

Pesticide Levels in Deer

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Introduction

During the summer of 1964, 525,000 acres of the Salmon National Forest in Idaho were treated with DDT for the control of spruce budworm, *Choristoneura fumiferana* (1). DDT at a concentration of one pound per acre was sprayed by fixed wing aircraft over 484,000 acres constituting the major portion of the infested area. The periphery of the region, encompassing an additional 41,000 acres, was sprayed by helicopter at a DDT concentration of one-half pound per acre, as a means of preventing undue contamination of adjacent localities. The DDT was dissolved in fuel oil carrier, which was applied at a rate of one gallon per acre.

As a part of the program, DDT residue levels were determined in representative samples of biota from the spray area and from control areas, in order to monitor the impact of spraying on the forest ecosystem. Included in the 1964 sampling were adipose tissue specimens from mule deer, *Odocoileus hemionus*, inhabiting both the spray region and a control area which had not been sprayed. Five years later, during the fall hunting season of 1969, mule deer adipose tissue samples were again obtained from the same areas. This report deals with the changes in adipose tissue body burden noted in these browsing animals during the five year recovery period.

Materials and Methods

Adipose tissue samples from adult male, adult female, and fawn mule deer were obtained from the spray and control areas by personnel of the Idaho Fish and Game Department. Collections were made throughout the month of October in 1964, which was three months after the spraying program had been completed. The 1964 samples, consisting of adipose specimens from sixteen exposed and twenty-five control animals, were frozen in glass vials and sent to the Agricultural Service Research Laboratory, U. S. Department of Agriculture, Yakima, Washington, for pesticide analysis.

Pesticides were extracted from the homogenized tissues with chloroform, which was then filtered through anhydrous sodium sulfate and cleared with several washings of concentrated sulfuric acid. The chloroform fraction was then filtered through anhydrous sodium sulfate, evaporated to dryness and taken up in the n-hexane.

Analysis was by Sr-90 electron capture gas chromatography (Research Specialities, Model B). Additional details of the extraction and analytical procedures are reported elsewhere (2). Operating parameters for gas chromatographic analysis were as follows:

Column:	5% DC-200, 10% QF-1 on Chromosorb W
Column Temperature:	210° C
Detector Temperature:	230° C

In October, 1969, deer fat samples were taken from twenty-seven deer inhabiting the spray area and twenty-seven control animals from outside the spray region. Attempts were made to select individuals that fell within the sex and age categories that had been tested previously. Samples were placed in tin containers, frozen and taken to the Idaho Community Study on Pesticides Laboratory, Boise, Idaho for pesticide residue determination.

These samples were extracted with petroleum ether and partitioned against acetonitrile, using a modified procedure of the de Faubert Maunder, *et al.* (3). The petroleum ether extract was then subjected to fractionation on a florosil column, details of which have been previously reported by Mills, *et al.* (4,5). Analysis was by tritium foil electron capture gas chromatography, using a Micro Tek 220 instrument equipped with two differing columns for confirmatory analysis. The following parameters were observed:

Columns:	4% SE-30, 6% QF-1 on Chromosorb W, DCMS, 80-100 mesh.
	1.5% OV-17, 1.95% QF-1 on Chromosorb W, DCMS, 100-120 mesh.
Temperatures:	Columns 200° C
	Injection Chamber 220° C
	Detector 205° C
Carrier Gas Flow: (nitrogen)	SE-30, QF-1 90 ml/min. OV-17, QF-1 70 ml/min.

Results

Results of the deer fat analyses are shown in Table I. In the control animals sampled in 1964, total adipose tissue DDT

TABLE I

Means, Ranges and Standard Deviations of Pesticides in Mule Deer Adipose Tissue in 1964 and 1969

