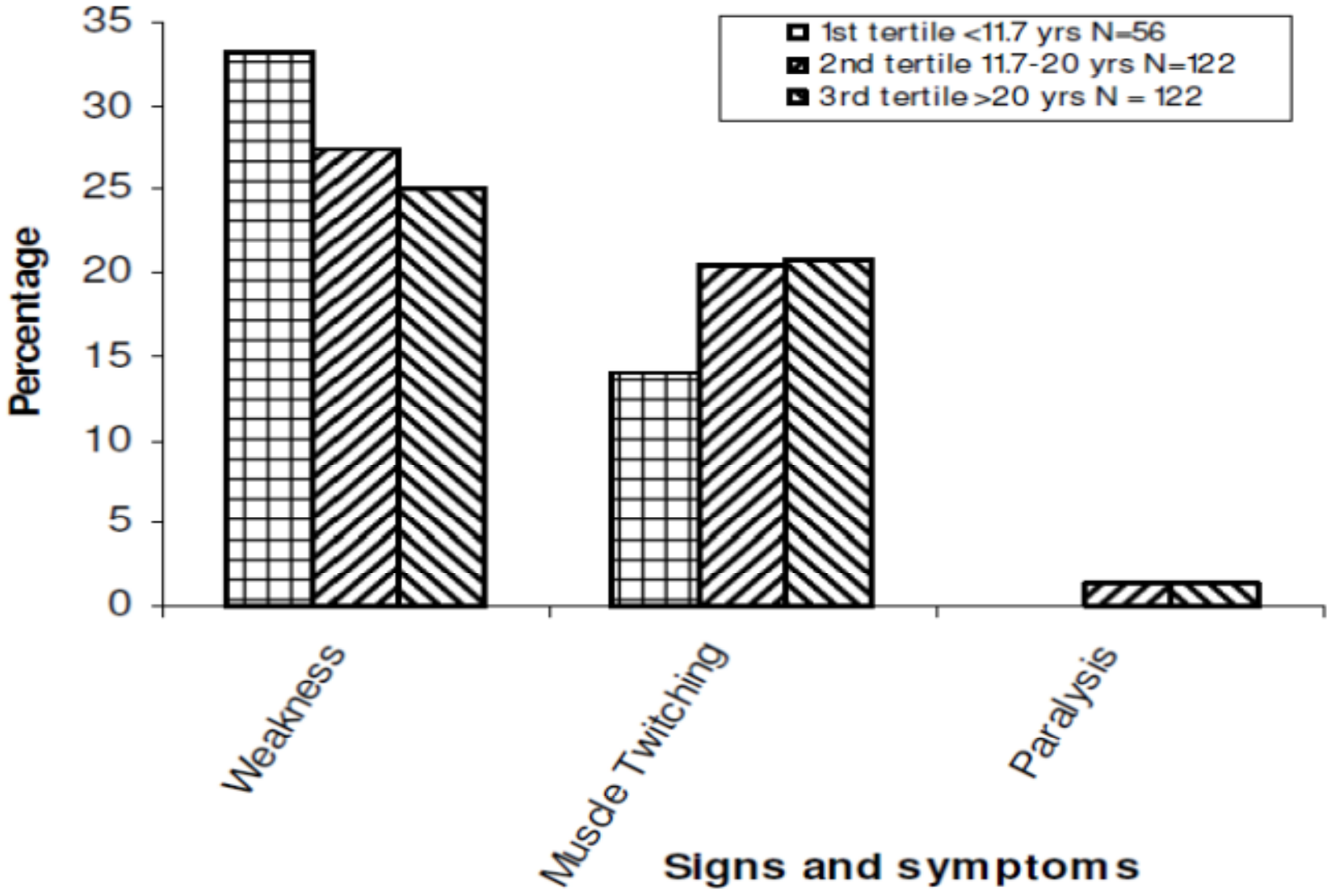

Figure 3. Nicotinic signs and symptoms (%) of the exposed subjects.

