INSECTICIDE RESIDUES IN MILK AND MILK PRODUCTS[1]

III. INSECTICIDE RESIDUES IN DAIRY PRODUCTS AND ASSOCIATED PROBLEMS

E. H. MARTH AND B. E. ELLICKSON

Research and Development Division
National Dairy Products Corporation
Glenview, Illinois

INTRODUCTION

Insecticides gain entrance into milk primarily as a result of spraying dairy barns and cattle and ingestion of treated forages by dairy cattle. Information on these subjects has been summarized in two previous papers (*14*, *15*). Milks which contain insecticides may be mixed with insecticide-free milk during processing operations and hence the insecticide level of the final product may be changed. This paper will attempt to summarize information on: (*a*) the presence of insecticides in market milk and milk products; (*b*) problems created by the presence of insecticides in dairy products and (*c*) remedial measures which have been taken.

MARKET MILK

Surveys conducted by the Food and Drug Administration (*5*) in 1948, 1949 and 1951 indicated that trace amounts of DDT were detected in 25 per cent of the market milk samples tested.

In the fall of 1955, 800 samples of market milk, 50 from each of 16 Food and Drug districts, were analyzed for insecticide residues (*5*). Results of this survey showed the following: (*a*) 62 per cent of the samples contained insecticide residues; and (*b*) most residues were present in trace amounts.

One hundred and sixty samples with highest concentration were checked for the presence of specific insecticides. It was found that BHC was present in 60 per cent of the samples, DDT in 54 per cent, lindane in 26 per cent, DDD in 24 per cent, methoxychlor in three per cent and DDE (a breakdown product or metabolite of DDT) in 36 per cent.

Milk samples were checked for anticholinesterase activity to determine the presence of organic phosphate insecticide residues. These insecticides were not detected in the samples tested.

A later survey (*5*) was conducted during the winter of 1956-1957 by the Atlanta, New Orleans, Los Angeles and San Francisco Food and Drug districts. This survey showed little or no contamination of market milk with either BHC or DDT.

A limited survey for the presence of DDT in milk samples obtained from individual producers was conducted by Berruti (*2*) in January of 1958. Residues of DDT in the range of 0.06 to 10.0 p.p.m. were detected in 14 of 59 samples.

OTHER DAIRY PRODUCTS

Several authors have reported the presence of DDT in butter made from milk which contained residues of the insecticide. Smith, *et al.* (*24*) reported that 65 p.p.m. of DDT was present in butter made from milk which contained 2.3 to three p.p.m. of the insecticide. Higher levels of DDT were found by Schechter, *et al.* (*22*) who reported the presence of 456 to 534 p.p.m. in butter made from milk which contained three to 26 p.p.m. Telford (*25*) reported the presence of DDT in butter made from the milk of a goat which had been fed the insecticide.

Mann, *et al.* (*13*) found the following concentrations of DDT in dairy products made from milk which contained the insecticide: pasteurized cream, 70.2 p.p.m.; buttermilk, 1.9 p.p.m.; whey, 0.5 p.p.m.; butter, 100 p.p.m. and cheddar cheese 47.0 p.p.m.

Benzene hexachloride was found in butter made from milk produced by cows grazing on pastures that were previously sprayed with the insecticide (*16*).

Information about residues of other insecticides in dairy products or about insecticide residues in other dairy products appears to be lacking in the literature. Results of studies on the effect of manufacturing processes on insecticide residues are also scarce. Mann, *et al.* (*13*) reported that pasteurization had very little effect on the amount of DDT in milk.

PROBLEMS CREATED BY THE PRESENCE OF INSECTICIDES IN MILK AND MILK PRODUCTS

The literature fails to cite instances in which insecticide residues in milk have interfered with any of the processes employed in the manufacture of various dairy products. It must be noted, however, that the presence of a wettable DDT powder in milk has been found to interfere with the methylene blue test (*11*).

The presence of insecticides in milk, however, does create public health problems. No entirely "safe" insecticide has been developed. The misuse of any one of them in the production of milk (or the manufacture of milk products) may endanger the health of the consumer.

Chlorinated Hydrocarbon Insecticides

Most reported toxicological studies of this group of insecticides have involved DDT. This insecticide can gain entrance into the bodies of man and animals through absorption from the gastrointestinal tract, the lungs after inhalation and through the skin (*7, 17*). Neal and Von Oettingen (*17*) found no toxic symptoms in humans that were exposed to DDT as an aerosol spray or dust at a rate ten times greater than that which would be normally used. Various types of dermatitis may be associated with exposure of the skin to DDT and its solvents according to Hayes (*7*).

