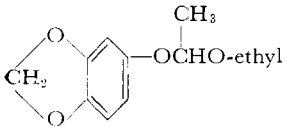
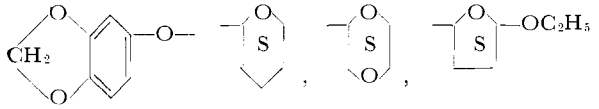
Table VIII. Synergistic Activity[a] of Acetals

	With Pyrethrins	With Allethrin
	9+	3.5+
-chloroethyl	7	3
-butyl	4	2
-isobutyl	4	2
-2-ethylhexyl	3	3
-2-methoxyethyl	9+	3.5+
-2-butoxyethyl	9+	3.5+
-2-(2-ethoxyethoxy)ethyl	9+	3.5+
-2-(2-butoxyethoxy)ethyl	9+	3.5+
	9+	3.5+

[a] Number of times the activity of pyrethrins or allethrin was increased.

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ACTION OF RUMEN FLUID ON PESTICIDES

In Vitro Destruction of Some Organophosphate Pesticides by Bovine Rumen Fluid

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Parathion and three other compounds containing *p*-nitrophenol and the thiono isomer of Systox were rapidly destroyed—as measured by an anticholinesterase method of analysis and by paper chromatography—when 100 p.p.m. each of parathion and nine other phosphate pesticides were added to aliquots of bovine rumen fluid. The *p*-nitrophenol moiety of the parathion molecule was reduced to *p*-aminophenol. Paper chromatography of extracts of incubation mixtures revealed that thiol isomers are not destroyed like thiono isomers. Evidence indicates that metabolism of parathion in the rumen can account for its apparent lack of toxicity to cattle as reported in the literature.

USE OF PESTICIDES in control of insects on forage and other dairy feed crops creates the possibility of incurrence of residues of these pesticides by milk. Studies in feeding of chlorinated pesticides such as DDT and lindane, (γ - 1,2,3,4,5,6 - hexachlorocyclohexane) have shown that residues of this type of pesticide are stored to varying degrees in animal fat and excreted in milk (*4, 14–16*).

Considerably less has been reported on the feeding of organophosphate pesticides. Development of the facts is important, not only because of the current trend toward increased use of this type of pesticide on feed crops, but also because of the potential value of organo-

phosphates as systemic agents to control insects and parasites in and on animals themselves (*23*, *29*).

Dahm and coworkers (*11*) reported in 1950 that no parathion (*O,O*-diethyl *O*-*p*-nitrophenyl phosphorothioate) was detected in cows' milk when it was fed at the rate of 1 and 5 p.p.m. The same investigators (*28*) reported in 1952 that they fed cattle as high as 32 mg. of parathion per kg. per day (or about 2000 p.p.m. in the diet) for 1 week and found no evidence of blood cholinesterase depression, or of the presence of parathion or its potential degradation products, *p*-nitrophenol or *p*-aminophenol, free in the blood, milk, or urine. *p*-Aminophenol was obtained from urine after acid hydrolysis, and ascribed to be from the hydrolysis of *p*-aminophenylglucuronide, but they were unable to identify any of the glucuronide itself. They commented that the amino derivative of parathion, if present, would yield *p*-aminophenol by their acid treatment. An increase in diazotizable material in the urine was found.

Wilber and Morrison (*30*) studied the effect of parathion on goats, and the effects of feeding kids milk from parathion-poisoned goats. Their work indicates that the goat may be more sensitive to parathion than the cows studied by Dahm and coworkers. They showed that in parathion poisoning of goats an anticholinesterase substance apparently crosses the placenta-blood barrier and exerts its effect on the fetus, and that parathion or a like substance appears in the milk of poisoned goats and depresses the erythrocyte cholinesterase activity of kids fed with that milk.

Cook and Sykes (*10*) conducted an experiment in which two cows each were fed 0, 1, 10, or 50 p.p.m. parathion in the whole diet. No evidence of a cholinesterase inhibiting material was found in milk over a 30-day feeding trial, using a method of analysis sensitive to about 0.02 p.p.m. The method of analysis was an unpublished modification of the method of Fallscheer and Cook (*17*) in which parathion was converted to paraoxon (*O,O*-diethyl *O*-*p*-nitrophenyl phosphate) by the use of bromine water. The paraoxon was then measured by the colorimetric anticholinesterase method of Cook (*5*) using pooled human plasma as the source of cholinesterase. The modification of the method (*17*) consists of a relatively large increase in the amount of bromine required for the conversion of parathion to paraoxon necessitated by the presence of milk fat. At the end of the parathion feeding experiment (*10*), the blood cholinesterase level of each of the eight cows was determined and was found to be normal.

