CARBOHYDRATE DIGESTION IN THE YOUNG CALF

I. EFFECT OF PECTIN UPON THE CONSISTENCY OF CALF FECES.
II. CORN STARCH, DEXTRIN AND CORN SUGAR AS CARBOHYDRATE SOURCE IN CALF SYNTHETIC MILKS.

A THESIS FOR MASTER OF SCIENCE

BY

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This is to certify that the

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THESIS

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CARBOHYDRATE DIGESTION IN THE YOUNG CALF

I. Effect of Pectin upon the Consistency of Calf Feces

INTRODUCTION

Investigations have shown that scours is the greatest problem encountered in the raising of dairy calves, often accounting for as much as 40 percent of the total calf losses.

Advances such as the nipple pail, heavy wire floors and the use of the sulfa drugs in the treatment of scours are rapidly reducing the rate of calf losses. Regardless of the effectiveness of the sulfa drugs as curative agents, a need still exists for means of preventing digestive disturbances.

The present investigation was undertaken to determine the effectiveness of pectin in the regulation of the digestive tract of young calves.

REVIEW OF LITERATURE

Pectin

Chemistry

Ahmann and Hooker (1925) developed a method of estimating pectin by titrating the acidity which developed following saponification of pectin, and found that under constant conditions the amount of pectin was proportional to the acidity developed. The titration curves of pectic acid showed eleven carboxyl groups, but since they demonstrated that saponification was not complete pectic acid evidently contains more than eleven carboxyl groups. On the arbitrary basis of twelve carboxyl groups, Ahmann and Hooker calculated the minimum molecular weight of pectic acid as 2124. This theory proposed a pectic acid nucleus of galacturonic-galactonic acid, with at least six of these nuclei linked to form pectic acid.

Pectin in the unhydrolyzed condition, according to Myers and Baker (1934), is a monoarabino-monogalacto-diacetyl-hepta-methoxyl-octagalacturonic acid. Assuming that twenty molecules of water are eliminated when one molecule of arabinose, one molecule of galactose, two molecules of acetic acid, seven molecules of methyl alcohol and eight molecules of galacturonic acid are combined, the molecular weight of pectin would be 1866.784 and the empirical formula $C_{70}H_{98}O_{58}$. Apparently four molecules of galacturonic acid combine with the elimination of four molecules of water to form the ring compound, tetragalacturonic acid. Subsequently two molecules of tetragalacturonic acid combine with the elimination of one molecule of water to form octagalacturonic

acid, the nucleus of the pectin molecules. Thus the octa-acid probably is built up from eight molecules of galacturonic acid with the elimination of nine molecules of water. Seven of the eight carboxyl groups of octagalacturonic acid are methylated while the eighth one is free.

Studying the pectin obtained from apple pomace, Hirst and Jones (1939) reported that pectic acid was composed mainly if not entirely of anhydrogalacturonic residues, and that the pectin was a mixture of the methyl ester of pectic acid, araban and galactan. The araban portion was isolated in the form of its methylated derivative. Upon hydrolysis of the derivative, equimolar proportions were obtained of 2:3:5-trimethyl l-arabinose, 2:3-dimethyl l-arabinose and 3-methyl l-arabinose.

Bonner's (1936) conclusion, that for a complete understanding of pectic substances, much remains to be done by the chemist, appears to be as true today as the day he made it.

Preparation

Pectin has been prepared by several methods and from a number of sources. There is a long list of substances from which pectin may be prepared, but most practical methods apply either to citrus fruit or to the apple. Ohn (1926) described a procedure in which pectin is prepared by the extraction of orange peel with distilled water and alcohol precipitation. Griggs and Johnstin (1926) prepared pure pectin from lemon albedo, and stressed that a rapid alcohol extraction must be made before grinding, and that dialysis was the best method of removing calcium salts and other electrolytes. Pectin was precipitated by adding alcohol to the pectin sol a drop at a time; the precipi-

tate was flocculated in an electric field and filtered through a silk cloth. These workers felt that a knowledge of the colloidal behavior of thepectin sols was the key to commercial utilization of pectin.

The extraction of pectin from apple pomace was studied by Burroughs et al. (1944). Their report stated that pectin is most effectively extracted from apple pomace at a pH of 3. This work was conducted on the apple fruit at various stages of maturity and it was found that losses of pectin were large only when the apple is allowed to become over-ripe before being milled. Stage of maturity, however, had little or no effect upon the optimum pH for extraction of pectin.

Apple pomace containing varying amounts of moisture was stored for as long as five months by Kertesz and Green (1931), and changes in the quantity and quality caused by the moisture content determined. In general no mold growth was observed on samples containing 20 percent or less of moisture while those samples containing 33 percent or more of moisture had considerable mold growth and a consequent decrease in soluble pectin. The acid hydrolyzable pectin, as well as water soluble pectin, was decreased in the sample containing 50 percent moisture.

Properties

Bennison and Norris (1939) found that the composition of the pectin had some effect on its ability to form jellies, but that molecular size (as indicated by viscosity) was a much better indication of jellying power. It must be cautioned that any treatment which tends to disaggregate the poly-galacturonide chain of the pectin molecule tends to cause a loss of jelly

strength. Method of preparation, then, is a vital factor with respect to jelly strength and to viscosity. Autoclaving is sufficient to inhibit jelly formation, and both jelly strength and viscosity decrease at rapid rates when a pectin is heated. Even passing into solution will reduce viscosity at first, but viscosity becomes constant with respect to solution after a few hours. In general, it can be said that high urone content indicates high jelly strength, but otherwise there is not much relation between chemical composition and jelly strength; methoxyl groups in particular are no indication of jellying power.

Baker (1940) declared that since the physical properties of pectins are so important in determining their role in feeding stuffs, it would seem that a mere quantitative statement of the amount of pectin material in the nitrogen-free extract would be of little value in indicating the actual merit of the pectin. He suggests that in addition to quantity of pectin, such things as viscosity, methoxyl content, quality in terms of grade, ash content, and the initial pH of solution should be given.

Olsen (1940) states that commercial pectins are adjusted as to grade by dilution with various substances such as cerelose and glycerine, and that it is particularly important that when pectin is used in therapy, the physician know not only grade but also such things as what diluents are present, ash content, and the combining properties of the preparation.

Analysis

Innumerable methods have been described for determining pectin in various biological substances, but the method of Baier (1945) is probably one

of the most accurate and rapid, and is well adapted to work with animals. This method calls for extraction with alcohol and phosphoric acid. The furfural developed is treated with aniline and glacial acetic acid and measured by a photoelectric colorimeter.

Control of Laxation

Man's search for food which will regulate the activity of the digestive tract and such associated factors as the consistency and bulk of feces, regularity of bowel movement and laxation has continued through the years.

Bran

Bran has been shown to be laxative, as have various cellulose and hemicellulose products. When rations low in the B complex vitamins were used by Rose et al. (1932), a much larger amount of bran was required to produce a laxative effect than was required when normal rations were used. Rations free of vitamin B required a still higher proportion of bran to obtain laxation. Bran appeared to be as effective as such laxatives as senna tea, cascara and milk of magnesia. The effect of the addition of bran to vitamin B deficient diets in causing laxation is attributed to the combined effect of fiber and vitamin B. Histological studies of the alimentary tracts of rats on various levels of bran for various lengths of time gave no evidence of pathological lesions.

After these tests with rats, Rose et al. studied the effect of bran on humans, judging laxation by the number of defecations daily. The daily addition of 14 grams of bran to the breakfast cereal resulted in definitely

increased laxation in 50 percent of the cases, no effect in 15 percent and questionable results in 35 percent of the cases. Large quantities (34 grams daily) of bran ingested for extended periods produced as satisfactory results the second month as were obtained the first. In other words, the effect of bran was not reduced upon continued ingestion.

In substantiation of this report, Hoppert and Clark (1942a) tested human subjects at a level of two ounces of bran daily orver a 30 week experimental period. Their results are summarized as follows:

Subject	Initial Weight	Final Weight	Total Number of Stools	Average Number Stools Daily	Average Moisture Content
	Pounds	Pounds			Percent
1	128	125	221	1	76.1
2	198	201	376	2	77.9
3	176	182	395	2	77.7
4	163	162	214	1	74.5
5	142	142	360	1.8	77.8
6	212	215	319	1.6	75.9

Bran, in twice the amount usually recommended, produced no loss in weight of the subjects and showed no diminuation in its laxative properties even upon long continued use. It should be noted, however, that no controls were used in this study.

Bran in the form of muffins exerts a laxative effect, according to Hoppert and Clark (1942b). In this study a basal diet low in crude fiber was supplemented by five, four, three and two muffins daily, containing 1.0, 0.8, 0.6 and 0.4 ounces of bran, respectively. The number of movements which were classified as easy averaged 33 percent for the basal periods as compared

to 96 percent for the various supplemented periods. The conclusion reached was that the addition of three or more muffins to the low fiber diet produced a marked effect upon laxation whereas two muffins daily failed to produce a similar effect on overall laxation but demonstrated a desirable effect on consistency of the stools and on the ease of movement.

In a third laxation study Hoppert and Clark (1945) tested the effect of several common foods such as bran, lettuce, cabbage, oranges and apples on digestibility and laxation. It was found that a certain optimum intake exists for bulk-forming foods of the bran class beyond which no added benefit is obtained as a result of increased intake. For bran this optimum intake was about one ounce per day.

Fantus and Frankl (1941) studied the effect of bran upon the composition of stools, and reported that (1) the most significant change following the addition of bran was a softening and an increase in stool weight, (2) the increase in bulk of bran stools is greater than the increase in weight, (3) volatile fatty acids, chiefly acetic and butyric acids, increase but not in proportion to the increase in weight and (4) the proportion of acetic to butyric acid showed no consistent correlation with laxative action as measured by the increase in stool weight.

A review of the world literature by Fantus and Kopstein (1940) revealed only four cases in which bran was associated with intestinal obstruction. One of these was not sufficiently well described to permit analysis as to its nature, but in the other three the impaction was preceded by gross intestinal pathology. It appears, then, that bran is not likely to produce intestinal obstruction in the absence of a predisposing cause.

Unavailable Carbohydrates

In the course of their passage through the alimentary tract, cellulose disappears in larger amounts than does lignin, but in smaller amounts than does hemicllulose. Williams and Olmstead (1936) attacked this problem by concentrating the so-called indigestible residues (hemicellulose, cellulose and lignin) of several food substances and adding these concentrates to the non-residue diets of human subjects. The volume of feces appeared to be influenced more by the amount of cellulose and hemicellulose which disappears during passage through the tract than by either the amount of residue fed or the amount of residue recovered in the feces. It was found that the quantity of volatile fatty acids in the stool varied directly with the disappearance of a residue during its passage through the gastrointestinal tract.

Foods which are nearly equal in total residue often vary markedly in the proportionate amounts of lignin, cellulose and hemicellulose present. Little or no significance was attached to such findings until Hummel and associates (1940) demonstrated that although the total residue remained the same throughout a series of changes in food intakes, the mean daily intakes of lignin, cellulose and hemicellulose were changed as follows:

Lignin,	from	62.8	to	79.8	milligrams	per kilogram
Cellulose,	"	114.2	to	117.1	"	" "
Hemicellulose,	"	117.9	to	109.7	"	" "

This alteration in the distribution of the unavailable carbohydrates has a definite influence upon the nutritional processes of the body. The authors also state that the food in which the fiber is provided affects the response of the body to it, and that alkalinity and such constituents of the food as vitamins, pectin and tannin may alter the physiologic response.

Apple and Pectin

The use of pectin for the treatment of diarrheal conditions in infants has been widely accepted in the field of pediatrics. Pectins of various origins and combined in numerous ways have proved effective, but perhaps the most widely acclaimed of all is the apple pectin, either in the raw fruit or after purification. Hunt (1936) preferred the use of raw apple for the treatment of pediatric diarrheas, chiefly because of the ease of administration and its cheapness. He found, however, that some cases that did not respond to raw apple were benefitted by pectin agar treatment. In 1936 Malyet declared that the value of the apple in diarrheal therapy was in its pectin content, and recommended apple in preference to pure pectin because of the pleasant acid taste, the utilizable sugar provided and the ability of the apple to satisfy thirst. He agreed with Hunt that pure pectin was frequently successful where the apple diet had failed.

Contradictory to these results were those secured by Frank (1937), who found that a dried apple product cured diarrhea in children in four days in 83 percent of the cases, whereas a pectin preparation produced cures in only 16.2 percent of the cases. The pectin preparation was particularly ineffective in the treatment of younger children. Frank concluded, therefore, that the effect of apple was not through its pectin content.

Manville and coworkers (1937) summarized the many proposed theories of the action of apple and apple powder. These they grouped into the following classes: acids, including tannic, acetic, butyric and lactic; sugar and starches; cellulose and hemicellulose; vitamins, andpectin. Also in 1937 the Council on Foods of the American Medical Association endorsed the

use of the apple as a therapeutic agent in the dietary management of diarrhea, although the mode of action responsible was not known.

Dack et al. (1939) compared the bacterial flora from the intestine and cecum of monkeys on control and raw apple diets. Fistulas at three levels of the bowel were used to study the quantity and types of organisms present. No essential differences were observed between the two groups; thus these investigators concluded that the therapeutic effect of the apple is not through changes in the bacterial flora.

Reithel and Manville (1938) indicated that the addition of apple to milk formulas for infants accomplished two purposes, a lowering of the pH and the formation of a softer curd. In those cases in which infants had difficulty in digesting cow's milk, the incorporation of four to five percent apple in the formula was advisable.

As early as 1936 Winters and Tompkins (1936) offered a pectin agar substitute which overcame some of the disadvantages of the scraped apple treatment. This preparation consisted of 175 grams of dextrin-maltose, 6 grams of pectin and 8 grams of agar-agar boiled in a pint of milk or water for three to five minutes. The authors obtained better responses in patients treated with the substitute than in those treated with the scraped raw apple. Winters and coworkers (1939) reported on the pectin-agar treatment of 52 cases of bacillary dysentery and 27 cases of infectious gastroenteritis. A gradual and steady improvement of 73.4 percent of the cases was obtained, with the formation of soft stools within an average of 34 hours. Caloric intakes averaged 33 to 52.4 calories per pound per day and average weight gains were observed in all groups. The diet was well adapted to infectious cases, being high in calories, well balanced and easily assimilated, and capable of

maintaining nutrition while the body attempts to build the necessary immunity. The mode of action was believed to be both physicochemical and mechanical, with uronic acids playing an important part.

A similar pectin-agar mixture was successfully used by Kutscher and Blumberg (1939). They tested the preparation with and without the dextrin-maltose, and concluded that the results justified the addition of the carbohydrate. Howard and Tompkins (1940) extended the pectin-agar therapy to include older infants and children with favorable results. The simplicity of this method of treatment is of particular value in home treatment.

Block and associates (1939) compared the effects of pure pectin and nickel pectinate in the treatment of chronic bacillary dysentery. Nickel pectinate was found to be superior, possessing detoxifying, bactericidal and antihemorrhagic properties of value in the treatment of bacillary dysentery. The antihemorrhagic properties of pectin had previously been demonstrated by Gohrbandt (1936). He showed that the coagulation time was reduced for as long as six days following the intramuscular injection of pectin, both in normal and diseased persons. The action was believed to be indirect, since intravenous injection produced results no more rapidly than did the intramuscular injection, and the coagulation time was increased by pectin in vitro.

The detoxifying mechanism of pectin was studied by Manville et al (1936). Analysis of the daily urinary excretion of urea and uronic acid by rabbits on test diets led to the discovery that menthol stimulates the breakdown of body protein and that pectin tends to reduce this effect, probably through the galacturonic acid formed on cleavage of the pectin molecule.

Pectin has received favorable acceptance as a blood substitute, as indicated by the results of Hartmand and associates (1941). Pectin solutions appeared to be valuable in the management of shock, a one-half percent solution having about the same viscosity and osmotic pressure as whole blood. It was found to be nonantigenic and nontoxic, and although pectin is temporarily retained in the blood and liver, it is rapidly excreted in the urine in the unchanged form.

Further studies on the fate of ingested pectin were made by Werch and Ivy (1940,1941a,b,c). Early studies with dogs indicated that when 20 grams of pectin were fed per day with a mixed diet, 90 percent of the pectin was decomposed, but when the same amount of pectin was fed during fasting only 50 percent of it was decomposed. In humans on a mixed diet with pectin added, 90 percent of the pectin was decomposed, but in fasting the decomposition of pectin was greater in humans than in the dog. It was shown that pectin was decomposed largely in the large intestine by bacterial enzymes and that very little if any breakdown occurred in the small intestine. Later, when pectin-broth media were inoculated with feces of humans and canines on normal diets or on diets with added pectin, it was found that pectin was decomposed in the presence of the bacteria of the feces. This offered substantial evidence that the breakdown of pectin was accomplished by bacterial enzymes. These authors further demonstrated that pectin is not excreted in the urine as galacturonates, and that galacturonic acid is not absorbed in appreciable quantities from the small intestine and colon. From these observations they concluded that the favorable effects of a diet containing apple or pectin in the treatment of diarrhea cannot be ascribed to an improvement of the detoxicatory function of the liver as a result of the absorption of galacturonic

acid. They believed that the favorable effects were exerted exogenously in the lumen of the bowl.

Kertesz (1940) found that saliva and gastric juice had no effect on pectin, and that ingested pectin that had passed through part of the small intestine could be recovered without loss. Pepsin, rennet and trypsin were without effect on pectin in vitro. However, pectin incubated with feces was rapidly decomposed. The conclusion reached agrees with that of Werch and Ivy: that pectin taken orally is not attacked until it reaches the large intestine, where it is hydrolysed by bacterial enzymes.

Miscellaneous Uses of Pectin

In addition to its other properties, pectin has been demonstrated to have bacteriocidal and bacteriostatic properties under certain conditions. Prickett and Miller (1939) reported that bacteria flourish in many types of pectin due to the fact that these types do not greatly alter the pH. Pectin's effect upon bacterial growth depended entirely on the modification of pH. Sullivan and Manville (1938) added dehydrated apple to the diet of the rabbit with the result that the hydrogen-ion concentration in the intestinal contents was increased. The intestinal flora was changed from one in which Escherica coli predominated to one in which the acidophilic type of organism was dominant. The number of gas-producing organisms was considerably reduced.

Werch and coworkers (1942) attributed the favorable effect of pectin on diarrheas caused by intestinal pathogens to an alteration of the hydrogen-ion concentration. These workers found thatpectin, galacturonic acid and their decomposition products have both bactericidal and bacteriostatic

activity on the intestinal pathogens. The action was apparently due to the acidity of pectin and its decomposition products. However, at hydrogen-ion concentrations near neutrality, pectin failed to inhibit the growth of members of the colon-dysentery-typhoid group (Steinhaus and Georgi, 1941). Galacturonic acid, they found, failed to inhibit the growth of test organisms, while alpha methyl d-galacturonate did inhibit the growth of many dysentery bacteria. Based on these results, it is logical that the inhibition of growth of bacteria by pectin as reported by other investigators may be due to lowering of the pH in the intestinal tract and to the liberation of free alpha methyl d-galacturonate. Steinhaus and Georgi advanced a hypothesis that the action of pectin is based upon (1) buffer action, (2) inhibition of peristalsis through calcium and magnesium content, (3) a protective colloid which enmeshed bacteria and (4) detoxifying effect due to uronic acid content.

Manville and Sullivan (1940b) found the bactericidal action of pectins to vary with their composition, and with the pH of the medium. Of the pectins tested, only a non-acid pectin was found to be bactericidal at a pH of 5.0 to 5.5. However, as the pH was lowered to 4.6, the apple and citrus pectins became more bactericidal than the non-acid pectin. Pectic acid was found to be less inhibitory than pectin at the same pH. The use of K_2HPO_4 to bring the pH of the various pectins to 5.0 resulted in the loss of their bacteriostatic effect. Dehydrated apple powder, also used in diarrheal therapy, was shown to have no bactericidal or bacteriostatic action at a pH as low as 4.6.

Arnold (1939) reported that metal pectinates exert bactericidal action but pectin alone possesses no bactericidal power. Manville and Sullivan (1940a) found that the presence of dehydrated apple in culture media neither enhance nor detracted from bacterial growth, but advocated its inclusion because of (1) the copper it contained, (2) the organic acids for pH adjustment, (3) the materials from which organic acids may be derived in the intestine and (4) the carbohydrate calories which are not only nutritive but also combat acidosis.

Summary of Review of Literature

The pectin molecule is complex and its structure not too well understood. Several theories as to its structure have been advanced and none definitely proved. Apparently much remains to be learned of the structure and components of pectin.

