

PESTICIDE USE IN NIGERIA AND THE HEALTH IMPLICATIONS

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ABSTRACT

The increase in the application of pesticides to enhance agricultural production in Nigeria has been examined. Most of the pesticides used are imported into the country by multinational companies. The health implications of the active ingredients of these pesticides with particular reference to their mutagenicity and carcinogenicity have been assessed. Though most have been found to be mutagenic in the microbial system, and some carcinogenic in mammals, only a few are known to be carcinogenic in man.

The need to increase public awareness of indiscriminate use of pesticides with the resultant hazardous effect of uncontrolled exposure has been highlighted

INTRODUCTION

Even though the harmful effects of large doses of toxic chemicals have been recognised for a long time, the cumulative effects of small doses over a long period of human exposure were not anticipated until about two decades ago. Chemical mutagenesis was first reported shortly after the Second World War (Auerbach and Robson, 1946). Thereafter, the detection and evaluation of environmental mutagens became a major scientific challenge.

The suspected relationship between mutagenesis and carcinogenesis added another dimension to the interest already generated in this field. Consequently, within the last decade, various mutagenicity and carcinogenicity testing assays have been developed and large numbers of mutagens and carcinogens have been detected. As a matter of fact, the risk associated with environmental mutagens and carcinogens is now a major concern.

The problems of environmental mutagens and carcinogens is a universal one which confronts both the developed and developing countries. For this reason, the problem has received considerable attention from international bodies such as United National Environmental Programme (UNEP), International Agency for Research on Cancer (IARC), International Association of Environmental Mutagens Societies (IAEMS) and the World Health Organization (WHO). There is the need however, for each country or region to complement the efforts of these international bodies by analysing local or regional problems as well as utilizing local resources for solving the problems. In this regard, the developed countries have been in the forefront while the developing countries have lagged behind.

Acquisition of expertise in the field of environmental mutagenesis and carcinogenesis by local scientists is essential in order to solve peculiar local problems, and also contribute to the expansion of the frontiers of knowledge universally, in the area of genetic toxicology.

Training of experts in this area, though important, may not receive immediate and necessary attention where the problems of unemployment, contagious diseases, shortage of food, treated water and energy, are considered to be of priority. The few experts that are available are confronted with the problem of scientific communication, which is a vital tool in research. Discussions on new scientific discoveries and research strategies take place in international journals and conferences, the benefits of which are for economic reasons, not within the easy reach of scientists in most developing countries, for exploitation. Consequently, scientific information diffuse very slowly to developing countries and the implication of this can be grave in terms of environmental mutagens and carcinogens.

Yet the problems of environmental mutagens and carcinogens must be tackled from the local perspective. Since the local expertise are not adequately equipped to carry out mutagenicity and carcinogenicity testings at the moment, the primary source of information on the genotoxic risks of chemicals found in the Nigerian environment inevitably will come from reported data on chemicals. The objective of this paper is to evaluate, based on information available in literature, pesticides as sources of environmental mutagens and carcinogens and the implications for public health in Nigeria.

GROWING UTILIZATION OF PESTICIDES

The use of agro-chemicals to increase agricultural output has become necessary in a country like Nigeria, which is faced with the rising cost of food importation. Moreover, there is a need to increase agricultural output for export so that the economy of the country does not depend on petroleum which is subject to the vagaries of international trade and politics. The tropical climate of Nigeria favours a large number of plant and animal species which have been classified as pests by agricultural biologists. These have posed a great problem to the local farmer who had to resort to the use of pesticides. In most cases the utilization has been extensive, uncontrolled and not in accordance with recommended procedure and the consequence has been an uncontrolled human exposure to those chemicals. In fact some of the chemicals have been banned in the developed countries.

There is every indication that the importation of agro-chemicals will continue to be on the increase and there is the possibility that the indiscriminate use by local farmers will lead to human exposure beyond the permissible level.