Cases of DDT poisoning are most generally associated with the oral ingestion of the insecticide. Animal experiments have indicated that solvents such as digestible animal or vegetable oils enhance the toxicity of DDT (*7*). When ingested in high concentrations, DDT can cause death in man (*3, 8*) and animals (*26*). If ingested in lower concentrations by man, DDT may produce nausea, apprehension, stiffness in the jaws and throat (*23*), slow pulse, giddiness and dilated pupils (*4*). MacCormack (*12*) reported that his own blood was lethal for lice six and 12 hours after he ingested 1.5 g. of DDT in butter. He only suffered from a few subcutaneous hemmorrhages and this only after exercise.

In animals large doses of orally ingested DDT caused tremors, convulsions, incoordination and death (*17*).

Adult cats who were injected with DDT showed neurologic disorders which involved stiffness, tremor, clonic movements and death (*19*). An autopsy of the cats showed damage to ganglion cells (vacuolar degeneration or pyknosis) and capillary dilation in the liver.

When rats were chronically poisoned with DDT, increases in liver lipids and the size of the liver were noted (*21*). The increase in liver lipids was accompanied by an increase in phospholipids and cholesterol.

Oral dosages of DDT required for the production of illness in man have been reported (*7*). A single dose of 10 mg. per kg. of body weight produced illness in some but not all subjects even when no vomiting occurred. Smaller dosages generally failed to produce illness although perspiration, headache and nausea were noted in an already sickly man who ingested 6 mg. per kg. of body weight. Convulsions have been noted when 16 or more mg. per kg. of body weight were ingested. Dosages as high as 285 mg. per kg. of body weight have been taken without fatal result. Vomiting, however, occurred and hence the dosage was reduced. The least daily dosage, which will lead to illness in man is unknown. Experimental work with animals shows, however, that some individuals might show mild illness if they received 2.5 to 5.0 mg. of DDT per kg. of body weight daily.

DDT was stored in the fatty tissues of all mammals and birds that have been studied (*7*). When a given quantity of DDT was ingested for a period of time, the amount stored in fat gradually increased to a point at which it remained stationary as long as the ingestion rate was constant. If the ingestion rate increased, the quantity stored also increased gradually until a new point was reached at which it again remained stationary as long as the new ingestion rate remained constant.

DDT introduced into the bodies of humans was regularly broken down into DDA (the acetic acid derivative of DDT) and DDE (the dehydrochlorinated derivative) (*7*). The DDA was subsequently excreted in the urine (*18, 23*) while the DDE was stored in the fat. (*7*). Hayes (*7*) noted that a small group of people may become hypersensitive to DDT.

Little information appears in the literature on the toxicity to man of other chlorinated hydrocarbons. Furman (*6*) reported that no toxic signs were seen in cattle which were dipped in benzene hexachloride solutions up to 0.5 per cent concentrations. Ingestion of BHC also failed to produce symptoms of toxicity.

Princi (*20*) reported that there is no essential difference in physiological responses produced by chlorinated hydrocarbon insecticides, hence, information given about DDT is perhaps somewhat applicable to other insecticides of the same general type. Table 1 indicates the toxicity of chlorinated hydrocarbon insecticides to both man and rats. It can be seen that toxaphene and endrin are most toxic and that methoxychlor and perthane are least toxic. The other insecticides of this type rank somewhere in between.

Organic Phosphate Insecticides

This group of insecticides may be absorbed by ingestion, inhalation or through the intact skin (*20*).

Table 1 indicates that parathion and TEPP are high and thiodan is moderately high in toxicity to human beings. Chlorthion, diazinon and malathion are moderate or moderately low in their toxicity to humans.

Early symptoms of organic phosphate poisoning may be any combination of the following: headache, dizziness, blurring of vision, nausea, vomiting, diarrhea, and breathing difficulty (*20*). Later symptoms include profuse sweating, salivation, pulmonary edema with cyanosis, meiosis and convulsions.

Since these insecticides generally are not secreted by cows in their milk even if they are ingested (*14*), it is doubtful whether milk or milk products would be responsible for human intoxications. Gross misuse of the organic phosphate insecticides during the pro-

TABLE 1—TOXICITY RATINGS OF COMMON INSECTICIDES[a]

Insecticide	LD/50-Oral[b]	Toxicity to humans
Chlorinated hydrocarbon		
Aldrin	0.107	Moderately high
Benzene hexachloride	0.960	Moderate
Chlordane	0.732	Moderate
DDT	0.400	Moderate
Dieldrin	0.130	Moderately high
Endrin	0.040	High
Heptachlor	0.144	Moderately high
Lindane	0.200	Moderate
Methoxychlor	9.600	Low
Perthane	9.600	Low
TDE	5.450	Moderate
Toxaphene	0.110	High
Organic Phosphate		
Chlorthion	2.410	Moderately Low
Diazinon	0.200	Moderate
Malathion	1.600	Moderately Low
Parathion	0.005	Very high
TEPP	0.002	Very high
Thiodan	0.144	Moderately high

[a] Information in this table based on data by Lehker (10).