Pectin may be prepared from a number of substances the most important of which are citrus fruits and the apple. Methods of preparation markedly influence the properties of the pectin, with the pH being one of the most important factors. Pectin is commonly classified as to grade, but it is important that when used medicinally, such things as diluents, ash content and combining properties be known.

Regulation of the digestive tract in man has been attempted through the use of numerous substances. Bran has long been recognized as a regulator and has given little if any evidence of harmful effects. Cellulose, hemicellulose and lignin influence the volume of the feces and are thought

to be regulatory.

Pectin, either in the pure form or in the form of fresh or dehydrated apple, enjoys great popularity as a treatment for diarrhea in infants. Raw apple has been preferred by some pediatricians, while others have claimed that pure pectin is more effective. The mode of action is not generally understood, although it has been demonstrated that the apple does not alter the bacterial flora.

The combination of pectin with dextrin-maltose and with agar-agar produced a more effective and desirable treatment of gastrointestinal disorders. Some of the metal salts of pectin have proved more effective than pure pectin.

Normal digestive processes have little if any effect upon pectin, which is decomposed only through the action of bacterial enzymes in the lower tract. Bactericidal and bacteriostatic properties of pectin are believed to depend almost entirely upon the modification of pH, as pectin has little or no effect on growth of bacteria at or near neutrality.

OBJECT

The object of this study is to test the ability of pectin to alter the consistency of the feces of calves on normal calf rations. If successful with normal rations, pectin will be considered for use in synthetic rations for calves.

PLAN OF EXPERIMENT

Animals Used

Four calves varying in age from birth to two months of age will be available for the preliminary experiment. Whether or not additional animals will be used depends upon the results secured in the preliminary trial, and upon the availability of animals.

Feeding of Animals

Calves will receive the standard ration fed calves in the experimental herd. Feeding will be twice daily by the experimental barn herdsman. The only alteration of the normal feeding procedure is that the daily pectin allottment will be mixed with the milk just prior to the morning feeding.

Collection of Feces Samples

Calves will be maintained on wire floors without other bedding.

Feces samples will be collected at approximately the same time each day, and each pen will be cleaned immediately after the sample is collected. If more than one sample is available in a pen, the freshest sample will be used in the determination of consistency.

Measurement of Consistency

An instrument suitable for the measurement of consistency of feces will be selected, and a standard procedure adopted for testing all samples.

General

Observations of the condition and general well being of the calves will be made daily. Abnormalities will be noted and reported by the herdsman. Weekly weights will be obtained as per standard procedure for the experimental barn.

EXPERIMENTAL PROCEDURE

Animals Used

Four calves--three Holstein and one Ayrshire--were placed on the experiment as planned. The results secured led to the decision to proceed to another phase of the experiment without devoting additional time to the problem of regulation of the digestive tract.

Feeding of Animals

As planned.

Collection of Feces Samples

As planned.

Measurement of Consistency

Several instruments were tested for the measurement of consistency of calf feces. Each of these proved unsatisfactory in one or more respects. A simple instrument was devised which proved quite satisfactory for this type of measurement. The device is illustrated in Fig. 1. A lever (A) is balanced on a pivot (B) by means of a movable weight (C). A

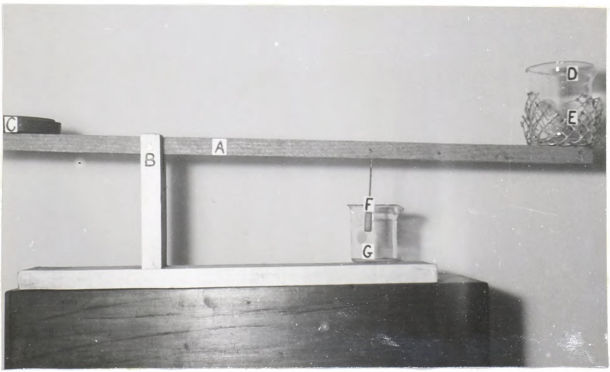


Fig. 1. Device for measuring the consistency of feces.

250 milliliter beaker (D) rests in a wire basket (E), and exerts pressure on a plunger (F) one-fourth inch in diameter. The plunger is marked at a distance of exactly 3/8 inch from the lower end. The feces sample to be tested is well mixed, then placed in a 150 milliliter beaker (G). Weight (C) is adjusted in such a manner as to allow the plunger to touch but not to rest upon the surface of the sample. Water is added slowly to beaker D until the plunger penetrates to the 3/8 inch mark. The quantity of water used is measured in a graduated cylinder, and the amount of water in milliliters is the consistency reading. Readings were taken with water at approximately 20° C.; the readings therefore approximate the weight in grams required to cause the one-fourth inch plunger to penetrate to a depth of 3/8 inches. This was not an absolute measurement but proved rather successful as a means of measuring relative consistency.

General

As planned.

EXPERIMENTAL RESULTS

The consistency of the feces of individual calves is shown in Tables IX, X, XI and XII. Each calf served as its own control, and alternated between control and supplemented periods.

Consistency During Control Periods

The consistency of the feces during the control periods is shown in Table I. From these data it is obvious that there is much variation

TABLE I. CONSISTENCY OF FECES DURING CONTROL PERIODS

Calf Number	Average Age	Number of Samples	Mean Consistency	Standard Error
	days			
C651	35	6	20.3	1.19
	70	7	31.7	4.14
C652	37	8	15.0	3.93
	68	14	78.5	9.94
	96	7	71.1	20.27
C658	7	5	10.6	3.65
	29	6	36.5	2.57
	65	15	18.8	5.19
A70	84	7	80.0	15.97

in consistency of feces under normal conditions and following accepted feeding practices. At no time during the control periods was there any indications of calves being off feed nor was there any scouring. It should be noted that despite the great variation, there is in general a tendency for the feces to become harder as the animal advances in age. Also apparent is the fact that as age increases there is greater variation in consistency. Variation, as evidenced by the standard error, definitely tends to become greater with advancing age through the range of ages studied.

Supplementation at the Five Gram Level

Table II shows the results obtained when the basal ration was supplemented with five grams of grade 50 liquid pectin daily.

TABLE II. CONSISTENCY OF FECES AS AFFECTED BY ADDING FIVE GRAMS OF PECTIN TO THE RATION

Calf Number	Average Age	Number of Samples	Mean Consistency	Standard Error
	days			
C651	48	8	43.4	4.12
C652	29	7	30.0	4.10
C658	18	14	39.7	5.08

These figures show much more uniformity both in mean consistency and in standard error than was shown by the data for the control periods; however, cognizance should be taken of the fact that the average age covers a narrower range in these trials than in the control trials.

In Table III the results obtained by supplementing with five grams

TABLE III. COMPARISON OF CONSISTENCY ON BASAL AND BASAL PLUS FIVE GRAMS PECTIN

Calf Number	Basal			Supplemented		
	Av. Age	Consistency	Standard Error	Av. Age	Consistency	Standard Error
	days			days		
C651	35	20.3	1.19	48	43.3	4.12
C652	37	15.0	3.39	29	30.0	4.10
C658	7	10.6	3.65	18	39.7	5.08
Wtd. Av.	28.5	15.5		28.9	38.4	

of pectin are compared with the results in the control period with the same calf, the ages paired as closely as possible. By comparing the results obtained when each calf was supplemented with five grams of pectin daily with the results in the control period which most closely approaches the average age in the supplemented period, it may be seen that in each case there was a definite hardening of the feces during experimental supplementation with pectin. For C651 and C652 the consistency reading was approximately doubled, while for C658 the reading was almost quadrupled by supplementation. Weighted averages for the two periods show practically no difference in average age but a marked difference in consistency.

Supplementation at Other Than the Five Gram Level

Results of feeding pectin in amounts other than five grams daily are shown in Table IV. In these trials the mean consistency ranged from 4.1 when 10 grams of pectin were fed daily to 108.0 when 15 grams were fed.

TABLE IV. CONSISTENCY OF FECES AS AFFECTED BY ADDING VARIED AMOUNTS OF PECTIN TO THE RATION

Calf Number	Average Age	Pectin Fed	Number of Samples	Mean Consistency	Standard Error
	days	gms			
C651	55	10	6	41.1	4.35
C651	62	15	7	47.3	4.67
C652	84	200	14	50.6	6.95
C658	36	1	6	60.7	11.62
C658	48	15	12	108.0	21.99
A70	96	45	16	82.1	7.25

This tremendous change in consistency can hardly be accounted for by the rather small change in the amount of pectin fed. The ages employed in these trials are much greater than those used when feeding five grams of pectin; possibly this accounts for much of the variation in consistency.

Presented in Table V is the comparison of results on the basal ration with the results of supplementation at other than the five gram level. These data show that despite the great variation and the

TABLE V. COMPARISON OF CONSISTENCY ON BASAL AND BASAL PLUS VARIED AMOUNTS OF PECTIN

Calf	Basal			Supplemented			
Number	Av. Age	Consistency	Standard Error	Av. Age	Pectin Fed	Consistency	Standard Error
	days			days	gms		
C651	70	31.7	4.14	55	10	41.1	4.35
C651	70	31.7	4.14	62	15	47.3	4.67
C652	68	78.5	9.94	84	200	50.6	6.95
C658	29	36.5	2.57	36	1	60.7	11.62
C658	65	18.8	5.19	48	15	108.0	21.99
A70	84	80.0	15.97	96	45	82.1	7.25
Wtd. Av.	65.5	46.3		69.9		70.5	

greater consistency due to age, the feeding of pectin in moderate doses increased the consistency of the feces. When massive doses (200 grams per day) were fed, there was a depression of consistency, that is, the feces became softer. Although these data are inadequate for definite conclusions, it would appear that pectin in amounts greater than 50 grams per day (grade 50 pectin) tends to increase the softness of the feces, since at 45 grams per day the consistency roughly equalled that during the corresponding control period.

Effect of Age on Consistency of Feces

The data presented in the foregoing tables indicate that consistency of the feces increases as the calf grows older. For example, control calves averaging 28.5 days of age had an average consistency reading of 15.5 (Table III), while at 65.5 days of age the average reading for controls was 46.3 (Table V), or roughly three times the average at 25.5 days. A grouping of consistency data based upon the age of the calves is presented in Table VI.

TABLE VI. CONSISTENCY OF FECES OF CALVES ON BASAL AND SUPPLEMENTED RATIONS GROUPED AS TO AGE OF CALVES

	Basal			Supplemented		
Calf Number	Av. Age	Number of Samples	Mean Consistency	Av. Age	Number of Samples	Mean Consistency
	days			days		
20 days and under						
C658	7	5	10.6	18	14	39.7
21 - 40 days						
C651	35	6	20.3			
C652	37	8	15.0	29	7	30.0
C658	29	6	36.0	36	6	60.7
41 - 60 days						
C651				51	14	42.2
C658				48	12	108.0
61 - 80 days						
C651	70	7	31.7	62	7	47.3
C652	68	14	78.5			
C658	65	15	18.8			
81 - 100 days						
C652	96	7	71.1	84	14	50.6
A70	84	7	80.0	96	16	82.1

A summarization of these data is given in Table VII. These two tables present the general trend of increasing consistency with advancing age. On

TABLE VII. AVERAGE CONSISTENCY OF FECES OF CALVES OF VARIOUS AGES

Age Classification	Basal		Supplemented	
	Number of Samples	Consistency Weighted Av.	Number of Samples	Consistency Weighted Av.
20 days and under	5	10.6	14	39.7
21 to 40 days	20	22.9	13	44.2
41 to 60 days	0	----	26	72.7
61 to 80 days	36	44.5	7	47.3
81 to 100 days	14	75.5	30	67.4

the basal ration the change is rapid and progressive. On the supplemented ration the change is more gradual and a smaller total change is exhibited. This would imply that pectin is most effective during the early days of life, and decreases in value with the increasing age of the calf. Graphic presentation of these data is made in Fig. 2. The slope of the line for the supplemented feeding is much less than that for the basal feeding (0.34 and 0.81, respectively), emphasizing the smaller rate of change in consistency with age when pectin was fed. Thus pectin, even after placing into one group the results at the various levels of pectin feeding, appears to have some regulatory capacity for the gastrointestinal tract of calves on normal rations.

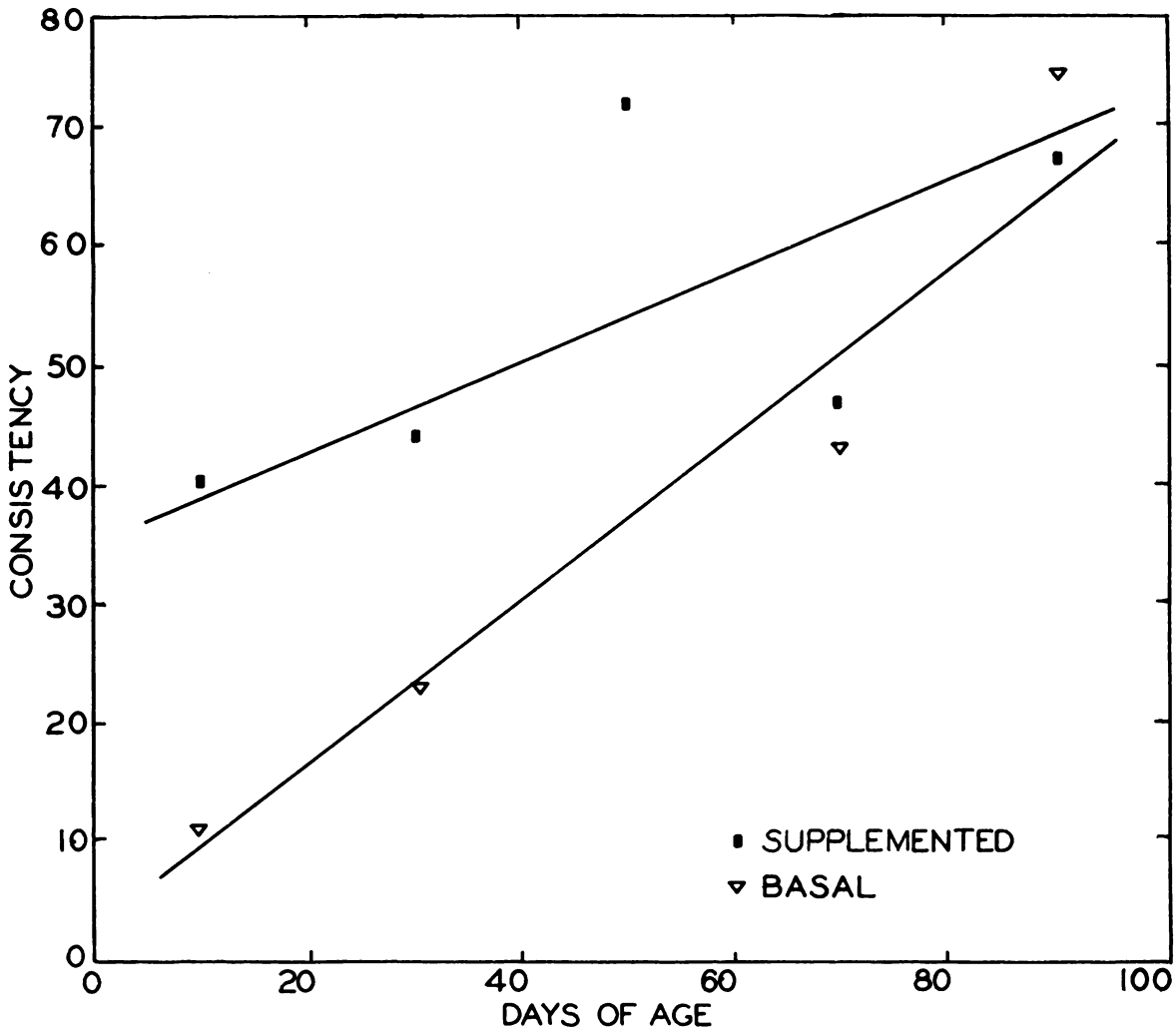


Fig. 2. Average consistency at various ages of calves on basal and supplemented rations.

Effect of Pectin Supplementation on Body Weight

Calves were weighed at weekly intervals throughout the experiment. The weekly weights are given in Table VIII.

TABLE VIII. BODY WEIGHTS OF EXPERIMENTAL CALVES

Age	C651	C562	C658	A70
days				
21			79	
28		104	80	
35	119	113	84	
42	121	124	90	
49	134	135	97	
56	146	143	105	
63	158	155	111	
70	165	165	120	
77	180	182	134	
84		197		147
91		211		162
98		216		181
105				201

Growth curves of the four calves during the experimental period are shown in Fig. 3. The growth curves of the calves during the period of the

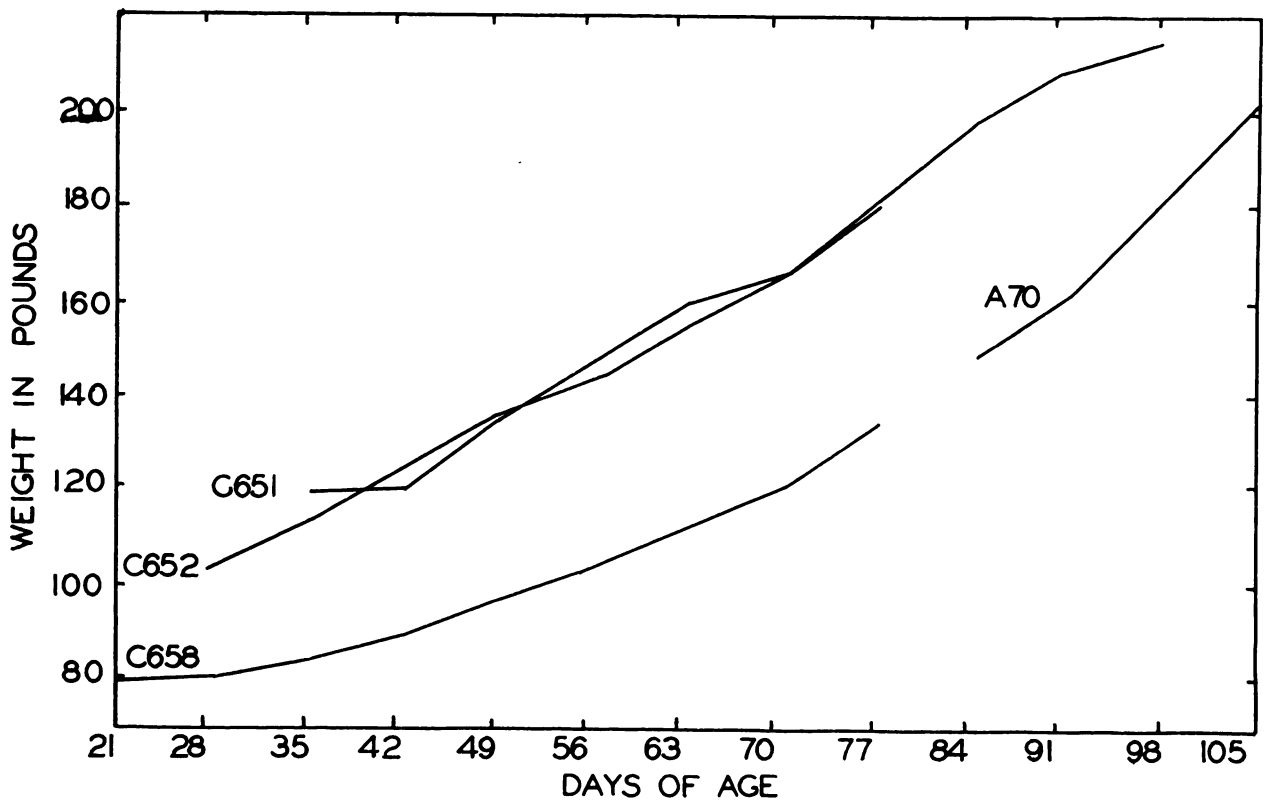


Fig. 3. Growth curves of calves used in pectin feeding experiment.

experiment give no indication of either favorable or unfavorable influences of pectin on the rate of growth. Although each control and supplemented period was short, it is impossible in any single case to point out from the growth curves a change from basal to supplement or from supplement to basal.

DISCUSSION

In the studies conducted in this investigation four calves from the dairy experimental herd were used. The Ayrshire C651 was placed on the experiment at the age of 33 days and removed at the age of 73 days. The three Holsteins, C652, C658 and A70, were placed on the experiment at the ages of 26, 3 and 81 days, respectively, and removed at ages of 99, 73 and 104 days, respectively. Each calf served as its own control, alternating between control and supplemented periods. In each case there was a two or three day break following the change before fecal samples were collected again. The pectin fed in these trials was a liquid apple pectin of grade 50. One trial, with C658 at the one gram per day level of feeding, was conducted with a powdered citrus pectin of grade 180.