HOUSEHOLD INSECTICIDES AND AGRICULTURAL PESTICIDES UTILIZED IN NIGERIA

Tables 1 and 2 present a list of household insecticides and agricultural pesticides available in the Nigerian market. Most of the formulations share com-

mon active ingredients. Though all the companies which market these chemical compounds are registered in Nigeria, they are multinational companies with the parent company based outside Africa.

Not all pesticides constitute health hazard from the information available on their genotoxicology. The class of pesticides that pose a great risk to human health are the organochlorines and the urea derivatives. Organophosphorus pesticides do not pose serious residue problems because they react with water as neophilles and form diesters of phosphoric acids, alcohols and phenols which can further be hydrolyzed to monoesters and eventually to inorganic phosphates (Wild 1975, Seiler 1977).

Organochlorines pesticides accumulate more readily in aquatic microorganisms which form the broad base of aquatic food chain. Table 3 presents organochlorine pesticide residues in a sample of fish species in Nigerian water. Pesticide residues in the soil (shown in Table 4) and in aquatic organisms can eventually get into man. Table 5 and 6 show the level of pesticide residues in the fat and blood of a sample of Nigerians respectively.

Urea derivatives which by themselves have insignificant genetic hazards have also been suggested to do so when nitrosated (Seiler, 1977). Nitrosation *in vivo* as a result of high nitrate food content is possible in a country like Nigeria where fertilizers are used without any control.

Table 1: HOUSEHOLD PESTICIDES AVAILABLE IN NIGERIA

Trade Mark	Ingredients	Country of Origin	Manufacturer
Afetox	DDVP or Dichlorvos	U.K.	Afema Chemical Industries
Attack	DDVP, Tetramethrin, Permethrin	U.K.	Jeyes Overseas Ltd.
Baycon	Propoxur, Dichlorvos	U.K.	
Best	DDVP, Sumithion Neopynamin	Holland	Intra Dal Ltd.
Black Flay	5 - Benzy-3-Funyl Methyl Chloropropane Carbonylate	U.S.	Boyle Company
Big D.	BHC. Synthetic Pyrethrin	U.K.	Domestic Fillers Ltd

Cooper	Diethyl-2-Isopropyl-6-Methyl-4-Pyrimidinyl Phosphorothionate	U.K.	Cooper and Robertson Limited
Denthalac	Diazinon	Diazinon	F.C. Dewitt Co. Ltd
Famid 80 WP	Atropin	Atropin	Giba-Geigy
Flak	Pyrethrin, DDVP, Propynyl Butoxide	U.K.	Murray Clark and Jones
Fly Tex	Not Indicated	Holland	
Fly Tox	Azamethidhos and Dichlorvos	France	Giba-Geigy
Fly and Wasp Killer	BCH and Synthetic Pyrethrin	U.K.	Domestic Fillers Ltd
Killer	KF 60	France	AGIR
Keen	BCH, Pyrethrin Propynyl Butoxide	U.K.	E.E.C. Keen Ltd.
Mobil	Neopynamium, Propynyl, Butoxide, DDVP, Lindane, Sumithion	Holland	Mobil Oil Nig. Ltd.
Moth Proffer	Lindane and Synthetic Pyrethrin	U.K.	Repto Kill Ltd. England
Off Repellant	Diethyltoluamide	U.K.	Sohron Wax Ltd.
Ozek	Pyrethroids, Piperonyl Butoxide N-Dicarboximide	U.K.	G.H. Wood Company Eng.
PAF-PAF	Pyrethroids	U.K.	Cooper, McDougal & Roserton Ltd. England.
Quench		U.K.	National Oil Product Nig. Ltd.