AchE levels VS various degrees of exposure to pesticides

Number of years of exposure	Erythrocyte AchE	95% CI
< 12 (N = 102)	1916.2 (282.95)[a]	1351.1 – 2481.2
12 - 20 (N = 130)	1786.0 (173.86)[ab]	1439.3 – 2132.8
> 20 (N = 68)	1317. 4 (81.34)[b]	1155.0 – 1479.8

Values of erythrocyte AchE are expressed as mean (standard error). Different superscripts are statistically significant at P < 0.05 by Least Significant Difference Method (LSD)

Liver and Kidney function parameters in exposed and unexposed subjects

Parameter	Group	N	Mean ± SE	95% CI
AST (IU/ml)	Exposed	224	22.79 ± 1.04*	20.75 - 24.83
	Unexposed	195	20.37 ± 0.57	19.3 - 21.5
ALT (IU/ml)	Exposed	224	16.59 ± 1.80	13.1 - 20.1
	Unexposed	195	13.29 ± 0.46	12.4 - 14.2
Creatinine (mg/dl)	Exposed	223	0.84 ± 0.029*	0.78 - 0.90
	Unexposed	220	0.75 ± 0.026	0.69 - 0.81
Urea (mg/dl)	Exposed	226	22.71 ± 0.91	20.9 - 24.5
	Unexposed	193	20.66 ± 0.95	- 24.6

*Significant at P < 0.05; SE = standard error; CI = class interval; N = total number of samples.

CATALASE ACTIVTY AND REDUCED GLUTATHIONE IN EXPOSED AND UNEXPOSED SUBJECTS

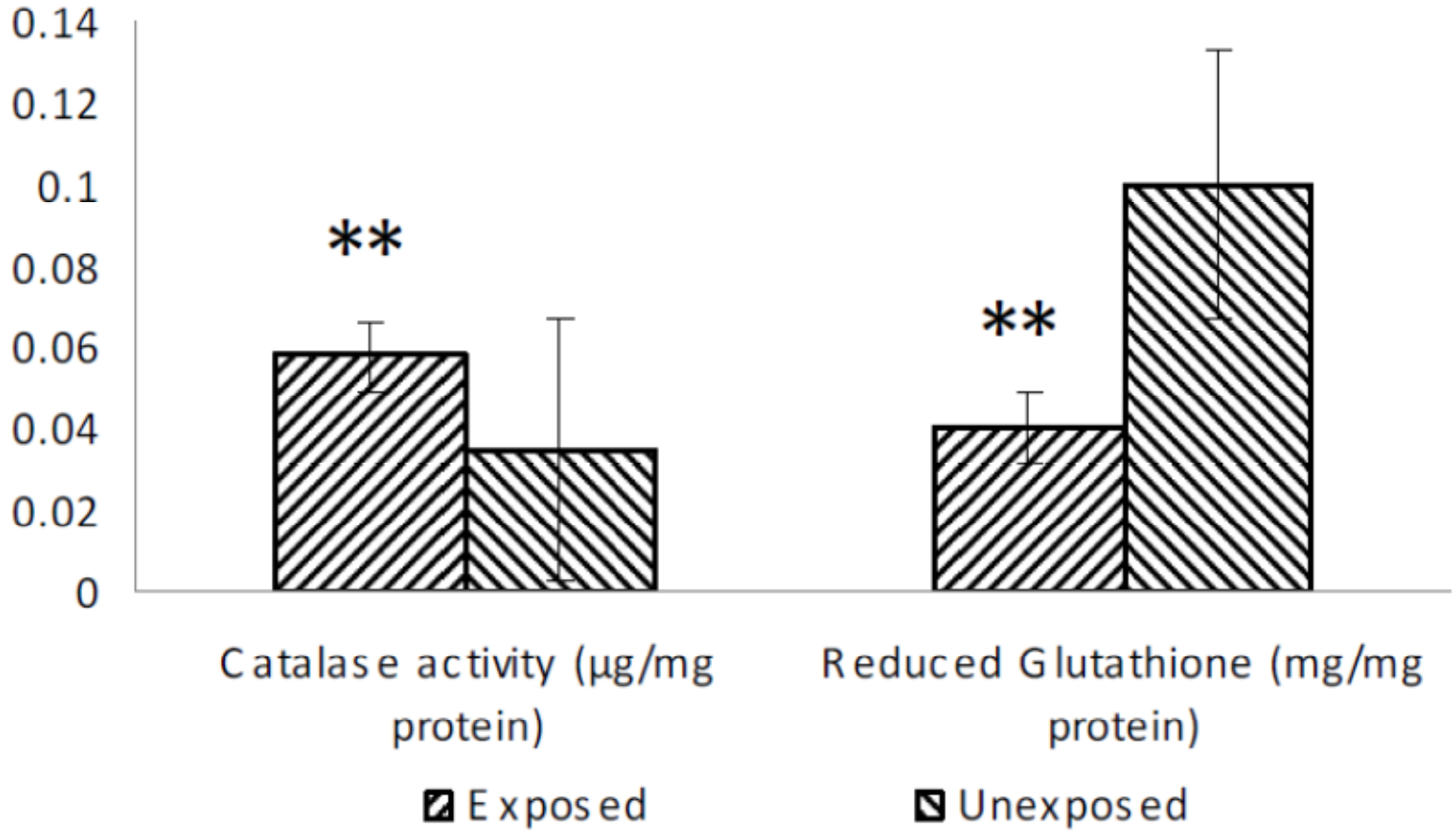

Figure 5. Catalase and reduced glutathione in the exposed and unexposed subjects. ** Significant at P<0.01.