Value of Feeding Pectin at the Five Gram Level

Feeding pectin at the five gram level (Table III) increased the consistency of the feces from two to nearly four times that of comparable control periods. The response of C658 to pectin feeding was more notable than that of either C651 or C652. It would appear from these limited data that the younger the calf the greater the effect of pectin on consistency. The average age during the control period was almost identical with the average

age during the supplemented period, whereas the average consistency during supplementation was more than double that during the control periods.

Along with the increase in consistency there was an increase in standard error. This was hardly to be expected, as it was thought that the regulatory action of pectin would tend to eliminate the extremely high and low consistency values and thus produce a smoother curve. The trials of this study were of short duration and it is possible that if pectin were fed for an extended period the regulatory effect, if any, would become apparent.

Value of Feeding Larger Doses of Pectin

Increasing the amount of pectin fed to ten grams daily did not alter the consistency values significantly from the values obtained at the five gram level. A further increase to 15 grams daily increased the consistency but the increase was not significant statistically. These data, obtained on calf C651, indicate that pectin in small quantities is as effective as a regulator as are the larger doses. This hypothesis is supported by the data obtained on C658; one gram daily of a 180 grade citrus pectin almost doubled the consistency of the control period. No attempt was made to establish a correlation between the grade of pectin and the quantity required to produce a change in consistency, but in all probability the quantity required decreases with an increase in grade.

The effect of massive doses of pectin were studied with C652, with 200 grams of grade 50 pectin being fed daily. Feeding such quantities resulted in a lowering of the consistency from 78.5 for the control period to 50.6 for the supplemented period. It would appear, then, that small quanti-

ties of pectin harden the feces while large quantities soften it. If this is true then there must be a transition phase in which quantities of pectin would apparently have no effect upon consistency. Evidently the 45 grams daily administered to A70 was within this transition phase, as there was practically no difference between the supplemented and control periods. The extent or range of this transition phase has not been investigated, and undoubtedly varies from one animal to another as well as from one age to another.

The difference in responsivity of different animals to the same treatment is shown by comparing the results with C651 and C658 when 15 grams of pectin were fed to each. C651 responsed with an increase of 29.6 percent over the control period, while C658 showed an increase of 474.5 percent. On the other hand, the results at the five gram level of feeding are comparatively consistent, ranging from increases over the control periods of 100 percent for C652 to 274.5 percent for C658. C658 showed more variability throughout the experiment than did any of the other calves.

Influence of Age on Action of Pectin

The data presented (Tables VI and VII) indicate that age is a very important factor in determining the consistency of the feces. These two tables, and Fig. 2, show how consistency increases with age through the age range of birth to 100 days. This influence of age is apparent both in the control and the supplemented periods, even though the feeding of pectin tends to mask the influence of age.

Pectin supplementation was the most effective at the early ages, and in general its effect decreased as age increased. Such a pattern of behavior

is to be expected, judging from results with infants in the control of diarrhea (Howard and Tompkins, 1940). Pectin is very efficient in controlling diarrhea of the newborn, but although effective, is not nearly as satisfactory in the treatment of diarrhea in older infants and children.

Possible Mechanism of Action of Pectin

The mechanism by which pectin functions is not thoroughly understood, but its hydrophilic nature probably plays a part in the regulation of laxation. Pectin is digested with difficulty, and is believed not to be acted upon at all by saliva, gastric juice and possibly not by secretions of the small intestine (Kertesz, 1940). Pectin is, however, readily decomposed by bacterial enzymes (Wertch and Ivy, 1940b, c). The digestive tract of the calf is relatively free of the rumen fauna and flora normally found in adult bovines for the first 30 days of life and frequently for as long as 90 days after birth (Pouden and Hibbs, 1947). Pectin ingested before the microorganisms of the rumen become active would pass intact into the lower digestive tract of the neonatal calf, and there exert its hydrophilic effect. As the microorganisms become more active, less undecomposed pectin escapes into the lower tract, and the apparent influence of pectin upon consistency becomes progressivly less.

The list of substances which have proved effective against one form or another of scours and other digestive disturbances is a long one, and is being extended almost every day. Some of these, such as the sulfa drugs, have proved highly efficient in treating such disorders. These remedies almost without exception must be classes as drugs and are limited to the treatment

of the disorder rather than the prevention. A substance which is cheap, effective in controlling scours, and yet safe in the hands of the layman would be of greatest practical value to the dairy farmer. There is also a place in the research field for such a substance; a digestive disturbance may so upset the balance of an experiment as to render the experimental results of little or no value. Such a substance must have a minimum of effects upon the animal other than physical regulation of the alimentary tract.

Pectin appears to have some possibilities as a physical regulator of the digestive tract of calves. It has certain disadvantages in that it does not always have the same effect on the same animal and that different animals respond differently to it. It is therefore, rather difficult to predict just what the exact response to pectin will be. Additional work certainly must be done to determine the effects, if any, of pectin upon digestion and other body processes before it can be accepted as a regulator of the digestive tract. The variability so obvious in this experiment is not too encouraging, and may rule out the use of pectin as a regulator. On the other hand, the difference between control and supplemented periods, particularly at the early ages, indicates that pectin supplementation may be of some value. At any rate, additional work should be done before definite conclusions are drawn.

SUMMARY AND CONCLUSIONS

Pectin in varying amounts was added to the milk of young calves on standard calf rations. The consistency of the feces of these calves was determined when pectin was fed and when the unsupplemented ration was fed.

Pectin was shown to influence the consistency of feces, the effect varying with the animal, the age of animal and the amount of pectin fed.

From these studies the following tentative conclusions are drawn:

1. Small amounts of pectin markedly increase the consistency of the feces of young calves.

2. The effect of pectin decreases as age of calf increases.

3. Small amounts of pectin increase consistency while large amounts decrease consistency.

LITERATURE CITED

Ahmann, C. F., and H. D. Hooker. 1925.
The estimation of pectin and a study of the constitution of pectin. Mo. Agr. Expt. Sta. Res. Bul. 77. 39 p.

Arnold, L. 1939.
The bactericidal action of pectin and metal pectinates. Amer. Jour. Digest. Dis. 6: 104-105.

Baier, W. E. 1945.
How the citrus industry measures it. VI. Chemical analysis of Pectin. Calif. Citrograph 30: 378-386.

Baker, G. L. 1940.
Pectic material as a constituent of nitrogen-free extract. Jour. Assoc. Offic. Agr. Chem. 23: 137-143.

Bennison, E. W., and F. W. Norris. 1939.
The pectic substances of plants. VI. The relation between jelly strength, viscosity and composition of various pectins. Biochem. Jour. 33: 1443-1451.

Block, L. H., A. Tarnowski and B. H. Green. 1939.
Pectin and nickel pectinate in acute and chronic bacillary dysentery. Amer. Jour. Digest. Dis. 6: 96-102.

Bonner, J. 1936.
The chemistry and physiology of the pectins. Bot. Rev. 2: 475-497.

Burroughs, L. F., M. E. Kieser, A. Pollard and J. Steedman. 1944.
The treatment of apple pomace prior to drying for subsequent pectin extraction. Fruit Prod. Jour. 24: 4-6.

Council on Foods. 1937.
The apple in the management of diarrhea in children. Jour. Amer. Med. Assoc. 109: 1636-1637.

Dack, G. M., R. Johnson and L. R. Dragstedt. 1939.
Effect of normal and raw apple diet on the flora of the duodenum, ileum and cecum of monkeys. Jour. Inf. Dis. 64: 52-58.

Fantus, B., and W. Frankl. 1941.
The mode of action of bran. I. Effect of bran upon composition of stools. Jour. Lab. & Clin. Med. 26: 1774-1777.

Fantus, B., and G. Kopstein. 1940.
Does bran produce intestinal obstruction? Amer. Jour. Digest Dis. 7: 60-63.

Frank, E. 1937.
Treatment of diarrhea in childhood with aplona and pectin. Deutsch med. Wochenschr. 63: 1328-1329. (Nutr. Abs. & Rev. 7: 4005).

Gohrbandt, E. 1936.
Effect of pectins on blood clotting. Deutsch med. Wochenschr. 62: 1625-1629. (Nutr. Abs. & Rev. 6: 5041).

Griggs, M. A., and R. Johnstin. 1926.
Preparation and colloidal properties of pectin. Ind. Engr. Chem. 18: 623-625.

Hartman, F. W., V. Schelling, H. N. Harkins and B. Brush. 1941.
Pectin solution as a blood substitute. Annals of Surgery 114: 212-225.

Hirst, E. L., and J. K. N. Jones. 1939.
Pectic substances. III. Composition of apple pectin and the molecular structure of the araban component of apple pectin. Chem. Soc. Jour. (1): 454-460.

Hoppert, C. A. and A. J. Clark. 1942a.
Effect of long-continued consumption of bran by normal men. Jour. Amer. Diet. Assoc. 18: 242-243.

Hoppert, C. A., and A. J. Clark. 1942b.
Bran muffins and normal laxation. Jour. Amer. Diet. Assoc. 18: 524-525.

Hoppert, C. A., and A. J. Clark. 1945.
Digestibility and effect on laxation of crude fiber and cellulose in certain common foods. Jour. Amer. Diet. Assoc. 21: 157-160.

Howard, P. J., and C. A. Tompkins. 1940.
Pectin-agar for diarrhea in infants and the newborn. Jour. Amer. Med. Assoc. 114: 2355-2358.

Hummel, F. C., M. L. Shepherd and I. G. Macy. 1940.
Effect of changes in food intakes upon the lignin, cellulose, and hemicellulose contents of diets. Jour. Amer. Diet. Assoc. 16: 199-207.

Hunt, J. S. 1936.
Observations on the use of raw apple diet and pectinagar mixtures in pediatric diarrheas. Arch. Pediat. 53: 736-739.

Kertesz, Z. L. 1940.
Pectic enzymes. V. The fate of pectins in the animal body. Jour. Nutr. 20: 289-296.

Kertesz, Z. I., and E. L. Green. 1931.
Factors influencing the pectin content of stored apple pomace. New York Agr. Expt. Sta. Tech. Bul. 179. 14 p.

Kutscher, G. W., and A. Blumberg. 1939.
The use of pectin-agar mixes in diarrhea. Amer. Jour. Digest. Dis. 6: 717-720.

Malyet, G. 1936.
Apple diet or pectin treatment of diarrheal diseases. Med. Klin. 32: 1401-1402. (Nutr. Abs. & Rev. 6: 5709).

Manville, I. A., E. M. Bradway and A. S. McMinis. 1936.
Pectin as a detoxication mechanism. Amer. Jour. Digest. Dis. 3: 570-572.

Manville, I. A., E. M. Bradway and A. S. McMinis. 1937.
The use of apple powder in the treatment of diarrheal conditions and its rationale. Can. Med. Assoc. Jour. 36: 252-257.

Manville, I. A., and N. P. Sullivan. 1940a.
The bactericidal action of metallic ions in broth containing dehydrated apple. Amer. Jour. Digest. Dis. 7: 106-110.

Manville, I. A., and N. P. Sullivan. 1940b.
Relationship of the diet to the self-regulatory defense mechanism. III. Organic acids and pectin. Amer. Jour. Digest Dis. 7: 111-114.

Meyers, P. B., and G. L. Baker. 1934.
Fruit jellies. VIII. The role of pectin. 4. The physicochemical properties of pectin. Del. Agr. Expt. Sta. Bul. 187. 39 p.

Ohn, A. 1926.
The viscosity of pectin sols. Ind. Engr. Chem. 18: 1295-1298.

Olsen, A. G. 1940.
Pectin therapy and pectin types. Amer. Jour. Digest. Dis. 7: 515-519.

Pouden, W. D., and J. W. Hibbs. 1947.
Some possible relationships between management, fore-stomach contents and diarrhea in the young dairy calf. Jour. Dairy Sci. 30: 582-583.

Prickett, P. S., and N. J. Miller. 1939.
In vitro effect of pectin and nickel pectin on bacterial growth. Jour. Pediat. 15: 710-721.

Reithel, F. J., and I. A. Manville. 1938.
Advantages of adding apple to milk formulas. Amer. Jour. Dis. Child. 56: 235-238.

Rose, M. S., G. MacLeod, E. M. Vahlteich, E. H. Funnell and C. L. Newton. 1932.
The influence of bran on the alimentary tract. Jour. Amer. Diet. Assoc. 8: 133-156.

Steinhaus, J. E., and C. E. Georgi. 1941.
The effect of pectin, galacturonic acid and alpha methyl galacturonate upon the growth of enterobacteriaceae. Jour. Infect. Dis. 69: 1-6.

Sullivan, M. P., and I. A. Manville. 1938.
The relationship of the diet to the self-regulatory defense mechanism. I. Hydrogen-ion concentration and bacterial flora. Amer. Jour. Digest. Dis. 5: 428-432.

Werch, S. C., A. A. Day, R. W. Jung and A. C. Ivy. 1941.
Role of the intestinal bacteria in the decomposition of pectin. Proc. Soc. Expt. Biol. Med. 46: 569-572.

Werch, S. C., and A. C. Ivy. 1940.
On the fate of ingested pectin. Proc. Soc. Expt. Biol. Med. 44: 366-368.

Werch, S. C., and A.C. Ivy. 1941a.
On the fate of ingested pectin. Amer. Jour. Digest. Dis. 8: 101-105.

Werch, S. C., and A. C. Ivy. 1941b.
A study of the metabolism of ingested pectin. Amer. Jour. Dis. Child. 62: 499-511.

Werch, S. C., R. W. Jung., H. Plenk, A. A. Day and A. C. Ivy. 1942.
Pectin and galacturonic acid and the intestinal pathogens. Amer. Jour. Dis. Child. 63: 839-846.

Williams, R. D., and W. H. Olmstead. 1936.
The effect of cellulose, hemicellulose and lignin on the weight of the stool: a contribution to the study of laxation in man. Jour. Nutr. 11: 433-449.

Winters, M., and C. A. Tompkins. 1936.
A pectin-agar preparation for treatment of diarrhea in infants. Amer. Jour. Dis. Child. 52: 259-265.

Winters, M., C. A. Tompkins and G. W.Crook. 1939.
Pectin-agar diets in the treatment of bacillary dysentery of infants and children. Jour. Pediat. 14: 788-797.

APPENDIX

TABLE IX. CONSISTENCY OF THE FECES OF C651.

Age in Days	Pectin Fed	Consistency
	grams	
33	0	19
34	0	20
35	0	21
36	0	18
37	0	17
38	0	27
45	5	46
46	5	46
47	5	54
48	5	52
49	5	58
50	5	30
51	5	27
52	5	34
53	10	42
54	10	25
55	10	53
56	10	33
57	10	51
58	10	43
59	15	56
60	15	42
61	15	42
62	15	42
63	15	53
64	15	29
65	15	67
67	0	29
68	0	30
69	0	32
70	0	45
71	0	47
72	0	18
73	0	21

TABLE X. CONSISTENCY OF THE FECES OF C652.

Age in Days	Pectin Fed	Consistency
	grams	
26	5	15
27	5	24
28	5	50
29	5	32
30	5	35
31	5	27
32	5	27
33	0	12
34	0	4
35	0	20
36	0	8
37	0	12
38	0	10
39	0	14
40	0	40
61	0	112
62	0	70
63	0	111
64	0	25
65	0	42
66	0	75
67	0	58
68	0	170
69	0	78
70	0	103
71	0	94
72	0	52
73	0	50
74	0	59
77	200	105
78	200	59
79	200	56
80	200	65
81	200	35
82	200	23
83	200	96
84	200	48
85	200	48
86	200	17
87	200	15
88	200	49
89	200	44
90	200	48

TABLE XI. CONSISTENCY OF THE FECES OF C658

Age in Days	Pectin Fed	Consistency
	grams	
3	0	2
4	0	10
5	0	--*
6	0	15
7	0	--*
8	0	--*
9	0	22
10	0	4
12	5	55
13	5	82
14	5	63
15	5	24
16	5	44
17	5	56
18	5	25
19	5	21
20	5	30
21	5	38
22	5	29
23	5	12
24	5	35
25	5	42
27	0	33
28	0	37
29	0	46
30	0	38
31	0	27
32	0	38
34	1	62
35	1	109
36	1	49
37	1	34
38	1	35
39	1	75
42	15	28
43	15	68
44	15	--*
45	15	74

*Sample contaminated.

Continued on next page

TABLE XI. CONTINUED

Age in Days	Pectin Fed	Consistency
	grams	
46	15	25
47	15	49
48	15	152
49	15	181
50	15	111
51	15	65
52	15	270
53	15	105
54	15	168
58	0	25
59	0	10
60	0	41
61	0	21
62	0	28
63	0	10
64	0	8
65	0	15
66	0	20
67	0	10
68	0	22
69	0	48
70	0	61
71	0	75
72	0	38

TABLE XII. CONSISTENCY OF THE FECES OF A70.

Age in Days	Pectin Fed	Consistency
	grams	
81	0	50
82	0	31
83	0	91
84	0	115
85	0	136
86	0	32
87	0	105
89	45	52
90	45	58
91	45	44
92	45	57
93	45	81
94	45	65
95	45	76
96	45	76
97	45	112
98	45	125
99	45	111
100	45	115
101	45	41
102	45	70
103	45	118
104	45	112

CARBOHYDRATE DIGESTION IN THE YOUNG CALF

II. Corn Starch, Dextrin and Corn Sugar as Carbohydrate Sources in Calf Synthetic Milks.

INTRODUCTION

Research in the fields of vitamin and mineral metabolism has advanced at a rapid rate in recent years. Regardless of the fine contribution in these fields, there is much fundamental work that remains to be done. So little is known, for example, of the nature of the carbohydrate or of the protein requirements of the young calf.

The purpose of this investigation is to study the relative values of corn starch, dextrin and corn sugar as the carbohydrate sources for young calves.

REVIEW OF LITERATURE

Purified Diets in Animal Nutrition

Rats and Mice

Gavin and McHenry (1940), in their studies of B vitemins and fat metabolism, placed rats on a diet of vitamin-free casein 10 percent, sucrose 54 percent, triolein 30 percent, salt mixture 4 percent and agar 2 percent. Richardson and associates (1941) determined the number of vitamins required by the rat using a ration of varying amounts of casein, sucrose, lard, salts, cellulose, and vitamin supplements. Eight vitamins were found to be required, including vitamin A, vitamin D, alpha tocopherol, thiamin, riboflavin, pyridoxine, pantothenic acid and choline. The diet used by Henderson and coworkers (1942) in studying pantothenic acid in the nutrition of the rat consisted of sucrose 73 percent, purified casein 18 percent, salts 4 percent, corn oil 5 percent, and thiamin 2 mg., pyridoxine 2 mg., riboflavin 3 mg., nicotinic acid 2.5 mg. and choline 1 gm. per kilogram. The basal ration used by Sure (1943) follows; blood fibrin 25 percent, butterfat 10 percent, dextrose 56.95 percent, salts 5 percent, wheat germ oil 3 percent and nicotinic acid 0.05 percent. Various modifications of this diet were used in studying p-aminobenzoic acid and inositol in lactation and growth.

West et al. (1943) introduced sulfapyridine into a purified diet and thus produced pantothenic acid deficiency. The basal diet was composed of casein 6 percent, sucrose 15 percent, corn starch 50 percent, hydrogenated

cottonseed oil 24 percent, salt mixture 4 percent and cod liver oil one percent. The B vitamins were supplied in the form of brewer's yeast.

Succinylsulfathiazole was added to synthetic diets by Wright and Welch (1944) in the production of various vitamin deficiencies and in studying the hepatic storage of vitamins. The basal diet consisted of casein 18 percent, fat 10 percent, corn oil 2 percent, sucrose 59.9 percent, salts 4 percent and cellu flour 6.1 percent, plus various combinations of vitamins. Growth of rats on this diet was excellent.

Ershoff (1944) reported the use of a purified diet of sucrose 73.2 percent, vitamin test casein 22 percent, cystine 0.3 percent and salt mixture 4.5 percent. The vitamins added included thiamin, riboflavin, pyridoxine, pantothenic acid, niacin, p-aminobenzoic acid, inositol, choline, 2-methyl-naptho-quinone, alpha tocopherol, vitamin A and vitamin D. On this ration Ershoff obtained normal growth and reproduction but a failure of lactation.

A number of strains of mice were reared through several generations on highly purified diets by Ceresedo and Vinson (1944). They reported excellent growth of mice on their purified diet; in fact the growth was superior to that of mice on the stock diet. Reproduction and lactation on the purified diet was decidedly inferior to that on the stock diet. The purified diet consisted of purified casein 30 percent, salts 5 percent, alpha cellulose 2 percent, lard 5 percent, crisco 10 percent and sucrose 48 percent. Vitamins added included A and D concentrate, alpha tocopherol, choline, pantothenic acid, pyridoxine, riboflavin and thiamin.