[b] Figures given are the number of ounces of chemical orally administered per 100 pounds of body weight needed to kill 50 per cent of the test rats.

duction of milk or manufacture of milk products could, however, result in the presence of toxic levels in these products.

PRESENT STATUS OF REMEDIAL MEASURES

The Food and Drug Administration has attempted to eliminate certain insecticides from milk by setting tolerance levels for these chemicals at zero p.p.m. Affected by the zero tolerance level are: DDT (*2*) methoxychlor (*1, 9*) and malathion (*1*).

The U.S.D.A. has also attempted to help the situation through its recommendations on insecticide usage (*9*). The chlorinated hydrocarbon insecticides have virtually been eliminated from the list of products suggested for use in the control of insects on dairy cattle. Furthermore, the farmer is cautioned not to feed dairy cattle with plants which have been treated with aldrin, dieldrin, DDT, chlordane or toxaphene.

SUMMARY

Surveys of market milk supplies have shown that 25 to 62 per cent of the samples contained traces or larger amounts of chlorinated hydrocarbon insecticides. Benzene hexachloride and DDT were found most frequently. Organic phosphate insecticides were not found in samples tested.

Highest concentrations of chlorinated hydrocarbon insecticides were found in high-fat dairy products such at butter, cream and cheddar cheese.

DDT and other chlorinated hydrocarbon insecticides were found toxic when ingested in high concentrations and some may bring about chronic intoxication if ingested at low levels over long periods of time. DDT was stored in the fatty tissues of man, other mammals and birds after ingestion. Some people were hypersensitive to DDT.

Organic phosphate insecticides were found to vary from high to moderately low in their toxicity to humans. Milk generally did not contain these insecticides and hence only gross misuse would result in the presence of toxic levels in dairy products.

The Food and Drug Administration has set tolerance levels for DDT, methoxychlor and malathion in milk at zero p.p.m.

REFERENCES

1. Anonymous. Briefs. Food Eng. **30** (4): 27. 1958.
2. Berruti, R. Personal Communication. 1958.
3. Biden-Steele, K. and Stuckey, R. E. Poisoning by DDT Emulsion. Lancet **250**: 235-236. 1946.
4. Chit Thoung, U. Poisonous Effects of DDT on Humans. Indiana Med. Gaz. **81**:432. 1946.
5. Clifford, P. A. Pesticide Residues in Fluid Market Milk. Public Health Reports. **72**:729-734. 1957.
6. Furman, G. P., Toxicity of Benzene Hexachloride to Mammals. J. Econ. Entomol. **40**:518-521. 1947.
7. Hayes, W. J., Jr. Present Status of Our Knowledge of DDT Intoxication Am. J. Pub. Health **45**:478-485. 1955.
8. Hill, K. R. and Robinson, G. A Fatal Case of DDT Poisoning in a Child. Nature **156**:780-781. 1945.
9. Insecticide Recommendations of the Entomology Research Division for the Control of Insects Attacking Crops and Livestock—1958 Season. U.S.D.A. Handbook No. 120. 1958.
10. Lehker, G E. Dictionary of Insecticides and Their Use. Mod. San. Bldg. Maint. **10** (3): 13-14, 45-50. 1958.
11. Lillian, S. J. and Weiser, H. H. The Influence of DDT Wettable Powder on the Methylene Blue Test in Milk. J. Milk Food Tech. **16**:4-5, 8. 1953.
12. MacCormack, J. D. Infestation and DDT. Irish J. Med. Sci. **6**:571-592, 627-634. 1945.
13. Mann, H. D., Carter, R. H. and Ely, R. E. The DDT Content of Milk Products. J. Milk Food Tech. **13**: 340-341. 1950.
14. Marth, E. H. and Ellickson, B. E. Insecticide Residues in Milk and Milk Products. II. Insecticide Residues in Milk from Dairy Cattle Fed Treated Crops. J. Milk Food Tech. **22**:145-149. 1959.
15. Marth, E. H. and Ellickson, B. E. Insecticide Residues in Milk and Milk Products. I. Insecticide Residues in Milk from Treatment of Dairy Cows and Barns. J. Milk Food Tech. **22**: 112-116. 1959.
16. McDowall, F. H., Patchell, M. R., Hurst, F. and Kelsay, J. M. Effect of Treatment of Dairy Pastures with BHC and DDT on Flavor and Composition of Milk, Cream and Butter. New Zealand. J. Sci. Technol. (A) **37**:146-155. 1955.
17. Neal, P. A. and von Oettingen, W. F. The Toxicity and Potential Dangers of DDT to Humans and Warm Blooded Animals Med. Ann. Dist. Columbia **15**: 15-19. 1946.
18. Neal, P. A., Sweeney, T. R., Spicer, S. S. and von Oettingen, W. F. The Excretion of DDT (2, 2-bis-(p-chlorophenyl)-1, 1, 1-trichloroethane) in Man, Together with Clinical Observations. Public Health Repts. **61**:403-409. 1946.