Shelltox	Penitrothion, Dichlorvos	U.K.	National Oil Nig. Ltd.
Super Raid	Tetramethrin, Dichlorvos, Piperonyl, Butoxide	U.K.	John Wax Nig. Ltd.
Super TID	Propernyl Butoxide Shell Sol. K.	France	Miranto France
Tox	BCH, Pyrethrin Propynyl Butoxide	U.K.	Keen Ltd., England
Tus	Dichlorvos, Octachloro Dipropyl, Ehter, Epichlorohydrin		Phermakon-vienna
Uniflitt	Pyrethrin, DDVP, Propynyl Butoxide, Neopynamin		Unipetrol Ltd., Nigeria

Table 2: LIST OF INSECTICIDES AVAILABLE IN NIGERIA

Trade Name of Product	Active Ingredient	Country of origin	Manufacturer/ Nigerian Distributor
Abolant Killer	Lindane (&BHC)	U.K.	Chemical & Allied Products, Nig. Ltd.
Afrol 15	Lindane	U.K.	"
Agrocide 26DP	Lindane	U.K.	"
Albolineum	Refined Mineral Oil	U.K.	"
Actellic 25EC		U.K.	"
Actellic 2% Dust		U.K.	"
Actellic 50% EC		U.K.	"
Actellic Aerosol		U.K.	"
Ambush 25 E.C.		U.K.	"

Table 2 Contd.

Trade Name of Product	Active Ingredient	Country of origin	Manufacture/Nigerian Distributor
Aldrin 27% Dust	Aldrin	U.K.	National Chemical Limited
Aldrex 40 E.C.		U.K.	"
Azodrin		U.K.	"
Akar	Chlorobenzilate	Swiss	CIBA-GEIGY (Nig.) Limited
Arkotine D 25	DDT	U.K.	National Chemicals Ltd.
Basudin	Diazinon	Swiss	CIBA-GEIBY (Nig.) Ltd.
Bidrin	Dicrotophos	U.K.	National Chemicals Ltd.
Carbicon	Dicrotophos	Swiss	CIBA-GEIGY (Nig.) LTD.
Capsid-Tox 20	Lindane	Paris	Nigerian Hoechst Co. Ltd.
Cymbus 25 EC		U.K.	Chemical and Allied Products Ltd.
Cymbus 10 EC		U.K.	"
Dimcron	Phosphamidon	Swiss	CIBA-GEIGY Nig. Ltd.
Difolatan 80 WP		France	Chemical & Allied Products
Dieldrex 40 EC	Dieldrin	U.K.	National Chemicals Ltd.
Dieldrin 75% WP	Dieldrin	U.K.	National Chemicals Ltd.

Table 2 Contd.

Trade Name of Product	Active Ingredient	Country of origin	Manufacturer/ Nigerian Distributor
Deltia Gas	Phosphane	Germany	Nigerian Hoechst Comp. Ltd.
Decis	Delta Methrine	Paris	"
Dipterex	Trichlorphon		Bayer, Ag.G. Nig. Ltd.
Elecron	Dioxacarbamate	Swiss	CIBA-GEIGY Nig. Ltd.
Fernasan D	Thiram + Lindane	U.K.	Chemical & Allied Products
Gammalin 20	Lindane	U.K.	"
Gammexane 20	Lindane	U.K.	"
Galecron	Chlorodine Form	Swiss	CIBA-GEIGY Nig. Ltd.
Gardana 75% WP		U.K.	National Chemicals
Klerat	Brodifocoum	U.K.	Chemical & Allied Products Limited
Kokotene	Lindane	U.K.	National Oil and Chemical Marketing Comp.
Lindane Dust	Lindane	U.K.	National Oil and Chemical Marketing Comp.
Lindane Dust	Lindane	U.K.	"
Malathion	Malathion	U.K.	National Chemicals Ltd.
Mixol 20	Lindane	U.K.	National Chemicals Ltd.