The apparent lack of effect of feeding such high levels of parathion to cattle is in sharp contrast to the effect of feeding parathion to other species. Frawley and Fuyat (*18*) have reported a 25% depression of plasma cholinesterase in dogs fed parathion at the rate of 1 p.p.m. This evidence suggests the possibility that parathion may not be available to the blood stream of the cow and therefore not available for transfer to the milk.

In contrast to these observations on parathion, Dahm and Jacobson (*12*) have shown recently that the cow is many times more sensitive to Systox [*O,O*-diethyl *O* (and *S*)-ethyl-2-mercaptoethyl phosphorothioate] than to parathion. One feeding of 2.5 mg. of technical Systox per kg. of body weight caused serious reactions, including evidence of the presence of a cholinesterase inhibitor transferred to milk. Technical Systox is a mixture of 65% thiono and 35% thiol isomers whereas technical parathion is supposed to be a thiono compound exclusively.

The digestive system of ruminating animals, such as the cow and goat, is very different from other animals. The tremendously active microorganism population of the rumen substantially predigests most of the feed ingredients before they reach the true stomach. Enzyme systems in rumen fluid might be responsible for the apparent destruction of parathion before it is absorbed in the blood stream. The work reported here was undertaken to investigate this possibility. The author first supposed that the mode of action of rumen fluid on parathion would be much the same as the usual enzymatic action which ultimately destroys these insecticides—that is, first an oxidation of parathion to paraoxon (*3*, *19*) and then hydrolysis of the oxygen analog.

There have been studies on the hydrolysis of organophosphate compounds by enzymes called dialkylfluorophosphatase and tabunase (*1*, *24*, *27*) as well as by cholinesterases. These enzymes are not cholinesterases and are found in many biological sources, including microorganisms (*26*). The author found no mention in the literature that any of the phosphorothioates have been hydrolyzed by the enzyme systems (*1*, *27*). Even Isosystox (*O,O*-diethyl-*S*-ethyl-2-mercaptoethyl phosphorothiolate), easily hydrolyzed by cholinesterase, was not hydrolyzed by dialkylfluorophosphatase.

For this study, the compounds were chosen so that some of the various classes of thiophosphates and phosphates were included. These were subjected to rumen fluid action. Parathion, Diazinon [*O,O*-diethyl *O*-(2-isopropyl-4-methyl-6-pyrimidinyl) phosphorothioate], EPN (*O*-ethyl *O*-*p*-nitrophenyl phenylphosphonothioate), and the thiono isomer of Systox represented the thiono compounds. The thiol isomer of Systox represented the thiol compounds. (These two isomers of Systox were added as technical Systox which is a mixture of the two isomers as indicated previously.) Malathion {*S*-[1,2-bis(ethoxycarbonyl)ethyl]*O,O*-dimethyl phosphorodithioate} and Hercules AC-528 [2,3-*p*-dioxanedithiol *S,S*-bis(*O,O*-diethyl phosphorodithioate)] represented the dithio compounds. Finally, a last group which represented the phosphates, paraoxon, Dipterex [*O,O*-dimethyl 2,2,2-trichloro-1-hydroxyethyl phosphonate (Bayer L 13/59)], and Phosdrin (*O,O*-dimethyl 1-carbomethoxy-1-propen-2-yl phosphate), was included. All of the above phosphorothioates, except the thiol isomer of Systox, are very poor in vitro cholinesterase inhibitors until they are oxidized, whereupon they become very potent inhibitors (*8*, *17*). The phosphates are all very potent in vitro anticholinesterase agents.

Experimental

Analysis. Fresh bovine rumen fluid, obtained by stomach tube or from an animal with a rumen fistula, was brought to the laboratory and strained through two layers of cheese cloth. The time interval between collection of a sample and the beginning of an experiment was usually about 1 hour. No attempt was made to control temperature during this time; consequently, the fluid had cooled to room temperature before the experiment was started. Usually 20-ml. aliquots of this filtered fluid were transferred to a series of 50-ml. glass-stoppered bottles, which were then placed in a 37° C. water bath. When they had reached bath temperature, 1 ml. of an alcoholic solution containing 2000 γ per ml. of the various phosphorus compounds was added, giving 100 p.p.m. in the incubation fluid. The material was well mixed, sampled, and returned to the bath. Other samples were removed at increasing time intervals.