Scott (1946), in testing the ability of the rat to choose its diet,

allowed test animals their choice of sucrose, casein, hydrogenated fat and salts. Of the 87 rats used in the test, 34 failed to grow. These 34 showed a dislike for casein and on the average ate less than 0.1 gram of casein daily. On the other hand, rats that liked casein ate an average of 3 grams per day and grew well. Those rats which liked casein grew significantly better when placed on a standard diet than did those which showed a dislike for casein. Most of the rats had little appetite for sucrose although a few ate rather large amounts. The appetite for hydrogenated fat varied less than did the appetite for the other components. Those rats which grew ate about 0.25 grams of salts daily while those that did not grow ate about 0.05 gram daily.

Geyer and associates (1947) devised two types of cages, one circular and one tubular, for the purpose of preventing coprophagy in rats on experiment. Using a ration of sucrose 48 percent, casein (hot alcohol extracted) 20 percent, salts 4 percent and corn oil or butterfat 28 percent, plus vitamin supplements, it was found that weanling rats will grow even when closely confined. Rats kept in circular cages grew poorly if liver concentrate was not fed; if liver concentrate was fed the growth was almost as satisfactory as that obtained in ordinary cages. It was thought that the circular cage did not completely prevent coprophagy, since the rats could travel over the same floor area repeatedly. Those rats kept in tubular cages grew poorly without liver concentrate, and when the supplement was provided the growth was inferior to that obtained in circular cages or in ordinary cages. All rats in tube cages tended to lick their cages and anything else within reach, possibly suggesting some deficiency.

Nelson and Evans (1947) conducted extensive studies of the effect of purified diets on growth, reproduction and lactation in the rat. Varying proportions of casein, sucrose, hydrogenated vegetable oil and salts were used, and the vitamins supplied included A and D, alpha tocopherol, thiamin, pyridoxine, riboflavin, p-aminobenzoic acid, niacin, pantothenic acid, inositol and choline. These workers obtained excellent growth on the purified diets, and reproduction which approached the normal. Lactation was inhibited, as the growth of young while suckling was subnormal. However, the percentage of young weaned was equivalent to that of rats on the stock ration.

Guinea Pigs

Semi-purified diets were used as early as 1931 in studying the nutrition of the guinea pig (Goettsch and Pappenheimer, 1931). A diet of rolled oats 355 parts, wheat bran 180 parts, casein 75 parts, lard 80 parts, cod liver oil 10 parts, sodium chloride 10 parts, calcium carbonate 15 parts and skimmed milk powder 275 parts when fed to guinea pigs led to a progressive, highly selective and ultimately fatal dystrophy of the voluntary muscles. The diet was believed to be complete in all known requirements except for vitamin E and the addition of this vitamin did not lower the incidence of the disease. The muscle lesions produced were believed not due to inanition, infection or scurvy. Although the diet lacked some unknown essential, at least one guinea pig survived for as long as 165 days on the experiment.

Hogan and Ritchie (1934) developed a simplified diet which gave good results with rabbits but as unsatisfactory for guinea pigs. Modifications of the diet were made and fair results with guinea pigs were obtained on a

diet of casein 20 percent, dextrinized corn starch 28 percent, cellophane 14 percent, milk fat 13 percent, wheat germ oil one percent, cod liver oil 2 percent, salts 5 percent, yeast 15 percent and tikitiki 2 percent.

Madsen (1936) used a semi-purified diet composed of purified cellulose 20 percent, casein 15 percent, sucrose 10 percent, starch 43 percent (or less), lard or cottonseed oil 3 percent (or more), yeast 5 percent and mineral mixture 4 percent, plus vitamins A and D. Guinea pigs on this diet developed a skeletal muscle dystrophy, particularly when cod liver oil was used as a source of vitamins. Animals on cottonseed oil as a source of fat were less susceptible to the dystrophy than were those on lard when an A and D concentrate was included in the fat. No such protection was observed when cod liver oil was included as the source of vitamins.

Cannon and Emerson (1939) described a purified diet that was readily eaten by the guinea pig. This diet consisted of casein 24 percent, sucrose 50 percent, lard 14 percent, salt mixture 4 percent, agar 4 percent, cod liver oil 2 percent, wheat germ oil 2 percent, and a yeast extract equivalent to 10 percent brewers' yeast. This diet and various modifications of it, including additions of orange juice and sources of other vitamins, failed to support growth or maintenance of guinea pigs unless a factor found in lettuce and in grass was included.

Hogan and Hamilton (1942) reported that guinea pigs grow at a normal rate on purified diets if dried yeast or a water extract of dried liver is used as a source of water-soluble vitamins. Subnormal growth and high mortality resulted when water-soluble vitamins were supplied as pure compounds. Numerous diets were used, the basal usually consisting of combinations of casein, dextrin, cellulose, lard or soybean oil and salts. Various

crude vitamins carriers or combinations of all available vitamins in the pure form were used. The addition of vitamin K to the diets enabled the females to rear litters but failed to sustain growth.

Woolley (1942) found that at least three unknown dietary factors were required for the growth and survival of guinea pigs on highly purified diets. The first of these, which he termed GPF-1, was found to be soluble in 50 percent alcohol, and had been purified to such a point that 5 milligrams per day produced good growth. A second, GPF-2, was insoluble in 50 percent alcohol and could not be replaced by any of the several factors recently found essential for chicks. Woolley found that still a third factor (GPF-3) was essential for growth and life of guinea pigs for more than just a few weeks. Woolley's ration included casein, sucrose, inorganic salts, corn oil, vitamins A. D, K and E, thiamin, riboflavin, pyridoxine, nicotinic acid, pantothenic acid, choline, inositol and ascorbic acid.

Substantiation of the theory of three dietary factors required by the guinea pig in addition to those required by the rat was made by Sober and associates (1942) at the Wisconsin station. One of these factors they found to be supplied by 16 percent of grass but not supplied in adequate amounts by 16 percent of yeast and 20 milliliters of winter milk. A second factor was provided by 16 percent of yeast but not provided in adequate amounts by 16 percent of grass and 20 milliliters of winter milk. The third factor was provided by 20 milliliters of winter milk but was not supplied adequately by 16 percent of grass and 16 percent of yeast. Constituents of the basal ration included sucrose, casein, salt mixture, corn oil, and supplements of

halibut liver oil and the known crystalline vitamins.

Mannering and coworkers (1943) offered additional evidence that more than one unknown dietary factor is required by the guinea pig with a basal ration of sucrose, casein, salts and corn oil supplemented with thiamin, riboflavin, pyridoxine, pantothenic acid, nicotinic acid, choline, ascorbic acid, halibut liver oil and alpha tocopherol. The factor under consideration was not the same as those factors present in linseed oil meal, and was found in solubilized liver powder, grass juice powder and possibly other materials. These authors assumed the factor to be identical with Woolley's GPF-3.

Kuiken and associates (1944) concluded that at least two unknown dietary factors were required to complete the diet used in their work. One of these essentials was present in commercial casein, for when vitamin-free casein was used no growth response was obtained by the addition of brewers' yeast, liver extract, rice polish concentrate, skim milk powder or dried grass. When commercial casein was used as the protein source, the addition of small amounts of any one of these supplements produced a marked improvement in survival time and in growth. The authors were not able to determine what were the limiting factors when vitamin-free casein was used. Evidence available, however, indicated that neither biotin nor folic acid was the factor concerned.

Some clarification of the unidentified factors in guinea pig nutrition was provided by Woolley and Sprince (1945), in that GPF-1 was identified with if not as folic acid. GPF-2 was found to be replaceable by a mixture of cellulose and casein, or a mixture of cellulose, arginine, cystine and

glycine. These authors demonstrated that GPF-3 resembled strepogenin and was characterized by being insoluble in alcohol but soluble in alcohol and hydrochloric acid. Furthermore, it was not precipitated by lead acetate and was not readily adsorbed by norit. In tests with GPF-3-deficient animals, some improvement in survival time was observed upon the addition of biotin and p-aminobenzoic acid.

Dogs

Morgan and Simms (1940) studied the anti-grey hair vitamin and its requirement by dogs, using a basal diet of extracted casein 45.8 percent, sucrose 20.9 percent, corn starch 19.4 percent, crisco 10 percent, salts 2.4 percent, calcium carbonate 1.5 percent, and supplements of carotene-reinforced cod liver oil, thiamin chloride, riboflavin and wheat germ autolysate. Growth was poor and it was not determined whether or not the anti-grey hair vitamin was the only factor involved, as a yeast filtrate preparation was used to supply the vitamin.

A synthetic diet of casein, sucrose, crisco, bone ash and salt mixture, supplemented with thiamin chloride, riboflavin, nicotinic acid and crystalline B_6, was fed to adult dogs by Fouts, Helmer and Lepkovsky (1940). This diet, apparently deficient only in the factors contained in a purified liver extract, caused an initial gain in weight but a subsequent loss in weight. The loss in weight was associated with a progressive decrease in appetite as the experiment progressed. Diarrhea, vomiting and ulcers of the skin were characteristic of the deficiency.

A study of the vitamin B complex in the nutrition of dogs was made by Schaefer, McKibbon and Elvehjem (1942a) using a basal ration of sucrose

66 percent, acid-washed casein 19 percent, cottonseed oil 8 percent, cod liver oil 3 percent and salt mixture 4 percent. This ration was supplemented with thiamin chloride, riboflavin, nicotinic acid, pyridoxine hydrochloride, calcium pantothenate and choline. Growth of dogs on such a diet was good, but could be increased by the addition of liver extract. If the casein was further purified by alcohol extraction growth usually ceased and anorexia and loss of weight followed in from one to three months. This condition could be corrected by the addition of liver extract but not by mixtures of inositol, p-aminobenzoic acid and glutamine. These authors in a later report (Schaefer, McKibbin and Elvehjem, 1942b) tentatively fixed the calcium pantothenate requirement for young growing puppies at 100 micrograms per kilogram of body weight per day.

Lambooy and Nasset (1943) fed young dogs a purified casein-sucrose-hydrogenated cottonseed oil diet supplemented by eight of the B complex vitamins and found that dry whole yeast or concentrates of either yeast or liver were necessary to prevent the development of dermatitis and loss of hair. Without the crude vitamin supplements the skin symptoms appeared in from 75 to 125 days and death resulted in from 100 to 150 days. In this work the casein was extracted by acetic acid and ethanol until the fluoresence due to riboflavin disappeared when the filtrates were viewed in ultra-violet light, and the sucrose was extracted twice with ethanol.

Frost and Dann (1944) studied greying of hair in dogs on purified diets and substantiated the earlier reports that unknown factors in yeast and liver play an essential role in the pigmentation of hair. The diet used was of the casein-sucrose-corn oil type, supplemented by the available

crystalline vitamins, and produced fair to good growth of the dogs.

Ruegamer and associates (1945) reported good growth and maintenance of health of dogs on a basal diet of sucrose 66 percent, casein 19 percent, cottonseed oil 11 percent and salts 4 percent supplemented with thiamin, riboflavin, pyridoxine, pantothenic acid, nicotinic acid and choline. They found none of the dermatitis reported by other workers on a high fat ration, and ascribed their success to the high level of choline feeding used as contrasted to the relatively low levels employed by other workers. Ruegamer and associates found that the use of a highly purified alcohol extracted casein resulted in achromotrichia and a plateauing of hemoglobin levels at 11 to 14 grams percent. Biotin was necessary for the production of hemoglobin levels greater than 14 grams percent.

Seeler and Silber (1945) maintained adult dogs on a virtually vitamin B complex-free basal ration, supplemented only with thiamin, riboflavin, nicotinic acid and pyridoxine, for four and one-half years in apparent good health. As a result of this experiment the authors concluded that the requirements of the adult dog for pantothenic acid was very small and by no means critical. Folic acid, biotin and inositol were omitted from the ration without obvious effects on the adult dog.

Swine

Wintrobe and associates (1938) placed pigs 10 to 23 days of age on an artificial diet of casein, sucrose, butter or lard, cod liver oil, salt mixture, vitamin C and yeast. As the experiment progressed, thiamin and riboflavin gradually replaced the yeast in the diet. Following this change the

rate of growth decreased and the general condition of the animals became impaired. Histological examination disclosed degeneration of peripheral nerves and the posterior spinal cord. A subsequent report (Wintrobe et al., 1939) dealt with blood changes on this ration and the effects of various vitamin supplements. Anemia was prevented by yeast but not by thiamin, niacin, riboflavin, or combinations of these. Yeast therapy resulted in partial relief of anemia while a purified liver extract had no effect.

Hughes (1939, 1940a, 1940b) used various semi-purified and purified diets in studying the role and requirement of thiamin and riboflavin in the nutrition of the pig. A diet consisting of cane sugar, purified casein, lard, rice bran filtrate, salt mixture and cod liver oil was used extensively. Riboflavin deficiency resulted in slow growth and a crippled condition, while thiamin deficiency resulted in slow growth and a slight weakness in the legs. The minimum requirement of the young growing pig for riboflavin was established at between one and three mg. per 100 pounds; that for thiamin at about one mg. per 100 pounds of pig per day. Hughes and Ittner (1942) studied pantothenic acid requirements using a ration of sugar, casein, salt mixture and vitamin supplements. The requirement of the growing pig was found to be between 7.8 and 11.8 mg. per 100 pounds of pig daily.

Wintrobe and associates (1942) fed a ration of crude casein, lard, sucrose, salt mixture and vitamins to pigs and obtained normal growth. When pyridoxine or calcium pantothenate were omitted, sensory neuron degeneration occurred. Omission of thiamin failed to produce lesions of the nervous system.

An investigation of thiamin deficiency in pigs receiving various levels of fat in the diet was conducted by Ellis and Madsen (1944). Pigs on low fat

(2 percent) developed thiamin deficiency symptoms in an average of 25 days, those on medium fat (11 percent) in 28 days, and those on high fat (28 percent) in 33 days. Thiamin therapy in deficiency cases resulted in prompt recovery.

McRoberts and Hogan (1944) tested the efficiency of synthetic vitamins and crude vitamin carriers in supporting pigs on synthetic diets. Pigs were allowed to reamin with the sow the first two days, then were transferred to simplified diets of casein, sucrose, corn starch, lard, salts and various vitamins. In general, pigs seldom survived when only the synthetic vitamins were supplied. Crude vitamin carriers, such as water extracts of liver or yeast, appeared to meet the requirements of the pig.

The work of Wintrobe et al. (1945), in which an attempt was made to produce nicotinic acid deficiency at high and low levels of protein feeding, resulted in the conclusion that there is a close nutritional relationship between nicotinic acid and protein. These workers were unable to produce nicotinic acid deficiency when the ration included 26 percent casein, but deficiency symptoms resulted when the ration contained only 10 percent casein. On the other hand, Powick and associates (1947) reported nicotinic acid deficiency in growing pigs on purified rations containing 25 percent casein.

Lindley and Cunha (1946) produced biotin deficiency in pigs on a diet of casein, sucrose, lard, salts and vitamins by the inclusion of desiccated egg white or by adding sulfathalidine to the diet. The syndrome developed included spasticity of the hing legs, loss of hair, dermatosis of the skin, exudate around eyes and cracks in the feet. The addition of inositol to

the deficiency-producing diet alleviated to a large extent the deficiency symptoms.

Russell, Teeri and Unna (1948), after obtaining normal growth of pigs on a purified ration, attempted to carry some of the animals through a reproductive cycle. Hogs maintained on the purified diet failed to reproduce even though dried liver was added to the diet.

Calves

As compared to the other species discussed, synthetic rations in the study of bovine nutrition have virtually been ignored.

Johnson, Loosli and Maynard (1940) used a mixture of casein, lactalbumin, sugar, butter or lard, minerals and water as a substitute for milk in studying the growth requirement of calves. The subnormal growth rates obtained were attributed to poor feed consumption and periodic digestive upsets. Thiamin and riboflavin did not appear to be essential to the calf, and no improvement in well being was noted upon the inclusion of the grass juice factor in the diet.

Illinois workers (Wiese, Johnson, Mitchell and Nevens, 1947a) and (Johnson, Mitchell, Hamilton and Nevens, 1947b) developed a synthetic ration which apparently supported normal growth for at least 12 weeks. The ration consisted of casein, cerelose, lard, salts and vitamins, and was prepared by dissolving the casein, cerelose and salts and homogenizing in the lard.

Carbohydrate Digestion in the Calf

The digestion of carbohydrates by calves has not been studied extensively, and little evidence is actually available as to the calf's ability to

digest various carbohydrates. Shaw, Woodward and Norton (1918) investigated the ability of the calf to digest starch. Two calves were used, and starch was added to the milk which the calves received. All feces were collected, and the digestion of the starch determined. Typical results are illustrated by the data on calf 1:

Age of Calf	Starch Fed	Starch in Feces	Starch Digested	
days	gm	gm	gm	%
4	184.9	144.2	40.7	22.02
12	184.9	101.5	83.4	45.11
20	184.9	67.8	117.1	63.34
30	184.9	15.2	169.7	91.79
39	184.9	2.2	182.7	98.81

It was found that calves four to seven days old were able to digest about one-fifth of the quantity of starch consumed; at two weeks of age the amount had doubled, and at three to four weeks had tripled. At one month of age the percentage of starch digested was over 90.

Mattill and Hawk (1912) found that the ingestion of large amounts of water with meals caused a decrease in the excretion of carbohydrate and a better utilization of food.

Digestion of fiber was studied by Sheehy (1935). It was found that the fiber of bran was superior to that of oats, due partially to functional activity in the large intestine. Mucilaginous material such as that present in linseed cake, may have special merits in livestock feeding. Sheehy divided bulky feeds into two classes, those to which straw and the dried grains belong and those including the succulent and lubricative foods such

as roots, green food and steeped bran. Foods of the latter class exert their mechanical effect in the stomach and the intestine while those of the former class usually affect only the stomach. These investigations were conducted on swine and may not apply completely to cattle.

Nikitin (1939), in studying rumen digestion of calves, reported that the mono- and disaccharides fermented more rapidly than starch and much more rapidly than cellulose. The feeding of "yeast fodder" depressed fermentation while the addition of casein, peptone and glycine increased the decomposition of carbohydrates. The addition of glucose always resulted in increased fermentation.

The value of corn sugar in the grain mixture of calves was investigated by Ward, Cannon and Espe (1937). Three groups were used: one on a standard grain mixture, the second on a ration in which 10 percent of the grain was replaced by corn sugar, and the third on a ration in which 20 percent of the grain was replaced by sugar. Those calves receiving corn sugar ate their grain mixture at an earlier age and consumed slightly more in the early stages of the experiment than did the control calves. At six months of age the calves fed 20 percent sugar averaged 24 pounds heavier than the control calves, but the difference was found to be not significant.

Espe and Cannon (1940) found that high fiber content, such as used in milk substitutes, tended to cause young calves to scour, but that coursely cut roughage does not have such an effect. This they attributed to the fact that such roughage is held in the rumen until partially digested. The tendency to scour upon ingestion of high fiber feed disappeared as soon as the first three compartments of the stomach became large enough to permit

storage so that bacteria and rumen fluid may act on the feed.

Mitchell and Hamilton (1940) reported that when glucose was given as a supplement to a basal ration, the glucose replaced insoluble carbohydrates as a substrate for bacterial fermentation in the rumen and the insoluble carboydrates were left undigested.

Phillipson and McAnally (1942) and McAnally (1943) studied the digestion of carbohydrates in the rumen of sheep. It was found that glucose, fructose and cane sugar ferment quite rapidly and pass through the stage of lactic acid to the lower volatile fatty acids. Lactose, galactose and maltose fermented less rapidly and without an accumulation of lactic acid. Starch and cellulose were found to ferment slowly with the result that the production of volatile acids is greatly prolonged.

Rojas, Schweigert and Rupel (1948) determined the galactose excreted by calves on whole milk and skim milk, and when lactose was added to the milk. When the ration consisted of whole or skim milk, only a small percentage (usually less than two percent) of the galactose ingested was excreted in the urine. However, when lactose was added to the milk the percentage of galactose excreted in the urine increased rapidly, reaching 16 percent in some cases. Diarrhea resulted shortly after the ingestion of the lactose-enriched milk.

Protein Digestion in the Calf

General

Protein digestion in the calf is another neglected phase of nutritional

research. In the development of a successful calf starter one of the most basic points for consideration is whether or not the calf requires animal protein. To date the problem has not been answered; in fact few attempts have been made to determine whether or not the calf can survive on vegetable protein.