TBARS AND α-TOCOPHEROL IN EXPOSED AND UNEXPOSED SUBJECTS

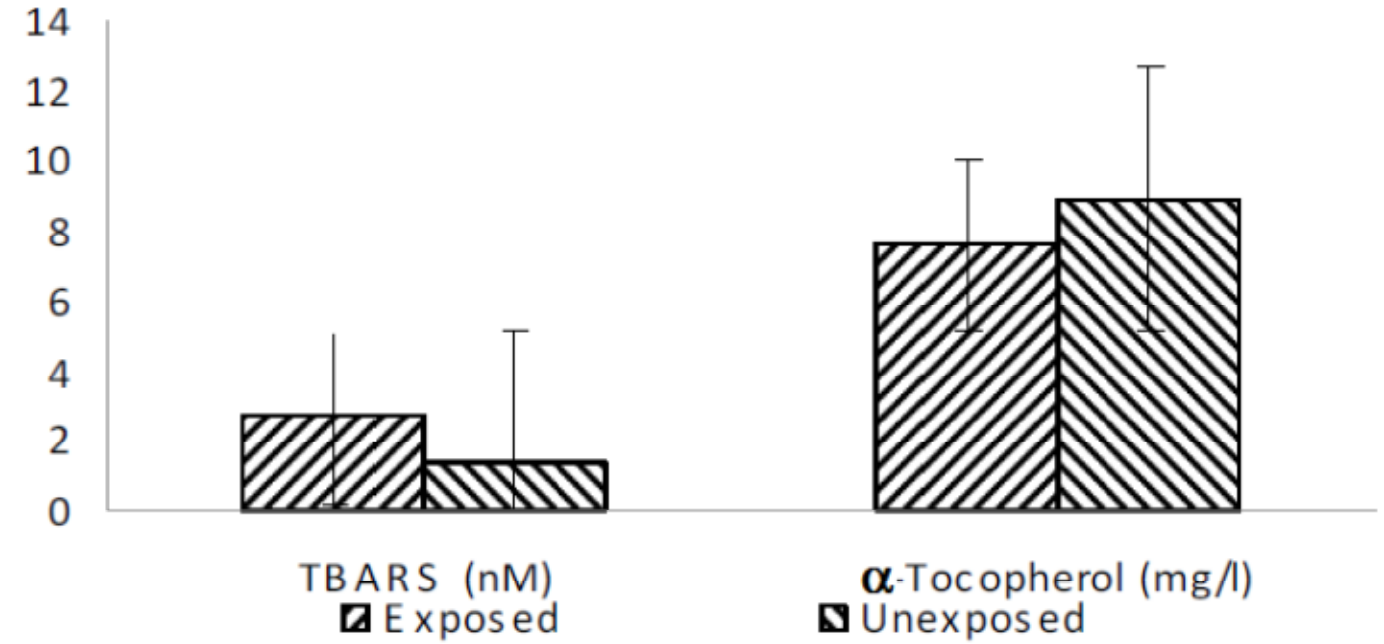

Figure 6. Plasma TBARS and α-tocopherol in the subjects.

CONCLUSIONS

- Many of the degenerative diseases will initiate at/after 40yr.s'/ later stage of life and the exposed subjects may become susceptible to non-communicable diseases at a later stage of life.
- Following up of the same subjects at later stages may provide some clinching evidence.
- Adequate steps should be initiated through Regional Farmers' Schools to impart pesticide safety education to the farming community involved in spraying activities.
- Usage of protective devices such as masks, gloves, aprons, shoes etc. should be encourage among farmers as it prevents from exposure to pesticides to a great extent.
- It is suggested that the farmers may adopt IPM practices where ever it is possible and also implement organic farming.

ENDOCRINE DISRUPTION

- Low doses of certain pesticides may mimic or block hormones or trigger inappropriate hormone activity

- Endocrine disruption may alter development and reproduction and induce birth defects

- Endocrine disruption has been linked to:
  - Infertility
  - Low sperm count
  - Early puberty
  - Hormone-dependent cancers (testicular, breast, prostate)
  - Altered sex ratio

(~Benoit Roig et al. Endocrine disrupting chemicals and Human health risk assessment: A critical review. Critical Reviews in Environmental Science and Technology. Nov 2012.

