UNITED STATES DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE

THE EFFECT OF HEAT AND MOISTURE ON THE FEEDING VALUE OF PILCHARD MEAL

RESEARCH REPORT 3

UNITED STATES DEPARTMENT OF THE INTERIOR
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By

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ABSTRACT

The fish meals produced today have in general proved to be excellent sources of protein for the balancing of farm-animal rations. Some potential users, however, have refrained from feeding these products because of reports implying that fish meals, when fed to farm animals and poultry, have caused symptoms of toxicity.

It was considered possible that the meals suspected of producing toxic symptoms may have been subjected to conditions after manufacture that had altered them in an unsatisfactory manner. Therefore, the effect of excessive heat and humidity to simulate extremely unsatisfactory storage conditions was studied with pilchard meal.

Pilchard meal was incorporated in otherwise purified diets containing as much as 25 percent of protein; of which more than 95 percent had been derived from pilchard meal. When these diets were fed to rats and chicks it was found that the excess heat and high humidity had not adversely altered the nutritive value of the meal. This indicates that fish meals can be used liberally in farm-animal rations as a source of minerals and superior protein, provided ordinary care is used to balance properly the ration for the other essential food elements.

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INTRODUCTION

In the United States, fish meals have been recognized as a potential source of protein for farm-animal feeding only in comparatively recent years. The Norwegian Government, however, recommended their use as early as 1892, and succeeded in promoting widespread utilization in continental Europe. Although the use of fish meals in animal feeding had been advised as early as 1835 (Atwater 1877)[1], it was not until 1914–18 that high prices and actual scarcity of other feeds caused the farmers of America to utilize this previously neglected material in appreciable quantity.

In general, the fish meals produced today have proved to be excellent sources of protein for the balancing of farm-animal rations. Some potential users, however, have refrained from feeding these products because published reports have implied that cod-liver oil and fish meals have caused symptoms of toxicity when fed to animals and fowls.

Gautier and Morgues (1888) reported the presence of relatively large quantities of volatile aliphatic amines in fish meals, with isoamylamine, which they reported to be an active poison, composing about one-third of the total bases present. The other products found were hexylamine, dehydrolutidine, asellin, morrhuin, and morrhuic acid. These compounds produced paralysis, convulsions, and lack of growth. Hawk (1908) confirmed these findings. Similarly, Norris and Church (1930) observed a toxic action that was ascribed to certain nitrogenous bases when 2 percent of cod-liver oil was included in the diet of chicks. They found that 18 percent of dry yeast in the diet alleviated the toxicosis induced by feeding isoamylamine dissolved in oil at a level of 0.14–0.56 mg. of the amine per day. Gutteridge (1935) reported

[1] Publications referred to parenthetically by date are listed in the literature cited, p. 9.

that the toxic action of sun-rendered cod-liver oil (0.31 percent nitrogen), when fed to poultry, was greatly lessened by deamination.

Holst and Holbrook (1933) reported a "scurvey-like" disease in chicks fed a diet containing 20 percent of fish meal. Dam and Schöenheyder (1934) described a deficiency disease in chicks with symptoms of leg paralysis, anemia, hemorrhage, and poor growth. Halpin, Holmes, Elvehjem, and Hart (1935) stated that too much fish meal in the diet produced crippled feet in chicks.

In many of these reports the symptoms described were similar—severe anemia with atypical blood cells, lesions on internal organs and muscles, both external and internal hemorrhages, paralysis, stunted growth, and death. Birds less than 8 days old showed similar symptons when fed diets that did not affect older birds. Cook and Scott (1935) stated that they could produce the disease symptoms in birds fed an otherwise satisfactory diet by including small quantities of nitrogenous bases, such as trimethylamine, diethylamine, and isoamylamine. These bases have been found in fish meals. Almquist (1936), however, was unable to confirm the experimental findings of Cook and Scott.

Many articles have been noted that report fish meals to be a satisfactory protein supplement in the rations of farm animals and fowls. It is possible, therefore, that the meals suspected of producing toxic symptoms may have been subjected to conditions after manufacture that had altered them in an unsatisfactory manner. It has been suggested by some of the previously mentioned investigators that certain amines resulting from protein decomposition may have been responsible for the apparent toxicosis.

Owing to the desirability of making full use of fish meal as a protein supplement in the rations of farm animals and fowls, an investigation was conducted by the Fish and Wildlife Service to determine the factors affecting the feeding value of commercial pilchard meal. This meal served as practically the only source of protein in diets fed to rats and chicks. Part of the fish meal was partially decomposed under conditions of excess moisture and heat in order to determine whether toxic compounds were formed and whether this spoilage was of the type, but in a more severe degree, of that occurring in meal stored in unsatisfactory warehouses or in ship holds during transport. The problem was to determine whether the unfavorable effects reported in the literature were due to the presence of one or more toxic compounds in the meal, or were symptoms of a deficiency of one or more vitamins or minerals in the diet.