Shoptaw (1936) used soybean flour as a substitute for milk in feeding calves by mixing one part of the flour with nine parts of warm water. Calves averaging 25 days of age were used in the 70 day feeding trial in comparing the soybean milk with cow's milk. The results are summarized as follows:

	Control	Experimental
Average daily gain	1.24 pounds	0.90 pounds
Average increase in height at withers	4.94 inches	4.37 inches
Condition	Normal thrifty	Rough hair coat, diarrhea.

In this trial grain and hay were allowed ad libitum. The soybean fed calves consumed an average of 156 pounds of grain and 43.1 pounds of hay, as compared to 110 and 51.8, respectively, for the control calves.

Shoptaw, Espe and Cannon (1937) studied the gastric digestion of soybean flour by calves. Pavlov pouches were used to collect gastric juice, and the free and total acidity of the gastric contents were determined. The flow of gastric juice was about the same on the soybean flour as it was on milk. Feces from the soybean fed calves were foul, indicating to the authors that the food was only partially digested, and these calves were observed to scour quite easily.

Daniel and Harvey (1947) found that the partial substitution of whey

proteins for casein resulted in reduced growth of rats. Feeding whey which had been dialyzed to remove minerals resulted in a much more favorable growth response. Ashing of the dialysate and reintroduction of these minerals into the dialyzed whey again impaired the growth. This would indicate that inorganic constituents rather than an organic compound affect detrimentally the nutritional value of whey.

Colostrum and Protein

Howe (1921) reported that euglobulin and pseudoglobulin were not present in the blood of the newborn calf until after colostrum had been ingested. If colostrum was withheld, some time was required before these globulins formed in the blood. Howe (1924) obtained both euglobulin and pseudoglobulin in the blood of calves when serum was fed in place of colostrum, but was unable to obtain them when milk was fed without colostrum or serum.

Smith and Little (1922a) attempted to raise calves without colostrum but lost nien out of the 12 calves that did not get colostrum. Ten other calves were given colostrum and all survived. Colostrum was found to be protective against miscellaneous bacteria which are harmless once the protective functions of the calf have begun to operate. The theory of a function inherent in colostrum which controls development or growth was discounted, since calves not receiving colostrum did as well as others once the infection was overcome. These authors (1922b, 1923) used cow serum as a substitute for colostrum in feeding newborn calves. When injected shortly after birth only two out of the five calves were saved. When serum was added to the milk of the first two meals three out of five survived. All of five calves treated

by a combination of the two methods survived and continued as normal calves. Smith and Little (1924) were unable to find protein in the urine of fetuses or of unfed calves. Proteinuria was almost universal in colostrum-fed calves, but the condition disappeared after the third day of life. The replacement of colostrum by serum produced the same effect to a lesser extent.

Smith (1925) reported that a focal interstitial nephritis (white spotted kidney), associated with a virulent type of Bacillus coli, could be produced by withholding colostrum altogether or by postponing the feeding of colostrum for 24 to 36 hours. The delay of the first feeding did not produce the condition unless the virulent strain of Bacillus coli was present in the herd.

Wise, Petersen and Gullickson (1940) supplemented milk-fed calves with casein with no adverse effects on growth or general appearance. The casein-fed group showed digestive disturbances more frequently than did other calves, and the feces produced were pasty in consistency and putrid of odor.

Fat Digestion in the Calf

Leach and Golding (1931) conducted two trials in which pilchard oil was substituted for butterfat in milk used for calf feeding. In the first trial three Jersey calves were started on the experiment at 15, 15 and 6 days of age. Eight days were used to make the change from whole milk to skim milk with 3.5 percent pilchard oil homogenized into it. These calves gained weight and manifested good appetites for the first week but after that developed scours and lost weight. On the 12th day of the experiment the youngest calf died; a second died on the 13th day and the third was in a moribund condition

and was killed on the 14th day. Upon post mortem examination severe inflamation was observed in the regions of the heart, lungs and intestines. In the second trial reconstituted skim milk powder, water and pilchard oil were emulsified and fed to two Holsteins, both 16 days of age, and one Jersey, 22 days of age at the start of the trial. One Holstein died on the 15th day of the experiment, the Jersey died on the 18th day and the remaining Holstein was killed on the 20th day. Rats after being on the same rations for 50 days were in perfect health and made weight gains similar to those of rats on butterfat as the fat source.

Espe and Cannon (1935) considered the physiological effect of fat on the rate of evacuation of the stomach. Their results are summarized in the following table:

Total Trials	Kind of Treatment	Total Time for Liquefaction of Curd in the Stomach
26	Skim milk	17 hrs. 25 min.
34	3 percent milk	14 hrs. 34 min.
19	6 percent milk	12 hrs. 56 min.

Curd from milk containing up to 6 percent fat tended to leave the stomach more rapidly than skimmed milk due to the difference in the texture of the curd formed. Milk containing as much as six percent fat did not inhibit gastric secretion or motility. It was concluded that fat, in addition to being a valuable source of food, appears to aid digestion when incorporated in the milk in limited amounts.

Davis and Maynard (1937) tested cod liver oil tolerance in calves

from birth to six or nine months of age on a ration of skim milk, hay and grain and supplemented with various levels of cod liver oil. The maximum dosage of cod liver oil was 0.7 grams per kilo of body weight per day. There was no evidence of adverse effect of the oil on growth or physical condition. Autopsy revealed no gross changes, but histologically slight dystrophic changes were observed in the muscles of some animals on the higher levels of feeding. The lesions, however, were of a very minor character and the authors suggest that cod liver oil may be fed in amounts sufficient to provide vitamin D without injury to calves.

Gullickson and Fitch (1944) administered 25 to 35 cc of cod liver oil daily to calves as a vitamin A supplement. The experiment involved 72 calves of the Guernsey, Holstein and Jersey breeds. These investigators reported less digestive trouble in the supplemented group than in the controls. Over the six month period there was no difference in rate of gain of weight of the Holsteins, but the Guernseys and Jerseys averaged 22.5 and 17.5 pounds, respectively, more in the cod liver oil group than they did in the control group.

The nutritive value of various fats and oils was compared by Schantz, Elvehjem and Hart (1942). Weanling rats were placed on butterfat, corn oil, cocoanut oil, cottonseed oil and soybean oil, each of which had been homogenized into skim milk to contain four per cent fat. Rats on butterfat showed better gains the first three weeks but subsequently there was little difference between groups. These results, however, have not been substantiated in calf work, as may be shown by the work of Gullickson, Fountaine and Fitch (1942). The Minnesota workers compared butterfat, lard, tallow,

corn oil, cottonseed oil and soybean oil for calves. Each of the fats was added to skim milk to form 3.5 percent fat, and the mixture homogenized three times at 3000 pounds pressure. A concentrate mixture which was low in fat was provided. The calves on corn oil, cottonseed oil and soybean oil did very poorly and had an unthrifty, listless and emaciated appearance. Several of them died and others were saved only by transferring to whole milk. Calves on butterfat gained more and looked better than any of the other groups. The lard, tallow and low fat (skim milk only) groups gained almost as rapidly as the butterfat group but lacked the bloom of the latter.

Mineral Metabolism in the Calf

An extensive survey of all the work in mineral metabolism of calves is beyond the scope of this review. A multitude of reports have accumulated on some of the minerals while the role of others in calf nutrition has not been considered. Dairy cattle have been shown to require calcium, phosphorus, magnesium, potassium, sulfur, sodium, chlorine, iodine, maganese, iron, copper and cobalt. Other minerals may be required.

Extensive studies of the calcium and phosphorus requirements of dairy cattle were made by Reed and Huffman (1930) and Huffman and associates (1933). These workers found that from six to 12 grams of calcium and from 10 to 21 grams of phosphorus daily would support the growth of calves. Lindsey and associates (1931) listed the calcium requirements for growth at more than 0.43 percent of the ration, while Henderson and Weakley (1930) stated that 0.25 to 0.28 percent of the ration was adequate. Mitchell (1947) reported the

calcium and phosphorus requirements of growing heifers, expressed as percentage of the dry ration, as follows:

Body Weight lbs.	Age Mos.	Calcium Required pct.	Phosphorus Required pct.	Ca:P Ratio
150	2.2	0.85	0.52	1.6
200	3.0	0.77	0.48	1.6
400	6.6	0.42	0.28	1.5
600	10.6	0.32	0.22	1.4
800	15.4	0.24	0.17	1.4
1000	21.3	0.18	0.14	1.3
1200	28.9	0.18	0.13	1.4

The requirements of calves for magnesium, according to Huffman and associates (1941), is 0.6 milligrams of magnesium daily per 100 pounds body weight when natural feeds are fed.

Sheehy and Senior (1936) stated that a deficiency of sodium chloride was reflected in unthriftiness, lack of bloom of coat and a low retention of calcium and phosphorus, but these authors did not establish the requirements for calves. Wise et al. (1939) remedied the symptoms developed in calves restricted to whole milk rations by supplementing the diet with ferric chloride, cupric sulphate, magnesium carbonate and cod liver oil. Apparently all of these supplements were required for correction of the symptoms.

Mitchell (1947) estimated the average requirements of the growing calf as follows:

Calcium	0.27	percent	of	dry	ration
Phosphorus	0.19	"	"	"	"
Magnesium	0.07	"	"	"	"
Cobalt	0.07	p.p.m.	"	"	"
Copper	3.00	"	"	"	"
Iodine	0.09	"	"	"	"

Other minerals are known to be essential to the calf, but the levels have not been established.

Vitamin Requirements of the Calf

Vitamin A

Jones, Eckles and Palmer (1926) reported the symptoms of vitamin A deficiency in calves as being a failure to grow, xerophthalmia, respiratory trouble and diarrhea. They were able to correct these symptoms by introducing cod liver oil. Less than one percent cod liver oil in a ration otherwise free of vitamin A was sufficient for normal growth. Moore and Hallman (1936) used a ration of skim milk, corn starch, bran, yeast and mineral for the production of vitamin A deficiency, and found that calves this ration develop a condition of white spotted kidneys similar to that which develops when colostrum is withheld from the calf.

In addition to previously reported symptoms, Flora, Ward and Bechdel (1939) found blindness and edema associated with avitaminosis A. The deficiency was corrected by commercial carotene consentrate, fresh carrots or alfalfa hay. Moore (1945) reported that the blood plasma vitamin A of calves up to four months of age was low when compared to the levels found in calves one year of age, and he suggested that vitamin A supplementation

might be helpful when calves are hard to raise.

Lewis and Wilson (1946), using levels of 32 to 1024 U.S.P. units per day, found that 32 U.S.P. units per kilogram per day provided the minimum requirements of the calf. Liver storage was proportional to the dosage of vitamin A; concentration of vitamin A in the blood was proportional to intake up to the maximum level at an intake of 512 units per kilogram daily. Maximum growth was obtained on an intake of 64 U.S.P. units per kilogram of body weight daily.

A study of the use of supplementary vitamins for calves was conducted by Hibbs and Krauss (1947), and it was found that daily administration of 10,000 U.S.P. units of vitamin A increased plasma and liver storage levels, but had no effect on the incidence or severity of scours. Vitamin A may help overcome a deficiency resulting from inadequate feeding of colostrum and whole milk.

Vitamin D

A deficiency of vitamin D in calves under farm conditions is not a common occurence, as calves ordinarily are exposed to sufficient sunlight to provide for their needs of this vitamin. The deficiency, however, is ages old, and was produced experimentally as early as 1926 by Reed and Huffman (1926) at the Michigan Experiment Station. Huffman (1931) showed that cod liver oil, sunshine or sun-cured timothy hay counteracted deficiencies in rations that would otherwise result in rickets. This was substantiated by Rupel, Bohstedt and Hart (1933), who demonstrated that cod liver oil or sunshine were not only preventative, but would cure the

rachitic condition in calves. Ultraviolet light was shown by Bechdel, Landsburg and Hill (1933) to have antirachitic effects when applied to the ration or to the calf.

Bechtel and associates (1936) described the symptoms of vitamin D deficiency in calves. The first noticeable symptom was a decrease in the concentration of calcium and/or inorganic phosphorus of the blood plasma. Following this there was anorexia, cessation of or a decrease in the rate of growth, stiffness and bowing of the forelegs and a reduction of the mineral content of the moisture-free, fat-free rib. Duncan and Huffman (1936) alleviated the decrease in concentrations of plasma calcium and inorganic phosphorus by exposing rachitic calves to sunshine. Solar irradiation was shown to enable the calf to utilize more effectively the calcium and phosphorus present in the ration. The complexity of the interrelationships involved in vitamin D metabolism were further demonstrated by the report of Huffman and Duncan (1935) that magnesium added to the ration may have an antirachitic effect, and reduce the requirement for vitamin D. The minimum requirement of the calf for vitamin D is not known, but has been estimated (Bechdel et al., 1938) at about 300 I.U. per 100 pounds of body weight per day for the growing calf.

Vitamins E and K

The role of these vitamins in the nutrition of the calf is unknown. Vitamin E deficiency has been suggested (Vawter and Records, 1947, and Schofield, 1947) as the cause of muscle dystrophy in calves, but proof of such deficiency is decidedly lacking.

Vitamin B Complex

An early study of the "vitamin B" requirement of the calf was made by Bechdel, Eckles and Palmer (1926). Their conclusion, that the B vitamin requirements of the calf were adequately provided by bacterial synthesis, was reached after experimental calves fed the vitamin B deficient ration grew to maturity and produced normal offspring. Rats on the same diet lived only two to five weeks. The calves were placed on the experimental diet at 100 days of age after receiving milk during early life.

Lardinois and associates (1944) tested the rumen synthesis of B complex vitamins through the use of a cow and a calf with rumen fistula. Little evidence of thiamin synthesis was obtained, but such synthesis could be masked by rapid absorption. When a basal ration of timothy hay and molasses was supplemented with urea, there was an increase in the rumen synthesis of riboflavin, niacin, pantothenic acid and biotin. Synthesis of B_6 was increased occasionally, and that of folic acid was not affected.

Combinations of vitamins have been used in an effort to combat calf scours and other calfhood maladies. Wisconsin workers (Phillips and Lundquist, 1941, and Lundquist and Phillips, 1943) have indicated that scours were largely of nutritional origin, and that vitamin A and certain members of the B complex were effective in eliminating scours. Vitamins A, C and nicotinic acid were effective in providing protection against navel ill, peritonitis and other active infections, as well as scours.

In contradiction to this work favoring vitamin supplementation, several reports have been published indicating little or no benefit from such supplementation. Norton and associates (1946) provided vitamins A, D,

E, several "B's" and ascorbic acid to 60 calves and obtained no reduction in incidence or severity of scours, and no effect on rate of growth or general appearance. Nevens and Kendall (1947), after testing 299 calves with supplements of vitamins A, D, niacin and ascorbic acid, questioned the value of vitamins for calves as measured by incidence and duration of scours, number of deaths and the cause of death. Judging from the indidence of scours and pneumonia, body weight and general appearance, Gilmore et al. (1947) observed no difference between control calves, those receiving a supplement of vitamins A, D, niacin and ascorbic acid, and those receiving a supplement of vitamin A, thiamin, riboflavin, choline, pantothenic acid, niacin and ascorbic acid. This study covered a period of three years and involved 159 calves. From these reports it would appear that the vitamin supplementation of calves is impractical unless a deficiency is shown to exist.

Choline studies by Waugh, Hauge and King (1946) showed that blood of calves fed milk increased in choline content the first three weeks of life and leveled off thereafter. When milk was removed from the diet at 35 days of age the choline content of the blood declined. The administration of choline chloride failed to maintain normal choline in blood.

Thiamin has been shown to be required by calves on synthetic diets and the symptoms of thiamine deficiency were described by Johnson, Hamilton, Nevens and Boley (1948). Symptoms of the deficiency include reduction in urinary excretions of thiamin, increase in blood and urinary pyruvate levels, weakness, incoordination of legs, convulsions and head retraction, and in some calves severe scouring, anorexia and dehydration.

Johnson and associates (1947a) were unable to show that nicotinic acid was essential for calves on synthetic rations. Urinary excretion of nicotinic acid and its metabolites remained almost constant throughout the three months of the experiment, and was unaffected by feeding one percent sulfathalidine. Thus it would appear that nicotinic acid used by the calf is synthesized by the tissues rather than in the digestive tract. The ration used contained 30 percent casein on the dry basis, and it is possible that nicotinic acid may be required by the calf on a lower protein ration.

Riboflavin deficiency was produced in the calf by Wiese and associates (1947b). The deficiency was characterized by hyperemia of the buccal mucosa, lesions in the corner of the mouth, along the edges of the lips and around the navel, loss of appetite, poor growth, scours, excessive salivation and lachrimation, loss of hair and almost complete disappearance of riboflavin from the urine.

Johnson and associates (1947b) demonstrated the need of the calf for pantothenic acid. Deficiency symptoms included cessation of growth, diarrhea, weakness of the legs, inability to stand and a reduction of the urinary level of pantothenic acid. Administration of calcium pantothenate corrected all symptoms.

Wiese, Johnson and Nevens (1946) produced a biotin deficiency characterized by a paralysis of the rear legs and inability of the calf to stand. Administration of biotin rapidly alleviated the paralysis and increased the urinary excretion of biotin.

Ascorbic Acid

Thurston, Eckles and Palmer (1926) studied the role of vitamin C in calf nutrition, and found that it was not required as judged by measuring feeds by feeding to guinea pigs. The ration consisted of alfalfa hay which was autoclaved 30 minutes at 15 pounds pressure, milk held at a temperature of 180° for one hour with oxygen bubbled through it at the rate of one cubic foot per minute, grain consisting of three parts corn, three parts oats and one part linseed oil meal, cod liver oil and calcium carbonate.

Wiese and associates (1947a) reported no reduction in the level of ascorbic acid in the blood of calves when ascorbic acid was omitted from the synthetic milk ration.

Summary of Review of Literature

The use of semi-purified and purified diets in studying the nutritional requirements of various species has attained considerable significance in the last decade. These diets have been perfected to the point where, for a few species at least, satisfactory growth may be obtained. Most of these diets use casein as the source of protein, butterfat, lard or vegetable oil as the fat source and sugar, dextrin or starch as the carbohydrate source.

The use of purified diets for rats has reached the stage at which excellent growth and normal lactation are obtained on the crystalline vitamins. Crude vitamin carriers must be used to obtain normal lactation.

With guinea pigs the problem has not been so simple, and at least three unidentified factors have been proposed. Some work toward the clarification of these factors has been done but their exact identity is not yet known. The maintenance of dogs on purified diets does not appear to be complicated by unknown factors. Normal growth of swine has been obtained on purified rations, but as yet any attempts to carry hogs through reproduction and lactation on such diets have failed. Calves have not responded well to purified diets, and it was not until recently that normal growth has been obtained. It is doubtful that such diets have any place in studying nutritional requirements for reproduction or lactation in the bovine.

Calves are unable to digest starch at birth and can not fully utilize this carbohydrate until nearly a month old. The digestibility of other carbohydrates for the young calf is unknown. Dextrose has been used in synthetic rations with good results. Lactose, when added to milk, resulted in diarrhea.

Blood serum when mixed with the milk in conjunction with intravenous injection proved a satisfactory substitute for colostrum and resulted in the appearance of globulins in the blood. Evidently the digestive tract is permeable to large molecules for about 36 hours after birth, for the ingestion of colostrum within the first 24 hours resulted in the appearance of globulins in the blood whereas if colostrum were given after the third day of life no globulins were found in the blood of calves.

The calf manages very well without fat, or with butterfat, lard or tallow, but does not fare so well when corn oil, cottonseed oil or soybean oil is used as the fat source. The latter result in unthrifty, emaciated calves, and frequently must be replaced by whole milk in order to save the calves.

Of the minerals, tentative requirements of the growing calf have been established for calcium, phosphorus, magnesium, cobalt, copper and iodine. Potassium, sulfur, sodium, chlorine, maganese and iron have been shown to be essential to the calf but the requirements have not been indicated.

The role of vitamin A in calf nutrition is fairly well known as a result of the comprehensive experimentation in this field. Vitamin D and its role in the utilization of calcium and phosphorus has been studied at the Michigan station and by other workers.

By the use of synthetic milk, Illinois workers have shown that the young calf requires at least thiamin, riboflavin, biotin and calcium pantothenate of the B complex. Nicotinic acid was not required when the ration contained 30 percent protein. Ascorbic acid apparently need not be supplied in the ration of the calf.

OBJECT

The object of this experiment is to compare glucose, dextrin and starch as the principal carbohydrate constituent of synthetic milk rations for young calves.

PLAN OF EXPERIMENT

Animals Used

Three lots of animals will be used in this experiment. Calves from the experimental herd will be allocated to the three lots in such a manner as to keep lots equallized as to size, strength and health of calves. Calves will remain with the dam the first 24 hours after birth, then will be removed and placed in individual pens.

Group 1 Three calves. These calves will be placed on synthetic milk ration in which corn starch is the principal source of carbohydrate.