Pesticides (ppm)	1964						1969					
	N = 25			N = 16			N = 27			N = 27		
	Mean	Range	S.D.	Mean	Range	S.D.	Mean	Range	S.D.	Mean	Range	S.D.
p,p'DDE	.03	<.01-.10	.027	.52	<.01-2.61	.743	.02	<.01-.10	.019	.03	<.01-.09	.024
p,p'DDT	.05	<.01-.25	.059	17.22	.46-118.18	28.447	.03	.01-.08	.017	.11	.01-.33	.089
p,p'DDD and o,p'DDT	<.01	<.01-.02	--	1.62	.05-7.86	1.989	.01	0-.06	.015	.03	0-.14	.040
Total Derived DDT	.08	<.01-.37	.068	19.36	.60-128.65	30.902	.05	.01-.24	.035	.18	.01-.56	.139

was at a mean level of less than 0.1 parts per million (ppm). Exposed deer obtained three months after the 1964 spraying episode had average pesticide adipose residuals that were much greater. Total derived DDT in these animals showed a mean level of 19.36 parts per million, which is nearly 250 times that noted in the controls. In terms of individual pesticide residues, the mean level of p,p'DDE in exposed deer was 17 times higher than that of the controls, while the mean for p,p'DDT was nearly 350 times higher (Table I).

Figure 1 shows pesticide levels in the 1964 animals as a function of sex. Control animals had very little pesticide content and did not appear to vary appreciably with sex. In the exposed group, however, males had considerably more of each DDT derived residue than did females. Since only three exposed and two control fawns were obtained for that year, meaningful comparison in juvenile animals is difficult. However, the exposed fawns sampled did have substantially greater levels of both p,p'DDT and total DDT than did the exposed females.

Adipose pesticides in deer sampled from the unsprayed area five years later, as shown in Table I, did not differ greatly from the comparable group analyzed in 1964, other than having slightly higher mean levels. However, animals obtained from the spray region in 1969 had adipose residuals that were much lower than the 1964 exposed deer samples. The mean total derived DDT concentration in these animals was only 0.18 ppm, which is approximately one one-hundredth that found in the deer samples five years previously. In 1969, mean levels of p,p'DDE in deer from the spray area were about one-seventeenth those found in 1964, while mean p,p'DDT concentrations in 1969 were lower by a factor of more than 150. The exposed animals of 1969 had only about twice as much p,p'DDE and four times as much p,p'DDT as did those from the control area.

As shown in Figure 2, spray area males in 1969 again had more of the DDT derived materials than did females from the same region. Except for p,p'DDE, exposed fawns usually had higher pesticide residues than females, but these figures are again based on only three exposed and three control individuals.