Nogos	Dichloruous.	Swiss	CIBA-GEIGY Nig. Ltd.
Neoron	Bromopropilate	Swiss	"
Nuvacron	Monocrotophos	Swiss	"
Nuvan	Dichlorvos	Swiss	CIBA-GEIBY Nig. Ltd.
Phestoxan Tab			National Chemicals Ltd.
Perenox		U.K.	Chemical & Allied Products
Prexervor	Carbaryl	Paris	Hoechst
Rospin	Chloropropylate	Swiss	CIBA-GEIGY Nig. Ltd.
Sapona 20 E.C.		U.K.	National Chemicals Ltd.
Sapona Saclets		U.K.	National Chemicals Ltd.
Sofratox	Methyl Parathion	Paris	Hoechst
Synexa 25	Lindane		Hoechst
Thiodan 35 EC	Endosulfan	Germany	Hoechst
Thiodan 35 ULV	Endosulfan	Germany	Hoechst
Vapona 48% EC	Dichlorvos	U.K.	National Chemicals Ltd.
Vetox 85% WP	Carbaryl	U.K.	National Chemicals Ltd.
Vetox 5% Dust	Carbaryl	U.K.	National Chemicals Ltd.

Table 3

Organochlorine Pesticides Residue in Nigerian Fish

	Concentration in $\mu\text{g g}^{-1}$ Fat Weight Basis							
	% Fat	r-BHC	Dieldrin	DDE pp	DDD pp	DDT pp	DDT pp	Total DDT
Obokun type	1.7	35.2	10.2	0.39	0.38	1.35	1.35	2.1
Cat Fish	2.7	5.4	1.2	0.16	0.14	ND	ND	0.3
Eja Aro	2.5	12.7	8.8	0.85	1.31	ND	ND	2.2
Eja Aro Fish Egg	0.1	0.8	0.5	0.03	0.04	ND	ND	0.07
IITA Lake Aro Fish	0.5	0.01	N.E	0.03	0.03	ND	ND	0.06
IITA Tilapia Fish	0.8	0.02	N.E	0.02	0.03	ND	ND	0.05

From Osibayo and Jensen (1980)

Table 4 Chlorinated Hydrocarbons Residues in Some Ibadan Soils

Soil Around Cocoa Research Institute (CRIN) Water Treatment Plant	Concentration in Ug g - 1 dry weight soil									
	r-BHC	Aldrin	Dieldrin	DDEpp	DDT pp	DDD pp	DUT pp	E DUT	1.11 DDE	1.11 DDD* DDT
CRIN Plot N1	2.1	1.3	1.7	0.1	0.09	-	0.07	-	-	0.28
CRIN Plot N1/I	6.5	2.5	4.2	8.2	3.4	-	20.9	-	-	33.4
CRIN Plot W4	3.8	1.2	11.8	2.7	-	-	5.3	-	-	8.3
CRIN Plot W6	4.6	1.4	5.1	13.5	4.3	-	28.4	-	-	5.1
NCRI Herbicide trial Plot H4	7.8	-	9.2	36.0	16.3	5.3	172.0	-	-	234.1
NCRI Herbicide trial Plot H3	19.4	1.1	12.1	1.7	7.2	-	12.4	-	-	21.5
	24.7	1.6	12.2	1.8	6.4	-	5.1	-	-	15.7

From Osibayo and Jensen (1980)

Table 5: Organochlorine Pesticide Residues in Nigeria in Human Blood

Sample Code	Fat	Concentration Ug g - 1 Fat Weight				Total DDT
		2-BHC	Dieldrin	DDE pp	DDD pp	
MA	0.25	0.65	2.6	4.9	7.4	14.9
FA	0.28	0.67	2.7	ND	ND	2.7
MA	0.31	0.12	1.1	NE	NE	1.1
FA	0.32	0.01	0.28	ND	ND	4.5
MA	0.47	Trace, NE	1.9	ND	ND	1.9
MA	0.37	0.89	0.56	NE	NE	0.6
FA	0.32	0.98	3.0	NE	NE	3.0
MA	0.48	Trace, NE	2.7	ND	ND	2.7
MA	0.29	Trace, NE	1.4	ND	ND	1.4
MA	0.32	0.13	0.5	ND	ND	0.5
MA (8 yrs.)	0.29	Trace, NE	0.02	0.05	ND	0.07
MC (12 yrs.)	0.25	Trace, NE	0.38	ND	ND	0.18
MC (6 yrs.)	0.25	Trace, NE	0.28	0.23	ND	0.51
MA	0.41	0.03	0.19	0.02	ND	0.21
MA	0.24	Trace, NE	0.20	NE	NE	0.20
MA	0.21	Trace, NE	0.14	ND	ND	0.14
MA	0.28	0.42	5.6	NE	NE	5.6
MA	0.23	0.03	0.24	0.02	ND	0.26
MA	0.34	0.04	0.32	0.04	ND	0.36
MA	0.29	0.04	0.23	0.03	ND	0.26