For the earlier part of the quantitative anticholinesterase work, for parathion determinations by the Averell-Norris procedure and for all the paper chromatographic work, sampling consisted of removing an aliquot and shaking it with an equal volume of chloroform, after which the mixture was centrifuged and the chloroform layer filtered through paper. Aliquots were diluted appropriately for analysis or the chloroform was spotted directly on paper for chromatography. In the latter part of the quantitative anticholinesterase work, sampling was done by removing a 1-ml. aliquot of the incubated mixture and diluting appropriately with water. Both procedures gave comparable results. Dilution of the incubation mixture stopped the enzymatic action.

The method of analysis used was the

colorimetric method described previously (*5*), modified by adding bromine water before the addition of the enzyme. This step converts all of the phosphorothioate and phosphorodithioate compounds dealt with in this paper (except the thiono isomer of Systox) to potent anticholinesterase agents (*17*).

Parathion was also determined by the Averell-Norris method (*2*). Diazotizable material was determined by the same procedure except for the omission of the reduction step.

Chromatography. Ascending paper chromatography was run using mineral oil as the immobile solvent, water as the mobile solvent, Whatman No. 1 paper, the anticholinesterase spot test (Figure 1) described by Cook (*7*), and the *N*-bromosuccinimide-fluorescein spot test (Figure 2) (*6*).

Results and Discussion

Table I shows the effect of the in vitro action of rumen fluid from a cow fed a high-alfalfa normal-maintenance diet, and from a cow on a high-grain fattening diet on a number of organophosphate pesticides as measured by an anticholinesterase method. Parathion, paraoxon, Chlorthion, and EPN disappear rapidly—except that EPN did not disappear from the fluid from the cow on the high-grain ration. All of these that disappeared rapidly are *p*-nitrophenol derivatives whereas the others are derivatives of other compounds. Malathion and possibly Phosdrin were affected in 24 hours, but Dipterex, Diazinon, Hercules AC-528, and Systox appear to be unaffected. Boiled rumen fluid had essentially no effect on the compounds tested, suggesting that the action may be enzymatic in nature.

Figure 1 shows tracings of paper chromatograms of technical Systox and parathion. The irregular areas represent the presence of cholinesterase inhibitors. The *a* chromatograms are from the technical products, the *b* chromatograms are from 0-time chloroform extracts of rumen fluid plus the pesticides, and the *c* chromatograms are from chloroform extracts of the same preparations after they had incubated at 37° C. for 1 hour. In the case of parathion, the spot at the origin is due to parathion (*8*). The highest spot is due to paraoxon, and the intermediate spot is believed to be due to a thiol isomer of parathion. The paraoxon present in the technical parathion is an impurity and is present at a low level of about 0.5% (*9*). The difference between the *a* and *b* chromatograms in the paraoxon position is presumably due to the loss of the small amount present through enzymatic destruction during the mixing, sampling, and chloroform extraction periods. It is shown that the thiono isomer disappears at the origin during the 1-hour incubation period and that the presumed thiol isomer persists during this period.

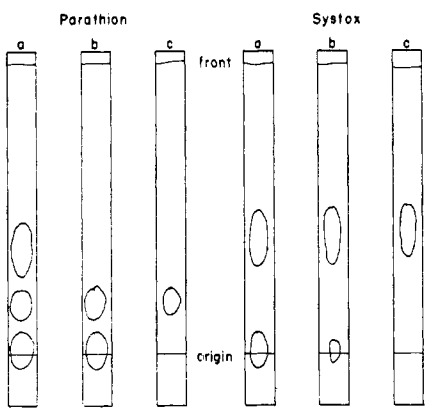


Figure 1. Tracings of chromatograms of parathion and Systox before and after subjection to rumen fluid activity

The spot at the origin on the Systox chromatogram is due to the thiono isomer of Systox and the spot at R_f 0.4 is due to the thiol isomer of Systox. Again the thiol isomer persists, whereas the other spot disappears after subjection to the rumen fluid activity. By comparison of these Systox results with those in Table I, it appears that the quantitative method measures only the thiol isomer and that it is entirely unaltered by the rumen fluid activity, whereas the thiono isomer does disappear with no resultant change in the quantitative results.

The quantitative action of the rumen fluid on parathion has been repeated a number of times, largely because it was included as a control in most of the experiments. The results, as shown in Table II, have been remarkably consistent.