FIGURE 1

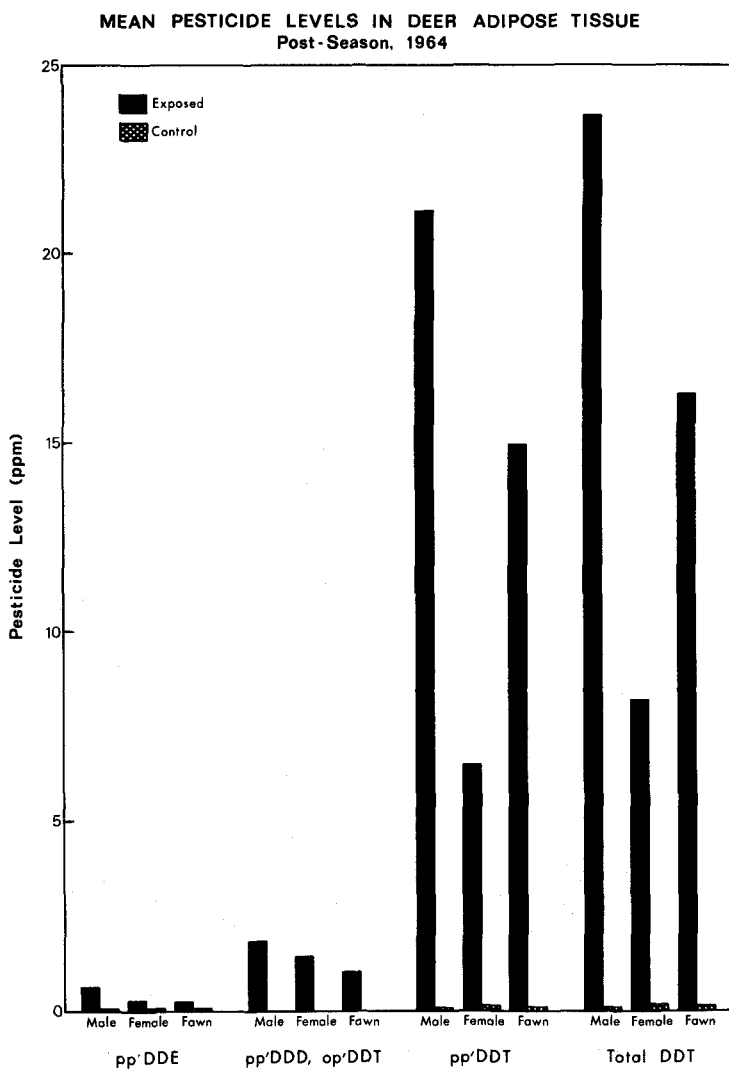
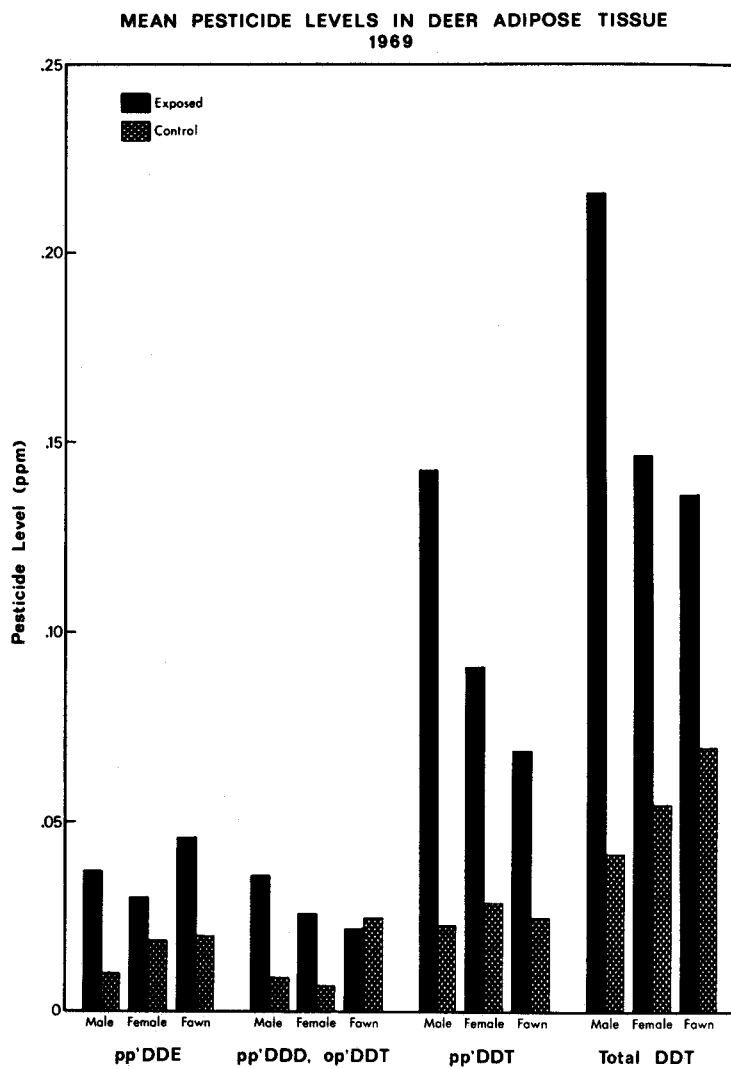


FIGURE 2



Discussion

According to the Idaho Department of Fish and Game (6), the great majority of mule deer in Idaho are less than three years old, and only about three per cent of Idaho's deer population can be expected to reach the age of five. It is thus quite unlikely that any of the deer sampled in 1969 were living at the time that the habitat was sprayed. Consequently, body burden in the 1969 animals tends to represent chronic exposure, is primarily due to diet, and probably reflects the pesticide content in local vegetation rather than the ability of the deer to decrease a once much greater body burden resulting from acute exposure. The 1964 animals, however, were obviously exposed much more acutely to the pesticides and consequently, residues were acquired by other routes in addition to diet. These would include an initial period of dermal and respiratory absorption of airborne DDT during and shortly after the spraying episode, as well as increased dietary exposure. This is in keeping with the much greater amounts of DDT, rather than DDE, that were noted in the 1964 exposed animals, since DDT as the p,p' isomer was absorbed well before any substantial degree of environmental degradation could occur.