Group 2 Five calves. These animals will be placed on a synthetic milk ration in which dextrin is the carbohydrate source.

Group 3 Five calves. In this group corn sugar (glucose) will be the carbohydrate component of the synthetic milk ration.

Basal Ration

The basal ration to be used in this experiment consists of 20 percent

corn gluten meal, 20 percent corn sugar, 10 percent linseed oil meal, four percent lard, three percent mineral mixture and 43 percent of the carbohydrate (corn starch, dextrin or corn sugar). These constituents will be ground to 30 mesh, then mixed thoroughly to secure uniform distribution of all ingredients. The 20 percent sugar is included to increase the palatibility of the ration; linseed meal is added as a stablizer to aid in keeping the mix in suspension. This ration is set up as a guide and will be changed as necessary to fit the needs of the calf.

Vitamins A (shark liver oil) and D (viosterol) will be administered via capsule at the weekly rate of 70,000 I. U. of vitamin A and 10,000 I. U. of vitamin D. Each calf will receive daily a capsule containing 20 mg. thiamin hydrochloride, 20 mg. riboflavin, 20 mg. niacin, 20 mg. calcium pantothenate, 20 mg. paraminobenzoic acid, 5 mg. pyridoxine, 2000 mg. choline, 5 mg. folic acid, 200 mg. inositol and 10 mg. 2-methyl-naphthoquinone.

Feeding Plan

Each calf will be fed enough of the synthetic milk mixture to meet the recommendations of the National Research Council for digestible protein and for total digestible nutrients. The feed will be weighed dry, mixed with sufficient warm water to produce a "synthetic milk" of about 12 percent solids, and fed to the calf via the nipple pail. Calves will receive no roughage, but will have access to the dry synthetic milk mix in the feed box.

Accurate records of feed consumption and of feed refusals will be kept throughout the experiment. Quantities fed will be adjusted weekly to conform with recommendations, based on changes in weight.

Bedding

All calves will be kept on heavy wire floors to prevent the ingestion of straw, shavings or other bedding.

Care

Animals will be fed and cared for by the herdsman of the experimental barn. Daily notes will be taken with specific reference to appetite, activity and any abnormalities such as scours or pneumonia.

Weights

Each calf will be weighed when placed on the experiment, and at weekly intervals thereafter.

Feces

Feces will be observed daily for indications of gastroenterological disorders which may accompany the departure from normal feeding regime.

Chemical Analysis

Venous blood samples will be collected from each calf at weekly intervals. These will be analyzed by the Department of Agriculture Chemistry for calcium, phosphorus, magnesium, ascorbic acid, hemoglobin and cell volume. Feed samples will be analyzed at regular intervals.

Post Mortem Examination

Autopsies and post mortem examinations will be conducted with the assistance of the Department of Animal Pathology. Special attention will be devoted to the condition of the gastrointestinal tract.

EXPERIMENTAL PROCEDURE

Animals Used

Thirteen calves were allotted to the three groups as originally planned. Two other groups, including seven calves, were added late in the experiment. Group four calves received the same rations as groups two and three, except that the feed was prepared by either soaking or cooking. As there appears to be no difference between results with soaking and with cooking, the two are considered as one group. For soaking the feed was first mixed dry, then mixed thoroughly with water to form a thin batter and stored under refrigeration in this condition until used. At the time of feeding this batter was mixed with sufficient hot water to form the usual synthetic milk of 12 percent solids. For cooking, all ingredients except the carbohydrate component were mixed dry, then mixed with water to form a thin batter, then baked in the oven at 140° C. for two hours. After cooling, the carbohydrate component and additional water were mixed in, and the mix stored under refrigeration until used.

Group five consisted of two calves on the same ration as group three,

except that linseed meal and corn gluten meal were omitted and sufficient casein was added to replace the protein removed. Table I shows the allocation of calves to their respective groups.

TABLE I. GROUPING OF EXPERIMENTAL ANIMALS

Group Number	Calf Number	Breed	Sex
1	0657	Jersey	Male
	0659	Holstein	Female
	0660	Holstein	Male
2	0661	Holstein	Male
	0663	Holstein	Male
	0665	Guernsey	Male
	0671	Holstein	Female
	0672	Jersey	Male
3	0662	Holstein	Female
	0664	Holstein	Male
	0667	Jersey	Male
	0668	Jersey	Female
	0669	Holstein	Male
4	0673	Holstein	Female
	0675	Jersey	Male
	0676	Holstein	Male
	0677	Holstein	Female
	0679	Holstein	Male
5	0678	Holstein	Male
	0680	Guernsey	Male

Basal Ration

The experiment was initiated using the proposed basal ration. After the first week the ration was modified to conform more closely to the composition of skim milk. The adopted basal ration consisted of 32 percent corn

gluten meal, 15 percent casein, 10 percent linseed meal, four percent lard, three percent mineral mixture and 36 percent carbohydrate (corn starch, dextrin or corn sugar). A comparison of this basal ration, whole milk and skim milk is given in Table II.

TABLE II. A COMPARISON OF THE COMPOSITION OF THE BASAL RATION, WHOLE MILK AND SKIM MILK.

	Basal Ration	Whole Milk	Skim Milk
	percent	percent	percent
Dry matter	91.9	12.9	9.5
Carbohydrate	57.2	37.6	53.1
Protein	30.2	26.3	37.6
Fat	5.3	30.3	1.1
Ash	4.5	5.8	8.2
Fiber	2.8	0	0

Each calf received a weekly supplement of 70,000 I.U. of vitamin A (shark liver oil) and 10,000 I.U. of vitamin D (viosterol) and a daily supplement of 20 mg. of thiamin hydrochloride, 20 mg. of riboflavin, 20 mg. of calcium pantothenate, 20 mg. of niacin, 20 mg. of paraminobenzoic acid, 5 mg. of pyridoxine hydrochloride, 5 mg. of folic acid, 200 mg. of inositol, 2000 mg. of choline and 10 mg. of 2 methyl-napthoquinone. These supplements were administered orally by capsule.

The feeding of calves was conducted as planned except that in the very early stages of the experiment, that is, before the basal ration was modified, it was impossible to get the calves to eat enough of the synthetic milk to meet recommended allowances for digestible protein and total digestible nutrients, as set forth by the National Research Council. After modification of the basal ration little difficulty was encountered in this respect.

Bedding

The original plan of using wire screens was discarded after the first month of the experiment, and wood shavings were used thereafter.

Care

Calves were cared for as planned.

Collection of Data

Data on body weights, general health and blood analysis were collected as planned.

Post Mortem Examinations

Autopsies and post mortem examinations were conducted as planned.

EXPERIMENTAL RESULTS

The rations used in the experiment are shown in Table III.

TABLE III. EXPERIMENTAL RATIONS USED

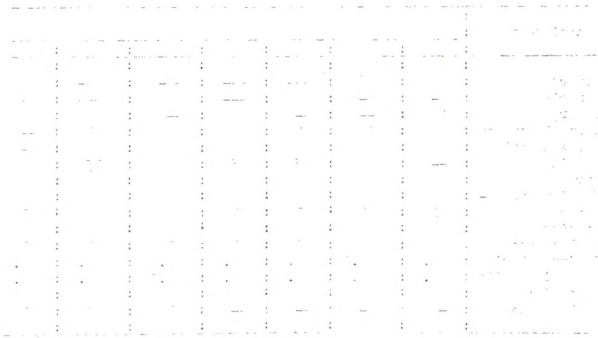
Constituent	1	1a	2	3	4a	4b	5
Starch	43	36	---	---	---	---	20
Dextrin	--	--	36	---	36	---	--
Sugar	20	--	--	36	--	36	36
Corn Gluten Meal	20	32	32	32	31	31	--
Linseed Meal	10	10	10	10	10	10	--
Casein	--	15	15	15	15	15	36
Lard	4	4	4	4	4	4	4
Calcium Carbonate	1	1	1	1	1	1	1
Calcium Phosphate	1	1	1	1	1	1	1
Sodium Chloride	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Magnesium Oxide	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Potassium Phosphate	--	--	--	--	1	1	1

The first calf started on the experiment, C657, was placed on ration 1 and changed to ratinn 1a after one week. The other calves of group 1 were on ration 1a exclusively. Calves of groups 2, 3 and 5 were on rations 2, 3 and 5, respectively. Calves C675 and C677 were on ration 4a while C673, C676 and C679 were on ration 4b.

Chemical analyses of the three rations, 1a, 2 and 3 are presented in Table IV.

Growth

The weekly weights of each of the calves is shown in Table XVII, and



the percent of normal weight (Ragsdale, 1934) is shown in Table XVIII. A summary of the latter is given in Table V.

TABLE IV. CHEMICAL ANALYSES OF RATIONS 1a, 2 AND 3.

	Ration Number		
	1a	2	3
	percent	percent	percent
Moisture	8.79	7.73	7.91
Crude Protein	30.87	29.37	29.62
Crude Fiber	2.68	2.71	2.47
Fermentable Carbohydrate	0.904	4.30	33.91
Ether Extract	4.21	5.87	5.89
Ash	4.91	4.02	4.53
Calcium	0.785	0.624	0.819
Phosphorus	0.555	0.503	0.563
Magnesium	0.445	0.343	0.284
Potassium	0.324	0.298	0.327
Manganese	0.001	0.001	0.001
Iron	0.0226	0.0244	0.0220
Copper	0.0015	0.0018	0.0014
	ppm	ppm	ppm
Cobalt	0.15	0.16	0.17

TABLE V. SUMMARY OF THE PERCENT OF NORMAL BODY WEIGHT OF CALVES BY GROUPS

Weeks on Experiment	Group Number				
	1	2	3	4	5
0	84.7	98.1	100.3	94.3	94.5
1	78.6	86.4	94.2	89.9	85.6
2	71.0	71.3	87.5	82.0	82.7
3	72.2	70.2	84.5	70.7	80.7
4	64.2	62.5	80.2	64.9	78.6
5	61.0	52.9	76.8	61.2	78.6
6	53.8	----	72.3	54.2	80.8
7	----	----	67.5	49.4	80.0
8	----	----	62.5	47.6	80.4

TABLE VI. SUMMARY OF THE AVERAGE PERCENT OF CHANGE FROM STARTING WEIGHT OF CALF

Weeks on Experiment	Group Number				
	1	2	3	4	5
0	0	0	0	0	0
1	+0.5	-4.5	+0.7	+2.6	-2.9
2	-1.7	-9.3	-0.9	+0.3	-2.3
3	-0.7	-4.4	+2.9	-8.6	+1.1
4	-6.9	-8.8	+3.3	-11.1	+5.6
5	-4.0	-17.6	-15.1	-2.0	+11.2
6	-9.3	---	-14.0	-6.9	+21.7
7	---	---	-14.0	+1.7	+28.3
8	---	---	-15.1	-5.6	+35.9

The growth of calves, as measured by the percent of change from the starting weight, is presented in Table XIX and summarized in Table VI. Only the calves of group 5 approached the Ragsdale growth standard after the first four weeks on the experiment. Group 3, the calves on glucose, did well the first four weeks but fell off rapidly after that. The other three groups failed to gain and in most cases lost weight rapidly from the start and continued to do so until death.

Blood Analysis

The results of the weekly analysis of the blood of calves on the experiment are presented in Tables XX to XXXIX, inclusive. These results are summarized in Tables VII to XII, inclusive.

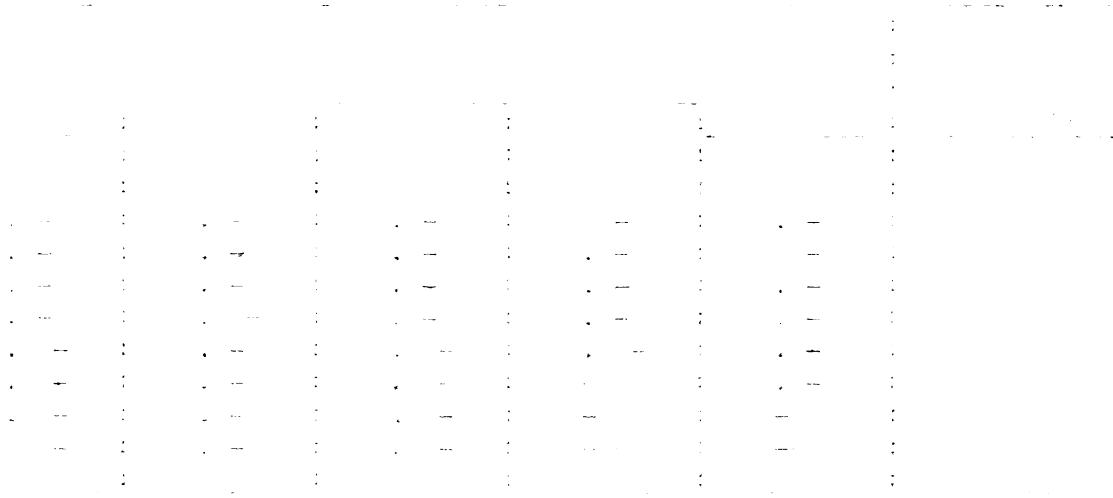


TABLE VII. AVERAGE WEEKLY PLASMA CALCIUM VALUES EXPRESSED AS MILLIGRAMS PER CENT

Weeks on Experiment	Group Number 1	2	3	4	5
0	11.6	11.1	12.1	11.8	12.9
1	10.8	10.8	11.3	10.6	11.5
2	11.2	11.0	11.3	10.0	11.1
3	10.2	----	10.8	9.6	10.8
4	9.8	8.1	10.7	9.7	10.4
5	8.9	10.5	10.5	9.7	10.7
6	9.8	----	11.1	9.3	10.7
7	----	----	10.0	10.1	10.9
8	----	----	9.7	10.2	11.5

TABLE VIII. AVERAGE WEEKLY INORGANIC PHOSPHORUS VALUES EXPRESSED AS MILLIGRAMS PERCENT

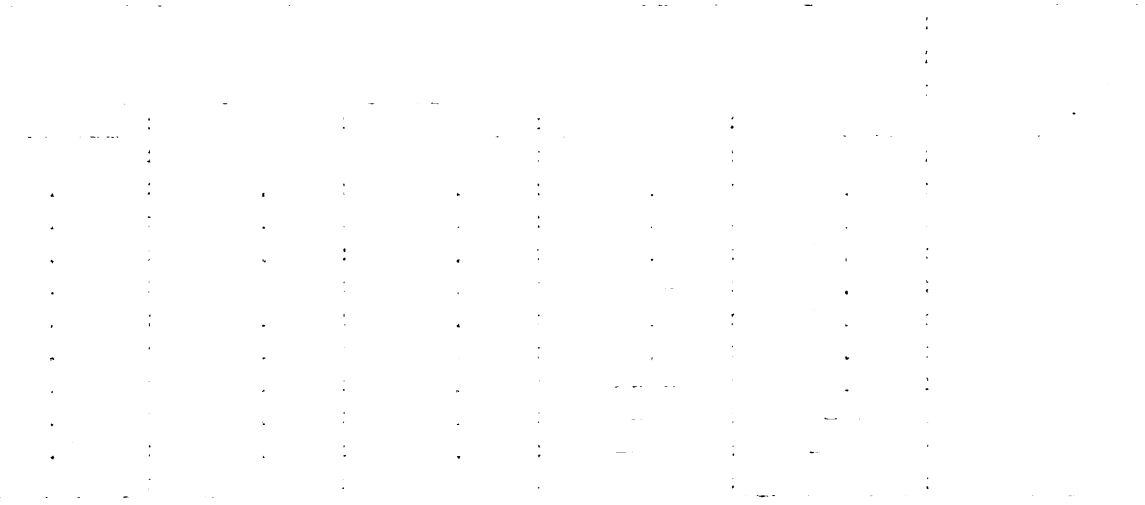
Weeks on Experiment	Group Number 1	2	3	4	5
0	6.45	6.23	5.66	5.77	6.79
1	5.75	6.24	5.78	6.28	5.84
2	5.49	8.09	5.68	5.18	6.07
3	6.33	----	6.36	5.88	6.07
4	4.88	6.48	8.99	5.80	6.12
5	5.02	4.72	6.69	5.39	6.16
6	5.12	----	5.43	6.00	6.43
7	----	----	5.27	5.79	7.10
8	----	----	6.48	6.37	7.34

TABLE IX. AVERAGE WEEKLY PLASMA MAGNESIUM VALUES EXPRESSED AS MILLIGRAMS PERCENT

Weeks on Experiment	Group Number 1	2	3	4	5
0	2.06	2.05	2.91	2.53	2.67
1	2.09	2.15	2.28	1.96	2.18
2	2.07	2.62	1.77	1.85	1.76
3	1.91	----	2.18	1.78	1.55
4	1.61	2.07	1.85	2.07	1.92
5	1.63	1.84	2.31	1.80	1.53
6	1.87	-----	2.16	1.59	1.73
7	----	----	1.87	2.03	1.75
8	----	----	1.83	1.67	1.68

TABLE X. AVERAGE WEEKLY HEMOGLOBIN CONTENT OF BLOOD, EXPRESSED AS GRAMS PERCENT

Weeks on Experiment	Group Number 1	2	3	4	5
0	10.42	10.80	12.45	10.75	8.27
1	9.90	11.29	10.70	13.19	8.00
2	10.42	11.58	11.14	13.13	7.76
3	12.20	-----	10.90	11.98	8.70
4	9.08	12.23	11.67	13.20	8.11
5	8.40	11.00	15.00	12.83	7.73
6	9.20	-----	12.65	12.42	9.05
7	-----	-----	12.27	11.20	10.10
8	-----	-----	11.50	10.21	10.72



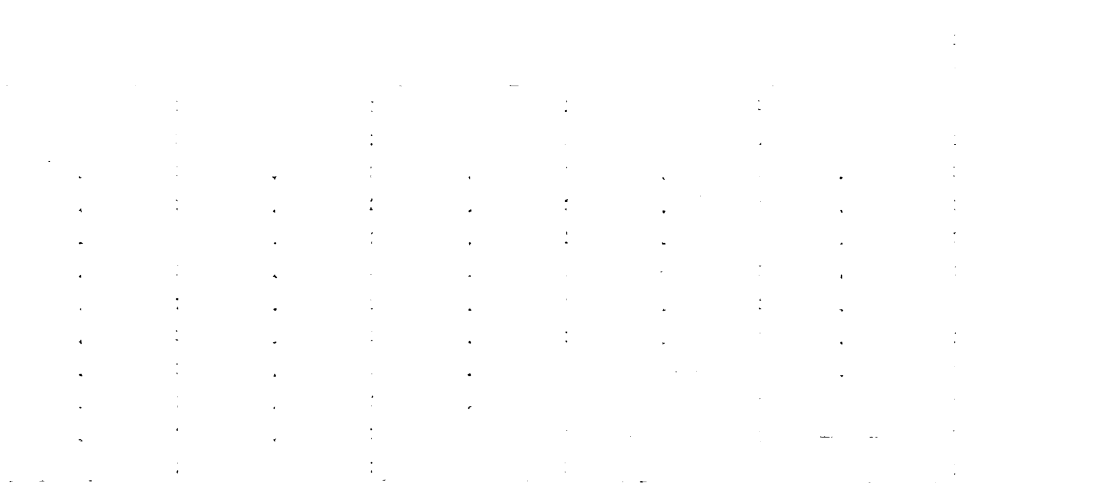
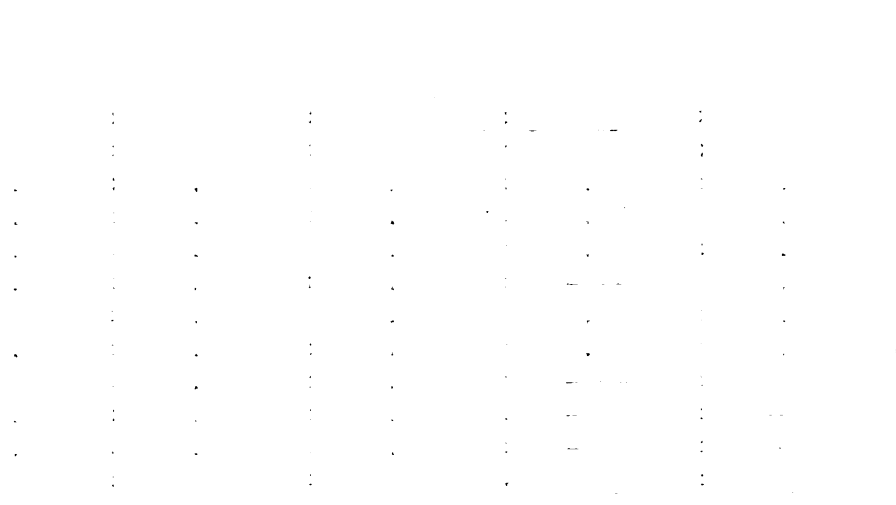