Various reviewers on the subject of rumen digestion (*13, 20, 21*) agree that the microbiology of rumen action is extremely complex and sensitive. Apparently the myriad types of microorganisms (there are estimates of as

Table I. Effect of Rumen Fluid on Ten Phosphate Pesticides

	A	B				C		
	Micrograms/ determination	Fresh Fluid, % Inhibition at				Boiled Fluid, % Inhibition at		
		0 hr.	1 hr.	3 hr.	25 hr.	0 hr.	3 hr.	25 hr.
Parathion[a]	0.01	57	8	1	5	95	74	68
Parathion[b]	0.01	69	0	5				
Paraoxon[a]	0.01	65	1	1	0			
Paraoxon[b]	0.01	56	0	0				
Chlorthion[a]	0.50	88	4	1	5	92	91	84
Chlorthion[b]	0.50	80	3	10				
EPN[a]	0.25	88	27	4	4	95	96	100
EPN[b]	0.25	96	100	100	100			
Malathion[a]	0.50	41	43	26	9	53	49	47
Malathion[b]	0.50	51	51	49	43			
Phosdrin	0.25	79	81	97	59	82	80	74
Diazinon[a]	0.005	40	45	37	36			
Diazinon[b]	0.005	44	44	57	41			
Dipterex	1.00	100	100	96	100			
Hercules AC-528	0.50	88	83	54	83			
Systox	0.75	59	63	54	57			

[a] High-alfalfa ration.
[b] High-grain ration.

Table II. Effect of Rumen Samples on 0.01 γ Parathion per Determination

Rumen Source, Ration	% Inhibition at 0 hr.	% Inhibition at 1 hr.
High-alfalfa	57	7
	57	8
	69	11
	58	9
	72	8
High-grain	69	0
	61	5

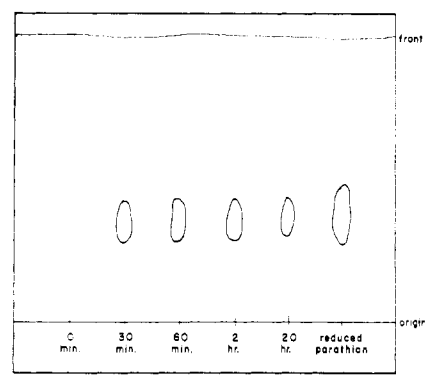
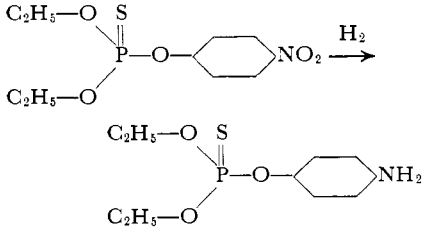


Figure 2. Tracing of chromatogram of reduced parathion and chloroform extracts of rumen fluid-parathion digests

many as 3 to 10 billion organisms per ml. of fluid) are very complexly interdependent symbiotically, both with regard to the organisms themselves and also with regard to the host animal and the medium created by the host. The diet of the animal influences the quantitative relationships of the various organisms. Because of these factors a number of the pesticides were run in fluid from different cows on very different diets. The animal used for most of the work was on an alfalfa-grain ration for normal maintenance and the second one was fed a high-grain fattening ration. The results, as shown in Table I, are substantially the same from both animals for parathion, paraoxon, Chlorthion, and Diazinon, but are different for EPN and malathion, the difference being that the fluid from the animal fed a high-grain ration did not destroy EPN and malathion like the fluid from the animal fed the high-alfalfa ration. However, further investigations should be made before definite conclusions are drawn.

In Table I, column A, the quantity of the pesticide required to produce the inhibition indicated in column B is shown. A comparison of these values shows that there is a wide variation in the anticholinesterase activity of these compounds and that the anticholinesterase activity bears no relationship to the amount destroyed by the rumen fluid. For example, Chlorthion is about 0.025 as potent as parathion and yet disappears at about the same rate, while Diazinon is a little less than twice as potent as parathion, as an anticholinesterase agent, and appears to be unaltered by the rumen fluid. This suggests that the action is not that of an esterase enzyme system such as cholinesterase. Lewis (*22*) has reported the presence of an enzyme system in sheep rumen fluid which reduces nitrates to hydroxylamine and ammonia. As the pesticides which are most readily attacked by rumen fluid are those which have a nitro group, an attempt was made to identify the presence of a derivative of parathion in which the nitro group was reduced to an amino group as shown below.



In the Averell-Norris procedure for the chemical determination of parathion, the first step accomplishes this reduction by boiling in the presence of hydrogen formed from zinc and hydrochloric acid. A quantity of the reduced compound was prepared by this procedure. It was chromatographed in the same manner as previously described for the other compounds. Figure 2 shows chromatograms formed by chromatographing the reduced compound and chloroform extracts of the rumen fluid-parathion digests sampled at 0, 30, 60, 120 minutes, and 20 hours. No spot appears in the R_f 0.3 position at 0 time, but does appear after digestive action has taken place as well as from the reduced compound. This evidence suggests that the rumen fluid forms the reduced compound and that it forms principally within the first 30 minutes.