FEEDING EXPERIMENTS

A search of the literature did not reveal any experimental fish-meal diets for chicks, which had produced reasonably satisfactory growth,

that did not derive a considerable part of their protein from sources other than fish meal. For this reason Pacific coast pilchard meal, an oily type, was selected, and a diet was formulated in which more than 95 percent of the protein was supplied by the meal.

Experimental diets.—Half of each meal sample was partially spoiled by first mixing it with water to a thick paste, and then heating it for 1 week in large shallow metal trays at about 50° C. The meal thus treated developed an extremely foul odor. It was dried at a temperature under 50° C., finely ground, and stored in covered metal cans.

TABLE 1.—*Approximate analyses of the fish-meal samples*

Sample	Percent by weight			
	Dry matter	Crude protein	Ether extract	Ash
Pilchard, No. 1	92.75	68.20	3.09	13.73
Pilchard, No. 2	90.57	65.50	4.35	11.88
Spoiled pilchard	91.16	57.80	3.92	20.36

Both bacterial decomposition and simple chemical hydrolyses took place. A bacterial count of the spoiled meal showed more than a 20,000-fold increase in numbers over the commercial sample, with no appreciable mold growth. The spoilage probably followed both possible paths—formation of nitrogenous bases and liberation of ammonia. This partial decomposition was purposely permitted to become more extensive than would ordinarily occur, in order to accentuate any alteration of the meal.

The commercial and experimentally spoiled meals were incorporated into the following otherwise purified diets: For rats—fish meal to bring the total protein content to 20 percent by weight; sucrose, 20; lard, 12; cod-liver oil, 2; salt mixture, U. S. P. XI No. 2, 4; dried brewer's yeast, 2; wheat germ, 1; and dextrin to 100 parts by weight. This was supplemented by 0.12 mg. of thiamin hydrochloride and 1.2 mg. of crystalline riboflavin per kg. of diet. For chicks—fish meal, 35; dextrin, 36; sucrose, 15; lard, 5; salt mixture, 2; agar, 2; cod-liver oil, 2; dried brewer's yeast, 2; and wheat germ, 1 part by weight. The crystalline vitamin supplements were similar to those incorporated in the diet for rats.

RAT-FEEDING EXPERIMENT

The rats were allotted, at an initial live weight of 48 to 55 g., into 2 groups of 9 animals each. Diets containing commercial and experimentally spoiled pilchard meal were self-fed for a period of 60 days. The rats were housed individually in cages with screen floors, and twice weekly records of body weight and food consumption were taken. All animals were in excellent health at the close of the experimental period.

The small difference in food intake between groups can be corrected for in large measure by comparing the efficiency of the 2 diets on the basis of gain in weight per gram of protein intake (table 2). No statistically significant difference was found between these indices of efficiency for the 2 diets. Apparently the spoilage of the commercial pilchard meal under these conditions produced no detrimental effects on the nutritive value of the meal for rats.

TABLE 2.—*Data on gain in weight and food intake of rats after 60 days, when fed a diet containing about 30 percent pilchard meal*

Diet designation	Number of rats	Average gain in weight	Average food intake	Gain in weight per gram of protein intake and standard error
		Grams	*Grams*	*Grams*
Commercial pilchard	9	165	548	1.51±0.13
Spoiled pilchard	9	155	564	1.37±0.13

CHICK-FEEDING EXPERIMENT

Most of the unfavorable reports in the literature concerned poultry-feeding experiments, so a possibility existed that the lack of significant differences when feeding spoiled and unspoiled meal to rats may have been due, at least in part, to variations in species reaction. For this reason chicks were substituted for rats and the feeding experiments were repeated.

Day-old, cross-bred chicks were allotted into 2 comparable groups that received diets containing unspoiled and experimentally spoiled meal, respectively. The diets were self-fed after the third day, and the chicks were kept by groups in cages with screen floors. Group feed consumption and individual body weights were recorded at weekly intervals. After 4 weeks some of the birds began to develop a leg weakness that was believed to be perosis. This condition did not seem to alter the growth rate of most of the chicks, however, so the experiments were continued for a total of 7 weeks. Apparently no toxic compounds were produced by the spoilage conditions, since the average weight of the survivors of the group fed the treated meal was greater than that for the group fed the commercial meal (table 3).