From Osibayo and Jensen (1980)

Table 6: Level of Pesticides Residues in Human Fat

	Level in Human Fat (PPM)		
	U.S.A.	U.K.	NIGERIA
Lindane	—	—	0.03 — 0.6
BHC	—	0.20	0.16 — 0.30
DUT	1.54	0.52	—
PP DCE	4.58	1.8	1.4 — 2.5
Total DDT	8.00	2.5	6.5
Dieldrin	0.34	0.16	0.02 — 0.13
Heptachlor Epodde lde	0.13	0.03	0.004 — 0.02

From Osibayo and Jensen (1980)

MUTAGENICITY AND CARCINOGENICITY.

Table 7 is a list of pesticides available in Nigeria on which information is available with regard to their mutagenicity and carcinogenicity.

Aldrin

Aldrin is one of the pesticides that have been thoroughly investigated in respect of its mutagenicity and carcinogenicity. Experiments with microbial assays, *E. coli*, *S. typhimurium* and Yeast did not produce results (Moriya et al, 1983). Grants (1973) and Georgian (1975) both reported the clastogenic effects of Aldrin in mammalian assay. There are contradictory results from experiments evaluating the carcinogenicity of Aldrin. In mice and rats when introduced orally, Aldrin was found to be carcinogenic in mice, producing malignant liver neoplasm and thyroid tumor in rat. Other studies with rats produced either negative or inadequate results (Deichmann et al, 1979). Evidence for carcinogenicity of Aldrin to humans is inadequate (IARC, 1982) as analytic epidemiological studies are scanty and difficult because of many compounding factors.

Table 7: List of Pesticides Available in Nigeria on Which Genotoxic Studies have been Carried Out

Pesticide	Mutagenic Evidence to Micro-Organisms	Mutagenic Evidence to Mammals	Carcinogenic Evidence to Animals (Mice)
Aldrin	—	++	++
BHC	—	+	++
Carbaryl	—	—	+
D D T	—	++	+++
Dieldrin	++	++	++
Dichlorvos	+++	+	—
Dichrotophos	===	+	—
Diazinon	—	—	—
Malathion	—	+	—
Parathion	—	+	—
Methylparathion	++	+	—
Chlorobromuron	+	—	+
Chloroturon	+	—	+
Fluomoturon	+	—	+
Motobromuron	+	—	+
Chloroxuren	+	—	+
Endosultan		+	+
Atrazine	+	++	+
Hyrazine	+	++	++
Simazine	+	++	+
2, 4 - D	—	++++	—

— Negative Evidence

+ Limited Evidence

++ Inadequate Evidence

+++ Sufficient Evidence

BHc (Lindane)

Extensive studies with Lindane did not produce any positive results in bacteria and yeast. In both cases Lindane did not induce mutation (Fahring, 1974; Van Dijck and Van de voorde, 1976). Mutagenicity testing with *Droso-*

Drosophila melanogaster also did not produce a positive result. However, Lindane was able to induce polyploidy, mitotic arrests and some chromosomal anomalies in human lymphocytes and Chinese hamster cells *in vitro*.

Lindane has been found to be carcinogenic to mice when administered orally, producing liver tumours. Nevertheless, evidence that Lindane is conclusively carcinogenic to animals is still limited. Similarly, evidence for carcinogenicity of Lindane to humans is inadequate, though three cases of leukaemia were reported in men exposed to it, cases of aplastic anaemia have been associated with exposure to it and the tissue level of this chemical compound has been reported high in a case of cancer.