Table III. Parathion by Averell-Norris Method with and without Reduction Step

Digestion Time, Min.	Recovery, %	
	With Zn	Without Zn
0	72	0
0	75	0
30	83	75
30	82	78
60	80	78
60	82	79

When these chloroform extracts were analyzed by the Averell-Norris procedure for parathion and again by the same procedure except for the omission of the reducing step—that is, omission of zinc in the usual Averell-Norris procedure—values as shown in Table III were obtained. These data can be interpreted to mean that the rumen fluid quantitatively converts the nitro group to an amino group in the first 30 minutes. The same value was obtained either with or without zinc at the end of 30 or 60 minutes, as was obtained at 0 time with zinc. Absence of color at 0 time without zinc indicates that no reduced parathion existed at that time and also no other diazotizable compounds were extracted from the digest. The colors produced with and without zinc have the same absorption peak at 555 mμ as does parathion, and the colors develop at the same rate.

Considerable attempts to prepare a stable preparation of rumen fluid with sufficient activity to characterize the enzyme action more easily have failed. Acetone and alcohol powders and a powder prepared by lyophylizing rumen fluid had no activity. Attempts to store the fluid at room, refrigerator, or freezing temperature were only partially successful, in that, under the best conditions, fairly good activity was retained to the second day but very little remained over 4 days. The activity decreased very rapidly unless the fluid was held under an atmosphere of carbon dioxide. Refrigerated fluid was as stable as frozen fluid. Utilizing a reducing agent such as sodium monosulfide did not seem to aid in retaining activity.

The activity does reside with the particulate matter. Very mild centrifugation separated a green particulate residue containing feed particles, bacteria, and protozoa. The residue contained all of the activity. None remained with the supernatant fluid even though the centrifugation was such that a great share of the bacteria and some of the smaller protozoa remained in the supernatant. The general population of protozoa does not appear to include the important agent. When fresh fluid was allowed to stand a short time, carbon dioxide evolved and buoyed the green particulate matter to the surface. This contained a large number of protozoa but the great majority of the protozoa remained on the bottom of the flask in a grayish-white layer. This type of fractionation was utilized to separate the fluid into three fractions—the green layer, the layer of protozoa, and the intermediate liquid. Again all the activity resided in the green layer. None was in the protozoan layer and none was in the intermediate liquid layer.

Conclusions

The in vitro experiments described explain, to some extent, why large amounts of parathion can be fed to cows (*10*, *28*), whereas they tolerate less Systox (*12*). The rumen fluid activity attacks some of the thiono compounds and does not attack any of the thiol compounds studied. However, each parathion sample that has been chromatographed by the author has had some "impurity," presumably the thiol isomer, as well as paraoxon. It is estimated that the thiol isomer present in the samples used for this in vitro work was about 4% (*9*). Metcalf and March (*25*) reported in 1953 that parathion from three different manufactures contained an impurity identified as the *S*-ethyl thiol isomer. It varied from 4 to 20% of the total. A variation as great as this may account, to some extent, for the apparent difference in toxicity of parathion to the goat as compared to the cow.

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SWEET POTATO CANNING

Effects of Processing Conditions on the Chemical Properties of Canned Sweet Potatoes

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Effects of processing conditions on the chemical properties of Unit I Porto Rico and Goldrush varieties of sweet potatoes were determined. The β-carotene content of the canned potatoes was relatively stable during processing with values of 20 to 25 mg. per No. 2 can of Unit I Porto Rico sweet potatoes and 40 to 45 mg. per No. 2 can of Goldrush. The dry weight of the cooked potato was about 80% sugar, averaging 120 to 150 grams per No. 2 can when packed in 35°-Brix sugar solution, with the Unit I Porto Rico variety containing more sugar than the Goldrush variety. Ascorbic acid content was reduced during processing.

SWEET POTATO CANNING is an established industry, and the pack in the United States has been reported in the "Biennial Census of Manufacturers" for more than 50 years. Selection of varieties suitable for canning, relationship of storage to canning quality, and canning processes have been determined by numerous investigators (*10–16*). Woodroof, DuPree, and Cecil (*17*) have recently issued a bulletin on the canning of sweet potatoes in which the selection of varieties, the conditions of processing, and the nutritional value of canned sweet potatoes were evaluated.

The effects of raw storage, conditions of processing, and variety on the chemical properties of dehydrated sweet potato products have been reported by the authors (*2–5*). These data provide an objective basis for evaluation of the potential nutritive value of the dehydrated products. Similar information on the effects of these variables on the chemical properties of canned sweet potatoes has not been reported in the literature.