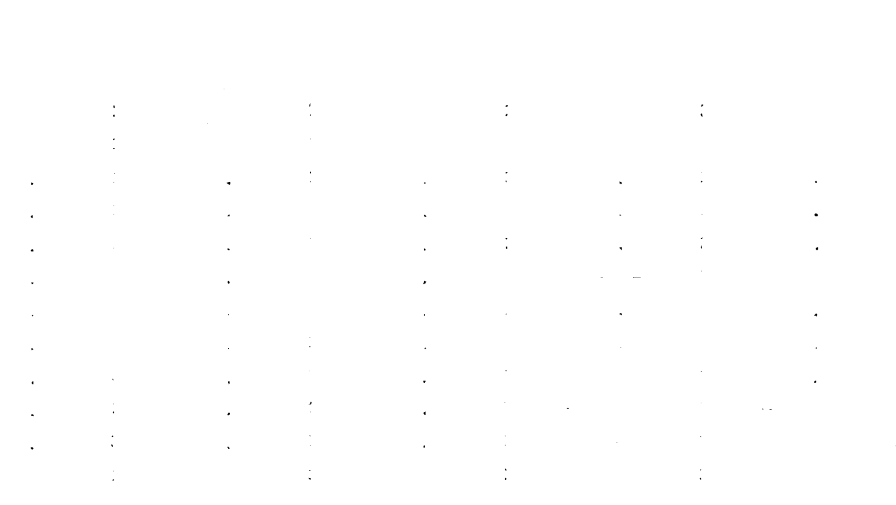
TABLE XI. AVERAGE WEEKLY CELL VOLUME (HEMATOCRIT) OF BLOOD, EXPRESSED AS GRAMS PERCENT

Weeks on Experiment	Group Number 1	2	3	4	5
0	27.5	28.0	35.0	29.0	25.0
1	25.8	30.2	29.9	36.2	22.5
2	27.7	29.7	29.2	35.3	22.0
3	27.2	----	29.1	32.2	23.7
4	23.5	29.5	30.5	35.7	22.2
5	22.0	28.0	42.0	35.2	21.5
6	22.0	----	34.0	33.2	23.7
7	----	----	32.0	31.2	27.5
8	----	----	31.0	28.0	27.7

TABLE XII. AVERAGE WEEKLY PLASMA ASCORBIC ACID VALUES EXPRESSED AS MILLIGRAMS PERCENT

Weeks on Experiment	Group Number 1	2	3	4	5
0	0.361	0.409	0.589	0.499	0.757
1	0.322	0.396	0.364	0.436	0.384
2	0.289	0.276	0.361	0.352	0.262
3	0.322	-----	0.380	0.272	0.228
4	0.242	0.215	0.538	0.280	0.240
5	0.271	0.190	0.412	0.227	0.195
6	0.235	-----	0.206	0.297	0.283
7	-----	-----	0.198	0.245	0.299
8	-----	-----	0.275	0.230	0.307





Survival Time

The length of time of survival on the experimental diet is given for each calf in Table XXXX. The average survival time for each group is shown in Table XIII.

TABLE XIII. TIME OF SURVIVAL OF CALVES ON SYNTHETIC RATIONS

Group	Average Age When Placed on Synthetic Ration	Average Survival Time on Synthetic Ration
	days	days
1	13.7	31.3
2	4.6	16.6
3	3.8	31.0
4	2.8	46.0
5	2.5	90.0*

* Calves still alive at termination of experiment.

The average age at the start of the experiment was much older for group 1 than for any other group. This is due to the fact that one of the three calves of group 1 was 31 days old when placed on the trial. If that calf (0657) is omitted, the average survival time for group 1 becomes about 20 days, and it may be seen that glucose definitely maintains life longer than either starch or dextrin. None of the groups approached the survival time of group 5; thus casein must contain some factor necessary for life that is not provided by corn gluten meal and linseed meal. Cooking or soaking the feed (not including the carbohydrate fraction) increased the life-supporting time, as may be seen by comparing the survival time of group 4 with that of group 2 and group 3. Calves in group 4 received the same rations as those in groups 2 and 3 except for the cooking or soaking treatment.

Necropsy Findings

Immediately after death each calf was taken to the veterinary clinic and a necropsy performed under the direction of Dr. Frank Throp, Jr., or one of his associates in the Department of Animal Pathology. Notes on general health and a report of the necropsy of each calf are presented under "Notes" in the Appendix. A summarization of necropsy findings appears in Table XIV. Necropsy examination consistently revealed no disturbances

TABLE XIV. NECROPSY FINDINGS IN THE GASTROINTESTINAL TRACT OF CALVES ON SYNTHETIC RATIONS.

Group	Percent of Animals Showing Lesions in			
	Rumen	Abomasum	Small Intestine	Large Intestine
	Percent	Percent	Percent	Percent
1	0	67	0	100
2	0	100	40	40
3	0	60	40	0
4	20	20	0	0
5	Not available	--	--	--

outside the gastrointestinal tract. Occasionally pneumonia or white spotted kidney were encountered but in general gross lesions were confined to the digestive tract. With the exception of one calf in group 4, the rumen was normal in all calves. This probably indicates that the food was passing directly to the abomasum in most of the calves most of the time. Further evidence of such passage is provided by the fact that the rumen in practically all instances was small andundeveloped and usually contained only a small amount of food when examined on post mortem. In the abomasum a condition of

petechial hemorrhages was quite prevalent. The condition is illustrated in Fig. 1, a photograph of the interior of the abomasum of calf 0665. This condition was found most extensively in calves of group 2, to a

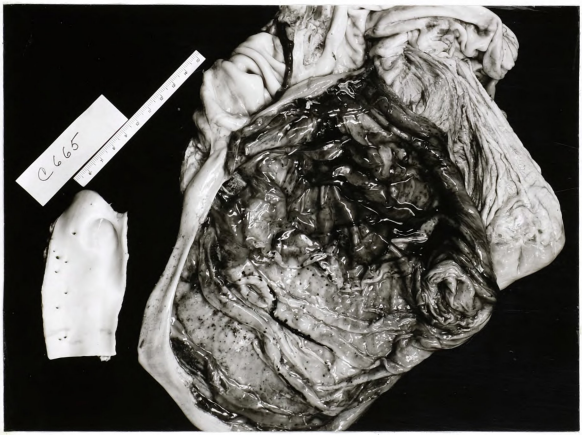


Fig. 1. Petechial hemorrhages of the abomasum of calf 0665.

lesser extent in groups 1 and 3 and in only one calf of group 4. The degree was much more severe in group 2 calves than in the calves of other groups.

Two calves of group 2 were affected with severe inflammation and con-

gestion throughout the small intestine, and two calves of group 3 were found to have moderate congestion of the first three or four feet of the duodenum. All other calves were free of lesions of the small intestine. Severe congestion of the colon and large intestine was encountered in all of the calves of group 1 and in two calves of group 2. Other calves were normal in this region.

Necropsy reports on the calves of group 5 are not available as these calves did not die.

Sections of heart muscle were collected systematically, and occasional sections were taken of thyroid, adrenal, kidney and liver tissue. These sections were examined histologically and no abnormalities noted upon preliminary observation. A detailed systematic study has not been completed.

Biotin-Potassium Deficiency Syndrome

The incidence of a characteristic paralysis in calves on this experiment is shown in Table XV. As shown by the table, only the calves in group

TABLE XV. INCIDENCE OF PARALYSIS IN CALVES ON SYNTHETIC MILK

Group	Number of Calves in Group	Number of Calves Affected with Paralysis	Percent Affected
1	3	0	0
2	5	0	0
3	5	5	100
4	5	0	0
5	2	0	0

3 developed paralysis, and all of the calves in this group were afflicted.

The mineral mixture of groups 1, 2 and 3 contained no potassium whereas the rations of groups 4 and 5 contained one percent K_2HPO_4. Symptoms, treatment and results of treatment of afflicted calves is presented in Table XVI. The first symptom observed was a weakness and inability to control the rear legs. This progressively became worse until the rear legs became completely paralyzed. Diarrhea usually was observed at this stage but this symptom was not found in all calves. Within four to 10 hours after the rear legs became involved, th fore legs were affected by paralysis. Shortly thereafter the paralysis involved the neck and evidently certain areas necessary for respiration. If untreated, death resulted in from 10 to 16 hours after the initial symptoms were observed. Bloat was observed in two of the calves. These symptoms could be corrected with either of two treatments: the subcutaneous administration of 100 micrograms of biotin or the oral administration of potassium in the form of the chloride or phosphate. Results with biotin were slightly more rapid in development than with potassium, but both appeared to be effective. Other potassium salts were not tried, nor were dosages of biotin other than 100 micrograms.

A typical case of paralysis is shown in Fig. 2. This is a photograph of 0662 taken at a time when the rear legs were completely paralyzed and the calf had very little control over the fore legs. The neck had not yet been involved. The same calf is shown in Fig. 3, about 18 hours after the subcutaneous administration of 100 micrograms of biotin. All visible symptoms had disappeared.

TABLE XVI. PARALYSIS IN CALVES OF GROUP THREE

Calf Number	Days on Expt. When Symptoms Developed	Symptoms	Treatment	Results
C662	8	Paralysis of rear legs Lack of control fore legs Unable to stand Bloat	100 ug biotin subcutaneously	Complete recovery in 12 hours
	15	Paralysis of rear legs " " fore legs Bloat	20 grams KCl orally	Complete recovery in 36 hours
C664	23	Paralysis of rear legs Diarrhea	20 grams KCl orally	Death - 2 hours after treatment
C667	27	Paralysis of rear and front legs, followed by paralysis of neck Diarrhea	15 grams KCl orally 100 ug biotin subcutaneously	Complete recovery in 24 hours
C668	63	Paralysis - no control over legs, head or neck Limbs and nose cold Respiration 10 per min.	None attempted	Calf killed and autopsied
C669	5	Overall weakness Paralysis of rear legs Anorexia	30 grams K_2HPO_4 orally (3 10-gram doses at 12 hr. intervals)	Complete recovery in 36 hours

Fig. 2. C66', showing paralysis of rear and fore legs.

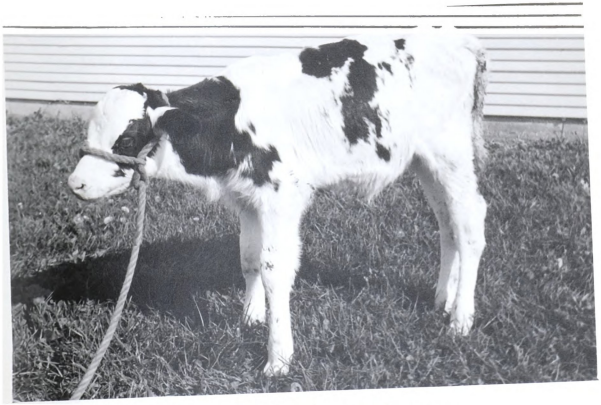


Fig. 3. C66', 18 hours after biotin therapy.

DISCUSSION

Carbohydrate Nutrition of the Young Calf

The use of synthetic rations offers many possibilities in the study of calf nutrition. It is well known that milk is such a complex and complete food that it is difficult if not impossible to conduct certain phases of nutritional investigation as long as milk must be the chief constituent of the diet. Synthetic milk, through the innumerable combinations of nutrients possible, and the many vitamin and mineral supplements which may be used, offers unchallenged advantages over milk as the basal diet for experimental use. The control calves on this experiment and previous work at Illinois (Wiese et al., 1947a) demonstrate that synthetic rations may be employed in studying the nutrition of the young calf.

In this study an attempt was made to use corn gluten meal and linseed meal as partial substitutes for casein as the protein source in synthetic milk. This substitution was unsatisfactory as may be seen by comparing the results of group 5 with those of the other four groups. The reason for this failure is obscure. It may suggest the hypothesis that animal protein is required by the calf, but such a requirement is doubtful in view of the fact that each ration used contained at least 15 percent casein. It is hardly feasible that this amount is insufficient to meet the requirements for the animal protein factor when additional protein is supplied by corn gluten meal and linseed meal.

Doubtless some significance should be attached to the results secured

when the ration (exclusive of the carbohydrate portion) was either cooked or soaked (group 4). Such treatment resulted in a notable increase in time of survival regardless of whether the carbohydrate was dextrin or sugar. There was also a decided reduction in the number and severity of lesions of the gastrointestinal tract. These results would further discredit the animal protein hypothesis, as the treatment used could not be expected to increase this factor in the ration. A second hypothesis which might be advanced is that some substance toxic to the young calf is contained in either corn gluten meal or linseed meal, and is destroyed by the treatment. It is improbable that any such substance would be destroyed or inactivated by such a mild process as soaking, particularly when the water used in soaking remains with the gruel and is fed to the calf. A more plausible explanation is that the treatment reduces the harshness of the feed and thereby reduces the physical irritation of the intestinal mucosa. Such an irritation may be caused by undigested food particles in the lower tract.

The comparison of the carbohydrates admittedly would have been more easily evaluated had casein been used to the exclusion of corn gluten meal and linseed meal. Nevertheless groups 1, 2 and 3 were on a comparable basis and certain conclusions can be drawn from the results obtained. With regard to growth, Tables V and VI, the calves on dextrin when placed on experiment began an immediate and progressive decline in weight which was terminated by death in an average of 16.6 days. These calves had good appetites but scoured profusely, developed rough hair coats, showed general emaciation and rapidly lost weight. Calves on starch also tended to scour but maintained

their original weights usually about three weeks before failing rapidly and dying at an average of 31.3 days. Previous mention has been made of the fact that one calf of this group was 31 days old when placed on the experiment; even when results with this calf are included the group failed to show a longer average survival time than that shown by group 3. The calves fed glucose as the carbohydrate maintained their weights the first two weeks, gained slightly the second two weeks and then declined rapidly. These calves scoured infrequently and showed a much smoother hair coat than did groups 1 and 2. The average survival time was 31 days for group 3; it is possible that the paralysis which occurred in this group caused some reduction in the rate of gain and in survival time.

A comparison of the blood data of the first three groups reveals no essential differences among these groups. When compared with average values obtained on the blood of 38 calves on normal rations, of similar ages and under similar managerial conditions, the calves on the experiment showed, in general, normal hematocrit, hemoglobin and plasma magnesium but subnormal levels of plasma calcium, inorganic phosphorus and ascorbic acid.

Some differences between groups were noted in the location of lesions in the digestive tract, as well as in the severity of the lesions (Table XIV). A suitable explanation for the differentiation in location of lesions is illusive, but one possible explanation will be proposed. Starch, being highly undigestible to the young calf (Shaw, Woodward and Norton, 1918), is not acted upon in the upper tract and the undigested food passes rapidly to the lower tract, where continued irritation creates lesions of the large intestine. Dextrin should be more digestible, yet some of it also escapes to the lower

tract to create irritation. Glucose, easily digested and quickly assimilated, causes some irritation of the abomasum and upper tract, but no irritating substance remains to disturb the lower tract. This hypothesis, however, does not explain why the lesions when dextrin was fed were more severe than those obtained on starch or sugar.

The data presented indicate that the digestive tract of the young calf is unable to handle the more complex carbohydrates such as starch or dextrin. The calves used were able to utilize glucose much more efficiently. Whether other simple sugars are compatable remains to be seen. Lactose frequently has been observed to produce diarrhea, and at least one report (Rojas, Schweigert and Rupel, 1948) indicates that calves do not tolerate lactose added to the milk. Maltose, used almost universally in the formula feeding of infants, might be expected to produce beneficial effects in the calf. The cost of such sugars as maltose and lactose prohibits their general inclusion in synthetic diets but it would be of interest to know how well they are tolerated by the neonatal calf.

Biotin-Potassium Deficiency Syndrome

The incidence of paralysis in this experiment ranged from zero for groups 1, 2, 4 and 5 to 100 percent for group 3. This striking differentiation is indicative of some fundamental difference between group 3 and the other groups. The absence of the syndrome from groups 4 and 5 can be accredited to the inclusion of potassium phosphate in the rations of these groups. The failure of the syndrome to appear in groups 1 and 2 is not so easily explained, but is probably accounted for by the property of starch and dextrin to favor

the synthesis of biotin. Glucose evidently did not stimulate the synthesis of biotin. Similar differences between dextrin and sucrose have been observed in the fowl (Couch and associates, 1948) and are generally recognized in rat studies.

Stimulation of bacterial synthesis of biotin by dextrin and starch but not by glucose would account for the appearance of the deficiency syndrome in group 3 and its absence in groups 1 and 2. It does not account for the therapeutic action of potassium salts when administered to paralysis-afflicted calves, or for the prevention of paralysis in groups 4 and 5 by supplementing with potassium phosphate. This action raises a question as to whether the primary deficiency is one of biotin or of potassium. If biotin is the primary deficiency, then the mode of action of potassium is difficult to understand. Ruegamer and associates (1946) have suggested that potassium increases the intestinal synthesis of biotin. This may be true, but in this experiment there was little if any difference in the recovery rate of paralyzed calves whether treated with potassium or with biotin. Theoretically, if biotin is the deficiency and potassium stimulates synthesis, a longer time should be required for potassium to produce cures than is required by biotin.

If potassium is the primary deficiency, then the curative action of biotin is even more difficult to explain. Potassium being the primary deficiency, one would expect paralysis in groups 1 and 2 as well as group 3. Even though colostrum is low in potassium (Garrett and Overman, 1940), it should be quite difficult to produce a deficiency of potassium in the young calf. The blood of the neonatal calf contains three times the amount of

potassium found in the blood of bovines over 10 weeks of age (Wise and associates,1947). From 170 to 270 days have been required to produce potassium deficiency in the calf when the ration contained 0.10 - 0.12 percent potassium (Sykes and Alfredson, 1940). After production of the deficiency, these workers increased the potassium in the ration to 0.20 percent with the result that the serum potassium was elevated to normal levels. The synthetic milk rations used in this experiment were analyzed and found to contain from 0.298 to 0.327 percent potassium (Table IV). With potassium levels in the ration so much higher than those used by Sykes and Alfredson, it appears unlikely that potassium deficiency could be produced in so short a time. Sections of the heart were examined histologically, but the lesions of the Purkinje network observed by Sykes and Moore (1942) in potassium deficient calves were not found. The evidence accumulated would indicate that the possibility of potassium being the primary deficiency is indeed remote.

A somewhat similar situation has been encountered with dogs. Smith (1945) described a progressive paralysis in dogs on synthetic diets which was cured with synthetic biotin or with yeast. The paralysis occurred suddenly after dogs had been on the deficient diet seven and one-half to 48 weeks. Dogs first developed an abnormal alertness, then spasticity of the hind legs and inability to get up from the sitting position. From four to eight hours after complete paralysis of the hind legs, the forelegs became affected. One to four hours later there was a loss of function of the neck. If untreated, the dog died from respiratory failure. When treated, dogs first recovered use of the neck, then of the forelegs and lastly of

the hind legs. Smith termed the syndrome neurologic, and obtained alleviation by administering 100 micrograms of biotin per kilogram of body weight. One dog was treated with potassium chloride but did not respond.

Ruegamer and associates (1946) used the same ration as that used by Smith, obtained the same syndrome, and obtained disappearance of paralysis in all dogs treated with potassium. Addition of biotin to the ration failed to prevent paralysis. Smith (1946) subsequently reported that either biotin or potassium were effective in correcting the paralysis, the response to potassium therapy requiring three to six hours and that to biotin usually eight hours before improvement started. Both groups of workers agreed that potassium is probably the primary deficiency.

It is interesting that the two species, the bovine and the canine, develop similar symptoms on a deficient ration, and that both potassium and biotin are effective in the treatment of both species. Two of the proposed explanations offered in this dog work might lead one to believe that the primary deficiency in the calf, as well as in the dog, is one of potassium. Ruegamer suggests that the paralysis is due to a breakdown in the enzyme responsible for muscular contraction, and that potassium is involved in the enzyme system. This still does not explain the failure of paralysis to occur in groups 1 and 2 of this experiment, unless biotin is involved in the enzyme system or in the mobilization of potassium. Smith suggests that biotin does have a role in the mobilization of potassium. Such a role would explain the results with calves; it is difficult to accept the proposal of potassium as

the primary deficiency when the ration fed the calves was so high in potassium. If biotin is required for the mobilization of potassium, then a potassium deficiency could occur on a high potassium ration if biotin were deficient. At any rate, an interrelationship apparently exists between the vitamin and the mineral. The exact nature of such an interrelationship can be only a matter of conjecture at the present time.