Carbaryl

It has been very difficult to interpret the results of mutagenicity tests on carbamates. While it has shown strong mutagenicity in *S. typhimurium* when nitrosated, (Elespuru, 1974; Blevins, 1977), it has produced negative results with other microbial assays. In both *in vitro* and *in vivo* mammalian tests, spindle poisoning and chromosome stickiness were the visible effects of Carbamate (Styles, 1973).

No conclusive statement can be made about the status of carbamates as a carcinogen because results obtained from experiments have been conflicting.

DDT

Most mutagenicity testings carried out for DDT using microbial systems have been negative (Shirasu, 1976; Bartsch et al, 1980; Moriya, 1983). However, DDT has been reported to increase the mean number of chromosome breaks in cultures from exposed pesticide workers; induce dominant lethal mutation in rats and block DNA synthesis in *S. notophore*, in rats, sea urchin embryo and plants.

Oral administration of DDT in rat caused nodular hyperplasia, hepatocellular carcinoma (Kimbrough and Linder, 1974, Kimrough, 1974), benign and malignant neoplasm, lymphomas and lung neoplasm. Elevated level of DDT has been found in persons with various forms of cancer (Kimbrough, 1974; IARC Monograph, 1982).

Dichlorvos

Both positive and negative results have been obtained for Dichlorvos in mutagenicity testings (Hauna and Dyer, 1975). Under conditions of normal usage as an insecticide, Dichlorvos most likely cannot exact significant mutagenic effects in mammals including man. Lymphocytes treated with Dichlorvos did not show an clastogenic effect. However, there was an increase of 6.5% above the mean frequency of chromated break as reported by Wild (1975) and chromosome aberrations in blood samples obtained from insecticide applicators during the spraying season. (Czeicel et al, 1973). Although it did not induce dominant lethal mutation in mice (Dean et al, 1976), it induced breaks in plasmid DNA.

Dichlorvos appeared to be a potent mutagen when tested in microbial assays. (Shirasu, 1976; Moriya et al, 1983).

Carcinogenicity tests with Dichlorvos produced inconclusive results. (Wild, 1975).

Dicrotophos

Although Dicrotophos has been demonstrated to be mutagenic in bacteria and yeast (Wild, 1975), it did not produce significant clastogenic effects in spermatogenic cells. Czeizel et al (1973) reported that Dicrotophos produced chromosome aberrations in human in a condition of acute intoxication. Studies of the carcinogenicity of Dicrotophos are scanty and no definite conclusion can be reached on their results.

Dieldrin

Although Dieldrin produced chromosome aberrations in mouse bone marrow cells and human embryonic lung cells *in vitro* (Ahmed et al, 1977; Majumdar et al, 1976) several reports showed that Dieldrin produced negative results in mutagenicity testing. (Shirasu et al, 1976; Darrel and Water, 1978; Ashwood, 1981; Moriya, 1983).

The reports on the carcinogenicity of Dieldrin have been contradictory. While Walker et al (1969) reported that Dieldrin failed to induce tumor in rats fed with Dieldrin, elevated serum level of Dieldrin has been reported in cases of cancer (Caldwell et al, 1981).

According to IARC monograph (1982) evidence for carcinogenicity and mutagenicity of Dieldrin is still inadequate.

Endosulfan

Although chromosomal aberrations have been reported in pesticide applicators exposed to endosulfan and other pesticides (Yoder and Walson, 1974), endosulfan has been reported to cause no significant increase in tumor after oral administration in mice.

Malathion

Mutagenicity tests with Malathion produced negative result in microbial systems (Shirasu et al, 1976; Moriya et al, 1983). However, it has been reported to induce significant increase in chromosome aberrations *in vitro* (Czeizel et al, 1973) and break in DNA (Darrel and Walter, 1978).

Methyl Parathion and Parathion

Studies with methyl parathion and parathion in both *in vitro* experiment and exposed workers have not shown that they are either mutagenic or carcinogenic. Negative results have been obtained in microbial system (Wild, 1975) and mamma-

lian in vitro assay (Rita, 1982). However, Van bao et al (1974) had reported an increased chromosome aberration in 5 patients who had suffered acute intoxication with methyl parathion.