In humans, p,p' DDT is broken down rather easily, and is ultimately converted to the aldehyde form, DDA, which is excreted in the urine and feces (7). Moreover, tissue-stored DDT does not appear to form appreciable amounts of DDE; storage of the latter isomer resulting primarily from biological sequestration of preformed DDE from the environment (8). Also, at least in humans, stored DDE appears to be quite stable and is probably not broken down by the same metabolic pathway as DDT (7,8). With these facts in mind, one would expect to find an abundance of DDE in the 1969 exposed animals. However, although DDE was found much more frequently in 1969 than in 1964, the exposed animals sampled in 1969 still contain considerably more DDT than DDE. This tendency for deer to not have appreciable adipose levels of DDE has also been reported by Pillmore and Finley (9) who examined deer and elk herds in Colorado and New Mexico that had been exposed to similar spruce budworm eradication programs. Their findings indicated that while elk adipose tissue was found to contain both DDE and DDT, deer adipose tissue from the same region contained primarily DDT. This could be an indication that much of the DDT in the various spray areas had not yet been broken down to DDE, but this would seem unlikely for the extended time period encompassing the present study. Another possibility is that deer, perhaps because of their differing intestinal flora and ruminant mode of digestion, do not metabolize DDT in the traditional manner. Whatever the reason, DDT was still the persistent residue found in the 1969 animals, and this would seem unlikely in terms of human biochemistry.

The much greater body burden found in male deer, particularly in the 1964 samples (Figure 1), is difficult to explain. Such a relationship parallels our findings in humans (10), in which male serum pesticide levels were consistently higher than those of females, and probably reflects hormonal differences. Despite the few fawns sampled, the relatively high levels noted nonetheless invite speculation. It is a well established fact that DDT and its derivatives can be present in milk at rather high levels (11). The possibility of maternal milk being a contributing factor to the high adipose tissue pesticide levels in the fawns sampled should not be discounted.

Although mule deer usually undergo a seasonal migration, such movements are primarily altitudinal in nature and do not normally involve long distances (12). Such migration in these animals is greatly influenced by local terrain and food availability. Although little is known regarding the particular mule deer population inhabiting the spray area, local topography and the limited available data do suggest that deer herds within the region are relatively static. Consequently, the chances are minimal that some of the deer taken from the spray area in 1969 were merely transient residents.

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References

1. CASEBEER, R. L., Monitoring the 1964 Spruce Budworm Aerial Spray Project. U. S. Forest Service Publication. U. S. D. A., Ogden, Utah (1965).
2. U. S. D. A. Agricultural Research Service mimeographed report PCY-65-16 (on file with Entomology Research Division, Agricultural Research Service, Yakima, Washington) (1965).
3. de FAUBERT MAUNDER, M. J., EGAN, H., GODLY, E. W., HAMMOND, E. W., ROBURN, J. and THOMSON, J., Analyst 89: 168-174 (1964).
4. MILLS, P. A., J. Assoc. Offic. Agr. Chemists 44:2, 171-177 (1961).

5. MILLS, P. A., ONLEY, J. H. and GAITHER, R. A., J. Assoc. Offic. Agr. Chemists 46:2, 186-191 (1963).
6. THIESSEN, J. L., Regional Game Manager, Idaho Fish and Game Department. (Personal Communication) (1972).
7. ROAN, C. C., MORGAN, D. P. and PASCHAL, E. H., Arch. Environ. Health 22: 309-315 (1971).
8. MORGAN, D. P. and ROAN, C. C., Arch. Environ. Health 22: 301-308 (1971).
9. PILLMORE, R. E. and FINLEY, R. B., JR., Proceedings of the 28th North American Wildlife Conference. 409-422 (1963).
10. WATSON, M., BENSON, W. W., and GABICA, J., Pesticides Monitoring Journal 4:2, 47-50 (1970).
11. QUINBY, G. E., ARMSTRONG, J. F. and DURHAM, W. F., Nature 207: 726-728 (1965).
12. BOURLIERE, F., The Natural History of Mammals, p. 205 (1960), A. A. Knopf, Inc., New York.