In the second series of experiments the protein content of the diets was adjusted to 22 percent by weight, while in a third series 30 parts per million of manganese were added as $MnSO_4 \cdot H_2O$ to prevent perosis. The results of the second and third series of experiments were essentially the same as the first, since chicks fed the spoiled-meal diets did about as well as those fed commercial meal. An excessive mortality was experienced, but this condition cannot be ascribed to the fish meal because all the chicks died when fed a diet in which

lactalbumin was substituted for the fish meal (table 3). Some essential ingredient was deficient in these diets. It was soon found that the chicks fed the commercial meal were suffering from a deficiency of vitamin K, as evidenced by prolonged blood-clotting time, while chicks fed the spoiled-meal diets did not show this deficiency symptom.

Another series of experiments was conducted to determine whether a deficiency of vitamin K was responsible for the high mortality. One group of chicks received the commercial pilchard-meal diet in which 3 percent of lard was replaced by an equal weight of soybean oil as a source of vitamin K and the antiencephalomalacia factor. Another group received a different sample of pilchard meal. Fewer chicks died, but evidently the diet was still not properly balanced for all food essentials. The vitamin K deficiency was corrected by the addition of soybean oil, so 3 percent was included in all subsequent diets. Various supplements were added to the basal diet in an effort to reduce the mortality by supplying the deficient substances. Three percent additional yeast, 1 percent liver extract concentrate, 1 percent wheat-germ oil, 0.005 percent of nicotinic acid, or 5 percent of alfalfa leaf meal had no beneficial effect when incorporated singly in the commercial pilchard-meal diet.

Only 2 of 8 chicks fed the lactalbumin diet, and 2 of 10 fed the commercial pilchard-meal diet, died when the quantity of thiamin hydrochloride was tripled. None died in the group fed the spoiled pilchard-meal diet. For control purposes a group of 8 chicks was fed a chick mash made up as follows: Fish meal, 7.5; meat scrap, 7.5; dried skim milk powder, 5; alfalfa meal, 7.5; bone meal, 1; ground limestone, 2; salt, 1; cod-liver oil, 1; wheat bran, 16; ground yellow corn, 30; ground wheat, 10; and ground barley, 11.5 parts by weight (Almquist, Jukes, and Newlon, 1938). This mash gave better results than the commercial pilchard-meal diet, but did not produce as large birds as those receiving the spoiled pilchard-meal diet (table 4). These tests were repeated with about the same results.

Most of the chicks fed the diet containing spoiled pilchard meal attained a greater final weight at the end of a 6-week period than did chicks receiving the commercial pilchard-meal diet. Evidently some factor in addition to vitamin K was being synthesized by the spoilage process. There is reason to believe that this factor may be a member of the vitamin B complex, since the addition of 1 percent of a rice-bran extract concentrate to the commercial pilchard-meal diet produced marked improvement in results. Also, when 3 percent of rice-bran concentrate was added to the diet the chicks attained a weight greater than those fed either the spoiled-meal diet or the chick mash (table 4, experiment No. 5). Rice-bran extract concentrate is known to be an important source of several vitamins of the B complex.

TABLE 3.—*Final weight, food intake, and number of cases of perosis of chicks fed diets containing about 30 percent pilchard meal*

Diet designation	Number of chicks		Average weight of survivors	Coefficient of variability in weight	Average food intake of survivors [1]	Total number of cases of perosis	Gain in weight per gram of protein [2]
	Initial	Survivors					
			Grams	*Percent*	*Grams*		*Grams*
EXPERIMENT No. 1 (7 weeks)							
Commercial pilchard	9	1	210		548	0	1.5
Spoiled pilchard	9	8	454	16.3	937	5	1.9
EXPERIMENT No. 2 [3] (6 weeks)							
Commercial pilchard	11	2	179	9.1	467	0	1.3
Spoiled pilchard	10	2	153	57.0	442	2	1.1
Commercial pilchard, plus manganese	8	0				0	
Spoiled pilchard, plus manganese	8	6	301	30.1	664	0	1.7
Lactalbumin [4]	5	0				0	
EXPERIMENT No. 3 [5] (6 weeks)							
Commercial pilchard, sample 1	8	0					
Commercial pilchard, plus soybean oil	7	4	223	20.8	519		0.8
Commercial pilchard, sample 2	8	4	237	26.0	620		1.0

[1] Data based on food intake per chick day.
[2] Data based on survivors.
[3] The protein content was adjusted to 22 percent by weight, and 30 parts per million of manganese were added to the diets.
[4] All dead at the end of 3 weeks.
[5] Diets contained 150 parts per million of added manganese.