Paraquat

Evidence for mutagenicity of paraquat is still inadequate. It showed negative results when tested with *S. cerevisiae* (Siebert and Lemperle, 1974) and bacteria (Moriya et al, 1983). Sieler (1977) however obtained positive results with *Salmonella typhimurium*.

Triazine and Amine Derivatives

Maleic hydrazine has been reported to be mutagenic in *Drosophila* and to induce mitotic inhibition and chromosomal aberrations in *Vicia faba*, *Allium sp.* and maize. Simazine are reported to be mutagenic. Trivazine induced polyploidy in mammalian cells (Grant, 1973). Both Atrazine and Simazine were reported to induce tumors after oral administration in rodents.

2, 4-Dichlorophenoxy acetic acid

2,4-D has been reported to cause no significant increase in the rate of mutation (Styles, 1973). However, it was claimed to cause chromosome stickness and endopolyploidy (Grant, 1973). It was reported to increase chromosome aberrations in human fibroblasts cultures (Yoder et al, 1974, Seiler, 1977). Kastenbaun and Bowman (1970) reported 2,4-D as being slightly mutagenic in *Drosophila* in recessive lethal tests.

Ronchi et al (1976) showed a positive correlation between polyploidy, chromosomal aberrations and 2,4-D concentration in plant tissue cultures. 2,4-D also induced chromosome aberrations in *Allium*, abnormal mitosis in *Vicia* and mutation in yeast (Seibert et al, 1974). Nevertheless, negative results have been obtained in tests with microbial systems (Shirasu, 1976; Moriya et al, 1983). There is no evidence for carcinogenicity of 2,4-D.

Urea Derivatives

Although most urea derivatives have been reported to show weak mutagenicity in bacterial assay, Couch (1975) suggested that nitrosation of urea derivatives can produce clastogenic effects.

Ethylene thioures has been reported to be definitely carcinogenic. (Weisburger et al, 1981).

CONCLUSION

Recent advances in carcinogenesis have revealed that there are two categories of carcinogens. These are the initiators and the promoters. An initiator is a mutagen because the first stage in carcinogenesis is mutation. However, a promoter is not

likely to be a mutagen if it is not an initiator. While there are chemicals which are both initiators and promoters, some are either initiators or promoters. The fact that a pesticide is not mutagenic does not rule out its being carcinogenic. For instance, mutagenicity testings carried out for DDT using microbial systems have been negative. Yet elevated level of DDT has been reported in persons with various forms of cancer. Caution must therefore be exercised in recommending the use of a pesticide even though results of mutagenicity testings have been negative. Since most users and applicators of pesticides are farmers in the rural areas, a deliberate effort must be made by marketers of these products, agriculture extension workers and health educators to highlight the hazardous effects of pesticides when not used according to prescription.

Apart from the residue problem of pesticides, applicators are at a high risk due to uncontrolled exposure since the applicator handles the chemicals in the most concentrated and therefore the most hazardous form. The correct use of pesticides will reduce the level of residue problem and degree of exposure of applicators.

The establishment of the Federal Environmental Protection Agency is timely. As it is done in the developed countries, the Agency should ensure that all pesticides to be used in Nigeria are properly screened for mutagenicity and carcinogenicity. Only those which produce negative results should be allowed into the market. Manufacturer's information should not be relied upon solely. The Agency should also consider encouraging marketers and manufacturers to embark on work site health education through which relevant information on their products are passed on to users and applicators in particular. Positive health action would be taken when individuals have perceived their vulnerability to a disease and assessed the benefits and costs of the required action to be taken. Such health education should emphasise the utilization of protective gadgets by applicators. It should emphasise the need for regular medical check up to ensure that hazards are detected early enough for appropriate actions to be taken.

The health of a nation through a healthy environment and the adequate production of food and cash crops are two priorities which are not mutually exclusive. An effective regulatory system coupled with effective environmental health education would make both priorities achievable *pari passu*.

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