~C. Frye et al. Endocrine disrupters: a review of some sources, effects, and mechanisms of actions on behaviour and neuroendocrine systems. J Neuroendocrinol. 2012 January ; 24(1): 144–159.)

IMMUNOTOXICITY

❖ Immunotoxicity is suspected, but evidence is limited

❖ Studies in Artic zone:

- Higher incidence of ear infections
- Cytokine panel abnormalities

(~Schaalan MF et al. Correlation between maternal milk and infant serum levels of chlorinated pesticides (CP) and the impact of elevated CP on bleeding tendency and immune status in some infants in Egypt. J Immunotoxicol. 2012 Jan-Mar;9(1):15-24.

~Duramad P et al, Expression of Th1/Th2 cytokines in human blood after in vitro treatment with chlorpyrifos, and its metabolites, in combination with endotoxin LPS and allergen Der p1. J Appl Toxicol, 2006, 26(5): 458-65.)

PESTICIDES AND CHILDHOOD CANCER

Some studies have shown an association between postnatal pesticide exposure and an increased risk of pediatric cancer

- Brain tumors
- Acute lymphocytic leukemia
- Non-Hodgkin lymphoma

(~ Guodong Ding et al. Pyrethroid Pesticide Exposure and Risk of Childhood Acute Lymphocytic Leukemia in Shanghai. Environ. Sci. Technol., 2012, 46 (24), pp 13480–13487.
~ Mary H Ward et al. Residential Exposure to Polychlorinated Biphenyls and Organochlorine Pesticides and Risk of Childhood Leukemia. Environ Health Perspect. 2009 June; 117(6): 1007–1013.)

PRENATAL EXPOSURE AND CHILDHOOD CANCER

❖ Maternal exposure to pesticide has been associated with pediatric cancer – acute lymphocytic leukemia
❖ Association with parental occupation exposure

- Leukemia
- Brain cancer
- Hodgkin and non-Hodgkin lymphomas
- Kidney cancer

(~ Florence Vinson et al. Exposure to pesticides and risk of childhood cancer: a meta-analysis of recent epidemiological studies. Occup Environ Med *2011; 68: 694-702.*
~ Donald T Wigle et al. A Systematic Review and Meta-analysis of Childhood Leukemia and Parental Occupational Pesticide Exposure. Environ Health Perspect. 2009 October; 117(10): 1505–1513.)

CASE STUDIES

(Reference: US EPA Website Organophosphate Pesticide Information)

1:

- **A family with two children ages 8 months and 6 years are referred to the clinic with concerns of pesticide exposure**
- **They are worried because their apartment was sprayed with pesticides for termites 3 times when the mother was pregnant with her 6 year old son**
- **She remembers feeling dizzy and fatigued during that period but attributed it to the pregnancy**

2:

- **Her six year old was born at low birth weight for gestational age and has been behind in meeting developmental milestones**
- **He now has behavioural problems and is behind in reading in school**
- **She recently read in the newspaper that chlorpyrifos is not being used in residential settings in the US because it stays on surfaces for long periods of time and may have harmful health effects**

3:

- **She is wondering if her son's low birth weight and difficulties in school could be due to chlorpyrifos exposure**
- **She asks if she should be worried about her 8 month old being exposed given that chlorpyrifos can stay on surfaces for long periods of time**

4:

- **Apartment was sprayed with chlorpyrifos 3 times during pregnancy with 6 yr old son She was dizzy and fatigued surrounding the times of spraying**
- **She is wondering if her six year old son's low birth weight and difficulties in school could be due to the chlorpyrifos exposure**
- **She asks if she should be worried about her 8 month old being exposed given that chlorpyrifos can stay on surfaces for long periods of time**

5:

- **What recommendations would you give to this family?**
  - **Primary prevention!**
  - **Encourage home dust/furniture testing if they think exposure is ongoing**
  - **Encourage blood testing for acetyl cholinesterase if they are worried about ongoing exposures**
  - **Encourage alternatives to pesticides for the future**

PREVENTION – NATIONAL LEVEL

- ❖ Education campaigns aimed at pesticide users, general population and children
- ❖ Restrict availability or limit use
- ❖ Establish and monitor maximum residue limits
- ❖ Surveillance and epidemiological vigilance for acute and chronic related illness
- ❖ Treatment capacities
  - Emergency services
  - Poison control centers
  - Education of health care providers

References:
•UNEP Chemicals. *Childhood Pesticides Poisoning. Information for Advocacy and Action. May 2004* (www.who.int/ceh/publications/en/pestpoisoning.pdf).
•US EPA Website Food Quality Protection Act, 1996. (www.epa.gov/oppfead1/fqpa/backgrnd.htm).


Thank You