TABLE 4.—*Final weight, food intake, and gain in weight per gram of protein of chicks fed mash and diets containing lactalbumin and pilchard meal*

Diet designation	Number of chicks		Average weight of survivors	Coefficient of variability in weight	Average food intake of survivors	Gain in weight per gram of protein
	Initial	Survivors				
EXPERIMENT No. 4 [1] (6 weeks)						
			Grams	*Percent*	*Grams*	*Grams*
Commercial pilchard	10	8	346	35.7	707	2.3
Spoiled pilchard	10	10	547	18.5	1,034	2.4
Chicken mash	8	8	402	22.6	1,152	1.9
Commercial pilchard plus 1 percent of rice-bran concentrate	10	10	488	12.6	1,003	2.3
EXPERIMENT No. 5 [1] (6 weeks)						
Commercial pilchard	9	9	298	15.1	872	1.3
Spoiled pilchard	10	8	463	15.2	1,261	1.5
Chicken mash	9	9	412	14.0	1,462	1.4
Lactalbumin	8	6	152	26.2	451	1.2
Commercial pilchard plus 3 percent of rice-bran concentrate [2]	9	8	493	13.2	1,268	1.6

[1] Fish meal and lactalbumin diets were supplemented with 0.36 mg. of thiamin hydrochloride per kg. of diet.

[2] The revised diet consisted of pilchard meal, 31.4; dextrin, 35.6; sucrose, 15; wheat germ, 1; yeast, 2; agar, 2; salt mixture, 2; lard, 3; soybean oil, 3; cod-liver oil, 2; and rice-bran concentrate 3 parts by weight. It was supplemented with 0.36 mg. of crystalline thiamin hydrochloride, 1.2 mg. of riboflavin, and 100 parts per million of added manganese per kg. of diet.

DISCUSSION

The hemorrhagic symptoms reported by Dam and Schöenheyder as due to feeding fish meal have since been shown to be caused by a deficiency of vitamin K. Almquist and Stokstad (1936) found that chicks fed a vitamin K deficient diet containing 17.5 percent of ether-extracted fish meal showed hemorrhagic symptoms and a high death rate after a 3-week period. In the experiments herein reported, the introduction of a known source of vitamin K in the diets at sufficient levels to reduce the clotting time of the blood to normal values did not significantly reduce mortality.

The crippled feet in chicks reported by Halpin and coworkers (1935) to be the result of feeding diets containing a high percentage of fish meal may possibly have been a type of perosis accentuated by the high ash content of such diets. This conclusion was tentatively corroborated by the data from the first experimental series with chicks. No evidence of perosis was apparent, however, when the chicks were fed diets containing more than 30 percent pilchard meal supplemented with at least 30 parts per million of manganese.

The experimental data show no evidence that the type of spoilage used in these tests produced toxic compounds, and such spoilage did not reduce the nutritive value of the meal. The mortality was admittedly quite high in the first experiments with chicks, but normal death rates were experienced after the diet was properly supplemented with vitamins and manganese. The supplemented commercial pilchard-meal diet compared very favorably with the chick mash in producing larger birds at the end of a 6-week period. There was a comparatively large intake of fish meal from the diets, a fact that should have emphasized the acute symptoms of toxicosis if deleterious compounds were in the meal. The data indicate, however, that the experimental spoilage of pilchard meal produced no significant quantities of deleterious compounds. Spoilage apparently augmented the vitamin content, but deliberate spoilage is not recommended since the vitamin content of practical poultry rations can easily be balanced by the addition of such feeds as leafy legumes and a variety of cereals. The addition of manganese to the diets largely alleviated the deficiency that was responsible for the symptoms of perosis.

It would seem, therefore, that any toxicity ascribed to feeding commercially available fish meal probably was due to a deficiency state rather than a toxicosis produced by the action of one or more organic toxic compounds. This simply means that ordinary care must be taken to balance properly the vitamin and mineral content of the rations, making full use of fish meal as a source of a superior protein and some of the necessary minerals.

CONCLUSIONS

1. Experimental spoilage of commercial pilchard meal by heat and moisture does not adversely alter the nutritive value of the protein.

2. Experimental spoilage of the meal greatly increased the vitamin K content.

3. Chicks fed an otherwise purified diet containing 31 percent of pilchard meal were successfully raised to the age of 6 weeks, after which the experiments had to be terminated owing to lack of equipment. This diet compared favorably with a standardized chick mash in producing healthy birds.

4. Pilchard meal is recommended for liberal use in farm-animal rations as a source of superior protein and some necessary minerals, provided ordinary care is used to balance the rations for other essential food elements.

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