19. Pluvinage, R. J. and Heath, J. W. Neural Effects of DDT Poisoning in Cats. Proc. Soc. Exptl. Biol. Med. 63:212-214. 1946.

20. Princi, F. Chlorinated Hydrocarbon and Phosphate Insecticide Intoxication. Pest Control 23:9-10. 1955.

21. Sarett, H. P. and Jandorf, B. J. Effects of Chronic DDT Intoxication in Rats on Lipids and Other Constituents of the Liver. J. Pharmacol. Exptl. Therap. 91:340-344. 1947.

22. Schechter, M. S., Pogorelskin, M. A. and Haller, H. L. Colorimetric Determination of DDT in Milk and Fatty Materials. Anal. Chem. 19:51-53. 1947.

23. Smith, M. I. Accidental Ingestion of DDT with a Note on Its Metabolism in Man. J. Am. Med. Assoc. 131:519-520. 1946.

24. Smith, R. F., Hoskins, W. M. and Fullmer, O. H. Secretion of DDT in Milk of Dairy Cows Fed Low-Residue Alfalfa Hay. J. Econ. Entomol. 41:761-764. 1948.

25. Telford, H. S. DDT Toxicity. Soap Sanit. Chemicals 21: 161-163, 167-168. 1945.

26. Virgili, R. and Giovanni, M. Quadri Anatomo-Patalogici Nell' Intossicazione Aucta Sperimentale da DDT Con Particolare Riferimento al Sistema Nervoso. Riv. Malariol. 28:107-124. 1949.

Special Service Article

TUBERCULOSIS AND BRUCELLOSIS AS MILK BORNE DISEASES

Editor's Note: Presented herewith is a Special Service Article on Tuberculosis and Brucellosis. Sometimes complacency may exist with regard to these two diseases, transmissible to man. While real progress has been and is being made, this Article indicates the need for constant vigilance. Also presented is a review of the situation as it now stands.

It is a tragic paradox that milk as one of the most important foods in the diet of the American people, is also an important vehicle for the transmission of disease of both human and animal origin. Bovine tuberculosis and brucellosis are probably the two most commonly know animal diseases which are transmitted to humans through the consumption of milk. Both can be completely eliminated in humans only be total eradication of the disease in animals.

TUBERCULOSIS

Tuberculosis is pathogenic to many animals, including mammals, birds, fish and reptiles, yet the only animals from which the disease is transmitted to humans are cattle and goats. (*1*) During the past 40 years, tremendous progress has been made in this country in reduction of tuberculosis in cattle through Federal and State test and slaughter programs. The nationwide incidence has been reduced from a high in 1918 of nearly 5 per cent of the animals and 25 to 50 per cent of the herds tested, to a low in 1952 of 0.11 per cent of the cattle tested. In 1940, the entire nation attained modified accredited status (infection rate of less than 0.5 per cent.) (*2*)

Unfortunately, after reaching modified accredited status, it became more difficult to obtain support for tuberculosis eradication programs, and, of course, during World War II, our fiscal and manpower attention and resources were diverted to the war effort. As a result of cutbacks in tuberculosis testing programs, the incidence of reactors in some States has increased since the war and this trend is expected to continue in certain areas until more intensified testing programs are instituted. In one of the States, the infection rate in cattle tested, rose from a low of 0.18 per cent during the war years to a high of 0.87 per cent in 1956. The average rate of infection in cattle tested in one county in this State rose as high as 5.14 per cent. To correct this condition the State, in cooperation with the U. S. Department of Agriculture, is now testing all cattle in each county as it comes due for accreditation. The State officials are also obtaining information on pretest movement of animals in and out of infected herds as a means of locating and eliminating other possible foci of infection, and tracing back to the herd of origin untested cattle that are slaughtered and show evidence of tuberculosis. Other states are similarly adopting more stringent programs in an effort to reduce infection where the infection rates have indicated that a problem was developing and to eliminate the disease entirely where the infection rate is low. The problem of eliminating the residual foci of infection will be particularly difficult, because of the lack of a rapid and practical method of screening herds for infection, short of periodic testing of all animals in all herds.

MILK BORNE BOVINE TUBERCULOSIS

At this point we might ask the question, is milkborne bovine tuberculosis in this country a serious public health problem? Only isolated cases of this disease

Presented at the Course on Epidemiology of Milkborne Disease, held at the Communicable Disease Center, Atlanta, Georgia, February, 1958.