SUMMARY AND CONCLUSIONS

Twenty newborn calves were distributed equitably to five groups for the purpose of studying the relative values of corn starch, dextrin and corn sugar as the carbohydrate source in synthetic rations.

All calves were placed on synthetic milk composed of the carbohydrate (starch, dextrin or sugar), corn gluten meal, linseed meal, casein, lard and minerals. Vitamins A, D. K and nine B vitemins were provided as supplements.

Groups of calves were compared as to growth, blood analyses, general appearance, diarrhea, survival time and necropsy findings.

It is concluded that glucose excells starch and dextrin on the above bases of comparison; however the growth of all calves was inferior to that of a control group in which casein replaced both corn gluten meal and linseed meal as the protein source.

Calves on sugar developed a paralysis, curable by either biotin or potassium, whereas no paralysis was encountered on starch or dextrin. Evidently the latter two carbohydrates promote synthesis of biotin in the gastrointestinal tract. The possible interrelationship of biotin and potassium is discussed.

LITERATURE CITED

Bechdel, S. I., C. H. Eckles and L. S. Palmer. 1926.
Vitamin B requirement of the calf. Jour. Dairy Sci. 9: 409-438.

Bechdel, S. I., N. W. Hilston, N. B. Guerrant and R. A. Dutcher. 1938.
The vitamin D requirement of dairy calves. Penn. Agr. Expt. Sta. Bul. 364.

Bechdel, S. I., K. G. Landsburg and O. J. Hill. 1933.
Richets in calves. Penn. Agr. Expt. Sta. Tech. Bul. 291.

Bechtel, H. E., E. T. Hallman C. F. Huffman and C. W. Duncan. 1936.
Pathology of richets in dairy calves. Mich. Agr. Expt. Sta. Tech. Bul. 150.

Cannon, M. D. and G. A. Emerson. 1939.
Dietary requirements of the guinea pig with reference to the need for a special factor. Jour. Nutr. 18: 155-167.

Cerecedo, L. R. and L. J. Vinson. 1944.
Growth, reproduction, and lactation in mice on highly purified diets, and the effect of folic acid concentrates on lactation. Arch. Biochem. 5: 157-164.

Couch, J. R., W. W. Cravens, C. A. Elvehjem and J. G. Halpin. 1948.
Relation of carbohydrate to intestinal synthesis of biotin and hatchability in mature fowl. Jour. Nutr. 35: 57-72.

Daniel, F. K. and E. H. Harvey. 1947.
Some observations on the nutritional value of dialized whey solids. Jour. Nutr. 33: 429-436.

Davis, G. K. and L. A. Maynard. 1937.
Cod-liver oil tolerance in calves. Jour. Dairy Sci. 21: 143-152.

Duncan, C. W. and C. F. Huffman. 1936.
Vitamin D studies in cattle. III. Influence of solar ultraviolet radiation upon the blood chemistry and mineral metabolism of dairy calves. Jour. Dairy Sci. 19: 291-303.

Ellis, N. R. and L. L. Madsen. 1944.
The thiamine requirement of pigs as related to the fat content of the diet. Jour. Nutr. 27: 253-262.

Ershoff, B. H. 1944.
Effect of purified rations on deciduomal formation in the rat. Amer. Jour. Physiol. 141: 365-367.

Espe, D. L. and C. Y. Cannon. 1935.
The relation of the fat content of milk to the passage of the milk curd from the stomach of the calf. Jour. Dairy Sci. 18: 141-147.

Espe, D. L. and C. Y. Cannon. 1940.
The length of the intestine of calves and its bearing on the absorption of the nutrients from the chyme. Jour. Dairy Sci. 23: 1211-1214.

Frost, D. V. and F. P. Dann. 1944.
Unidentified factor(s) in yeast and liver essential to the cure of achromotrichia in dogs on synthetic diets. Jour. Nutr. 27: 355-362.

Flora, C. C., R. E. Ward and S. I. Bechdel. 1939.
Carotene in the nutrition of dairy calves. Jour. Dairy Sci. 22: 321-328.

Fouts, P. J., O. M. Helmer and S. Lepkovsky. 1940.
Factor II deficiency in dogs. Jour. Nutr. 19: 393-400.

Garrett, O. F. and O. R. Overman. 1940.
Mineral composition of colostral milk. Jour. Dairy Sci. 23: 13-17.

Gavin, G. and E. W. McHenry. 1940.
The B vitamins and fat metabolism. III. The effects of vitamin B upon liver and body fat. Jour. Biol. Chem. 132: 41-46.

Geyer, R. P., B. R. Geyer, P. H. Derse, T. Zinkin, C. A. Elvehjem and E. B. Hart. 1947.
Growth studies with rats kept under conditions which prevent coprophagy. Jour. Nutr. 33: 192-142.

Gilmore, L. O., L. M. Jones, L. A. Kanegis and M. H. Roepke. 1947.
The prophylactic administration of vitamins to dairy calves.
Jour. Amer. Vet. Med. Assoc. 110: 390-393.

Goettsch, M. and A. M. Pappenheimer. 1931.
Nutritional muscular dystrophy in the guinea pig and rabbit.
Jour. Expt. Med. 54: 145-165.

Gullickson, T. W. and J. B. Fitch. 1944.
Effect of adding cod liver oil to the ration of dairy calves.
Jour. Dairy Sci. 27: 331-335.

Gullickson, T. W., F. C. Fountaine and J. B. Fitch. 1942.
Various oils and fats as substitutes for butterfat in the
ration of young calves. Jour. Dairy Sci. 25: 117-128.

Henderson, H. O. and C. E. Weakley, Jr. 1930.
The effect of feeding different amounts of calcium and phosphorus
upon the growth and development of dairy animals. West Va. Agr.
Expt. Sta. Bul. 231.

Henderson, L. M., J. M. McIntire, H. A. Waisman and C. A. Elvehjem. 1942.
Pantothenic acid in the nutrition of the rat. Jour. Nutr. 23:
47-58.

Hibbs, J. W. and W. E. Krauss. 1947.
The effect of supplementary vitamins on blood composition, liver
storage, and incidence of scours in calves. Jour. Dairy Sci.
30: 115-120.

Hogan, A. G. and J. W. Hamilton. 1942.
Adequacy of simplified diets for guinea pigs and rabbits. Jour.
Nutr. 23: 533-543.

Hogan, A. G. and W. S. Ritchie. 1934.
Nutritional requirements of rabbits and guinea pigs. Mo. Agr.
Expt. Sta. Res. Bul. 219.

Howe, P. E. 1921.
An effect of the ingestion of colostrum upon the composition of
the blood of newborn calves. Jour. Biol. Chem. 49: 115-118.

Howe, P. E. 1924.
Relation between the ingestion of colostrum or blood serum and the appearance of globulin and albumin in the blood and urine of the newborn calf. Jour. Expt. Med. 39: 313-320.

Huffman, C. F. 1931.
Make a study of richets in calves. Sunshine counteracts deficiencies in rations which would cause disease. Mich. Agr. Expt. Sta. Quart. Bul. 14: 42-45.

Huffman, C. F., C. L. Conley, C.C. Lightfoot and C. W. Duncan. 1941.
Magnesium studies in calves. II. The effect of magnesium salts and various natural feeds upon the magnesium content of the blood plasma. Jour. Nutr. 22: 609-620.

Huffman, C. F. and C. W.Duncan. 1935.
Vitamin D studies in cattle. II. The vitamin D sparing action of magnesium in the ration of dairy cattle. Jour. Dairy Sci. 18: 605-620.

Huffman, C. F., C. W. Duncan, C. S. Robinson and L. W. Lamb. 1933.
Phosphorus requirement of dairy cattle when alfalfa furnishes the principal source of protein. Mich. Agr. Expt. Sta. Tech. Bul. 134.

Hughes, E. H. 1939.
The role of riboflavin and other factors of the vitamin B complex in the nutrition of the pig. Jour. Nutr. 17: 527-533.

Hughes, E. H. 1940a.
The minimum requirement of riboflavin for the growing pig. Jour. Nutr. 20: 233-238.

Hughes, E. H. 1940b.
The minimum requirement of thiamine for the growing pig. Jour. Nutr. 20: 239-241.

Hughes, E. H. and N. R. Ittner. 1942.
The minimum requirement of pantothenic acid for the growing pig. Jour. Animal Sci. 1: 116-119.

Johnson, B. C., T. S. Hamilton, W. B. Nevens and L. E. Boley. 1948.
Thaimine deficiency in the calf. Jour. Nutr. 35: 137-145.

Johnson, B. C., A. C. Wiese, H. H. Mitchell and W. B. Nevens. 1947a.
The metabolism of nicotinic acid and its role in the nutrition of the calf. Jour. Biol. Chem. 167: 729-736.

Johnson, B. C., H. H. Mitchell, T. S. Hamilton and W. B. Nevens. 1947b.
Vitamin deficiencies in the calf. Fed. Proc. 6: 410-411.

Johnson, P. E., J. K. Loosli and L. A. Maynard. 1940.
Purified diet studies with calves. Jour. Dairy Sci. 23: 553-554.

Jones, I. R., C. H. Eckles and L. S. Palmer. 1926.
Role of vitamin A in the nutrition of calves. Jour. Dairy Sci. 9: 119-139.

Kuiken, K. A., R. H. McCoy, M. O. Schultze and C. G. King. 1944.
Simplified diets for the guinea pig. Jour. Nutr. 27: 385-394.

Lambooy, J. P. and E. S. Nasset. 1943.
The inadequacy of eight synthetic B vitamins for the nutrition of puppies -- unknown factor (factors) in yeast and probably liver. Jour. Nutr. 26: 293-302.

Lardinois, C. C., R. C. Mills, C. A. Elvehjem and E. B. Hart. 1944.
Rumen synthesis of the vitamin B complex as influenced by ration composition. Jour. Dairy Sci. 27: 579-583.

Leach, T. A. and N. S. Golding. 1931.
A preliminary report of the substitution of pilchard oil for butterfat in milk for calf feeding. Sci. Agr. 12: 204-205.

Lewis, J. M. and L. T. Wilson. 1946.
Vitamin A requirements in calves, part I. Cert. Milk 21: 244-245.

Lindley, D. C. and J. T. Cunha. 1946.
Nutritional significance of inositol and biotin for the pig. Jour. Nutr. 32: 47-59.

Lindsey, J. B., J. G. Archibald and P. R. Nelson, 1931.
The calcium requirements of dairy heifers. Jour. Agr. Res. 42: 883-896.

Lundquist, N. S. and P. H. Phillips. 1943.
Certain dietary factors essential for the growing calf. Jour. Dairy Sci. 26: 1023-1030.

Madsen, L. L. 1936.
The comparative effects of cod liver oil, codliver oil concentrate, lard and cottonseed oil in a synthetic diet on the development of nutritional muscular dystrophy. Jour. Nutr. 11: 471-493.

Mannering, G. J., M. D. Cannon, H. V. Barki, C. A. Elvehjem and E. B. Hart. 1943.
Further evidence for the existence of specific dietary essentials for the guinea pig. Jour. Biol. Chem. 151: 101-107.

Mattill H. A. and P. B. Hawk. 1912.
Studies on water drinking. X. Fecal output and its carbohydrate content under the influence of copious and moderate water drinking with meals. Jour. Amer. Chem. Soc. 33: 2019-2032.

McAnally, R. A. 1943.
Studies on the alimentary tract of merino sheep in South Africa. X. Notes on the digestion of some sugars in the rumen of sheep. Onderstepoort Jour. Vet. Sci. & Animal Ind. 18: 131-138.

McRoberts, V. F. and A. G. Hogan. 1944.
Adequacy of simplified diets for the pig. Jour. Nutr. 28: 165-174.

Mitchell, H. H. 1947.
The mineral requirements of farm animals. Jour. Animal Sci. 6: 365-377.

Mitchell, H. H. and T. S. Hamilton. 1940.
The utilization by calves of energy in rations containing different percentages of protein and in glucose supplements. Jour. Agr. Res. 61: 847-864.

Morgan, A. F. and H. D. Simms. 1940.
Greying of fur and other disturbances in several species due to a vitamin deficiency. Jour. Nutr. 19: 233-250.

Moore, L. A. 1945.
Vitamin A and carotene content of the blood plasma of dairy calves from birth to four months. Jour. Dairy Sci. 28: 821-826.

Moore, L. A. and E. T. Hallman. 1936.
Production of white spotted kidneys in calves. Jour. Dairy Sci. 19: 434-435.

Nelson, M. M. and H. M. Evans. 1947.
Growth, reproduction, and lactation in rat maintained on purified diets. Arch. Biochem. 12: 213-228.

Nevens, W. B. and K. A. Kendall. 1947.
The value of supplementary vitamin feeding in the rearing of dairy calves. Jour. Dairy Sci. 30: 175-181.

Nikitin, V. I. 1939.
Digestion in the rumen of calves. Ukrain. Biochem. Jour. 14: 203-222. (Nutr. Abs. & Rev. 10: 420).

Norton, C. L., H. D. Eaton, J. K. Looski and A. A. Spielman. 1946.
Value of supplemental vitamins for young dairy calves. Jour Dairy Sci. 29: 231-238.

Phillips, P. H., N. S. Lundquist and P. D. Boyer. 1941.
The effect of vitamin A and certain of the B-complex upon calf scours. Jour. Dairy Sci. 24: 977-982.

Phillipson, A. T. and R. A. McAnally. 1942.
Studies on the fate of carbohydrates in the rumen of sheep. Jour. Expt. Biol. 19: 199-214.

Powick, W.C., N. R. Ellis, L. L. Madsen and C. N. Dale. 1947.
Nicotinic acid deficiency and nicotinic acid requirements of young pigs on purified diet. Jour. Animal Sci. 6: 311-324.

Ragsdale, A. C. 1934.
Growth standards for dairy cattle. Mo. Agr. Expt. Sta. Bul.336.

Reed, O. E. and C. F. Huffman. 1926.
Feeding of concentrates to dairy cattle. Heavy feeding of concentrates without the proper quality of roughage is detrimental to the animal. Mich. Agr. Expt. Sta. Quart. Bul. 8: 118-122.

Reed, O. E. and C. F. Huffman. 1930.
The results of a five year mineral feeding investigation with dairy cattle. Mich. Agr. Expt. Sta. Tech. Bul. 105.

Richardson, L. R., A. G. Hogan, B. Long and K. I. Itschner. 1941.
The number of vitamins required by the rat. Proc. Soc. Expt. Bio. Med. 46: 530-532.

Rojas, J., B. S. Schweigert and I. W. Rupel. 1948.
The utilization of lactose by the dairy calf fed normal or modified milk diets. Jour. Dairy Sci. 31: 81-87.

Ruegamer, W. R., C. A. Elvehjem and E. B. Hart. 1946.
Potassium deficiency in the dog. Proc. Soc. Expt. Biol. Med. 61: 234-238.

Ruegamer, W. R., L. Michaud, C. A. Elvehjem and E. B. Hart. 1945.
Growth and hemoglobin production in dogs on purified rations. Amer. Jour. Physiol. 145: 23-27.

Rupel, I. W., G. Bohstedt and E. B. Hart. 1933.
Vitamin D in the nutrition of the dairy calf. Wis. Agr. Expt. Sta. Res. Bul. 115.

Russell, W. C., A. E. Teeri and K. Unna. 1948.
Growth and reproduction of swine on a purified diet. Jour. Nutr. 35: 321-332.

Schaefer, A. E., J. M. McKibbin and C. A. Elvehjem. 1942a.
Studies on the vitamin B complex in the nutrition of the dog. Jour. Nutr. 23: 491-500.

Schaefer, A. E., J. M. McKibbon and C. A. Elvehjem. 1942b.
Pantothenic acid deficiency studies in dogs. Jour. Biol. Chem. 143: 321-330.

Schantz, E. J., C. A. Elvehjem and E. B. Hart. 1940.
The comparative nutritive value of butterfat and certain vegetable oils. Jour. Dairy Sci. 23: 181-189.

Schofield, F. W. 1947.
Sudden death in calves associated with myocardial degeneration. Canadian Jour. Comp. Med. 11: 324-329.

Scott, E. M. 1946.
Self selection of diet. I. Selection of purified components. Jour. Nutr. 31: 397-407.

Seeler, A. O. and R. H. Silber. 1945.
Studies on the prolonged maintenance of adult dogs on purified diets. Jour. Nutr. 30: 111-116.

Shaw, R. H., T. E. Woodward and R. P. Norton. 1918.
Digestion of starch by the young calf. Jour. Agr. Res. 12: 575-578.

Sheehy, E. J. 1935.
The mechanical aspect of the nutrition of farm stock. Sci. Proc. Roy. Dublin Soc. 21: 257-280.

Sheehy, E. J., and B.J. Senoir. 1936.
Mineral metabolism in the calf and the addition of inorganic minerals to the calf's diet. Ireland Dept. Agr. Jour. 34: 1-32.

Shoptaw, L. 1936.
Soybean flour as a substitute for cow's milk in feeding dairy calves. Jour. Dairy Sci. 19: 95-99.

Shoptaw, L., D. L. Espe and C. Y. Cannon. 1937.
Gastric digestion of soybean flour. Jour. Dairy Sci. 20: 117-128.

Smith, S. G. 1945.
A progressive paralysis in dogs cured with synthetic biotin. Amer. Jour. Physiol. 144: 175-188.

Smith, S. G. 1946.
Potassium vs. biotin in the treatment of progressive paralysis in dogs. Proc. Soc. Expt. Biol. Med. 63: 339-341.

Smith, T. 1925.
Focal interstitial nephritis in the calf following interference with the normal intake of colostrum. Jour. Expt. Med. 41: 413-425.

Smith, T. and R. B. Little. 1922a.
The significance of colostrum to the newborn calf. Jour. Expt. Med. 36: 181-198.

Smith, T. and R. B. Little. 1922b.
Cow serum as a substitute for colostrum in new-born calves. Jour. Expt. Med. 36: 453-468.

Smith, T. and R. B. Little. 1923.
The absorption of specific agglutinins in homologous serum fed to calves during the early hours of life. Jour. Expt. Med. 37: 671-683.

Smith, T. and R. B. Little. 1924.
Proteinuria in newborn calves following feeding of colostrum. Jour. Expt. Med. 39: 303-312.

Sober, H. A., G. J. Mannering, M. D. Cannon, C. A. Elvehjem and E. B. Hart. 1942.
Nutrition of the guinea pig. Jour. Nutr. 24: 503-514.

Sure, B. 1943.
Dietary requirements for fertility and lactation. XXXI. Further studies in lactation and growth of the albino rat. Jour. Nutr. 26: 275-283.

Sykes, J. F. and B. V. Alfredson. 1940
Studies on the bovine electrocardiogram. I. Electrocardiographic changes in calves on low potassium rations. Proc. Soc. Expt. Biol. Med. 43: 575-579.

Sykes, J. F. and L. A. Moore. 1942.
Lesions of the Purkinje network of the bovine heart as a result of potassium deficiency. Arch. Path. 33: 467-471.

Thurston, L. M., C. H. Eckles and L. S. Palmer. 1926.
The role of the antiscorbutic vitamin in the nutrition of calves. Jour. Dairy Sci. 9: 37-49.

Vawter, L. R. and E. Records. 1947.
Muscular dystrophy (white muscle disease) in young calves. Jour. Amer. Vet. Med. Assoc. 110: 152-157.

Ward, C. A., C. Y. Cannon and D. L. Espe. 1937.
The value of corn sugar in the grain mixture of dairy calves. Jour. Dairy Sci. 20: 413.

Waugh, R. K., S. M. Hauge and W. A. King. 1946.
Choline studies with young dairy calves. Jour. Animal Sci. 5: 397-398.

West, H. D., N. C. Jefferson and R. E. Rivera. 1943.
The effect of aromatic hydrocarbons on growth. The reversible inhibition of pantothenic acid by sulfapyridine. Jour. Nutr. 25: 471-477.

Wiese, A. C., B. C. Johnson, H. H. Mitchell and W. B. Nevens. 1947a.
Synthetic rations for the dairy calf. Jour. Dairy Sci. 30: 87-94.

Wiese, A. C., B. C. Johnson, H. H. Mitchell and W. B. Nevens. 1947b.
Riboflavin deficiency in the dairy calf. Jour. Nutr. 33: 263-270.

Wiese, A. C., B. C. Johnson and W. B. Nevens. 1946.
Biotin deficiency in the dairy calf. Proc. Soc. Expt. Biol. 63: 521-523.

Wintrobe, M. M., M. H. Miller, R. H. Follis, Jr., H. J. Stein, C. Mushatt and S. Humphreys. 1942.
Sensory neuron degeneration in pigs. IV. Protection afforded by calcium pantothenate and pyridoxine. Jour. Nutr. 24: 345-366.

Wintrobe, M. M., D. M. Mitchell and L. C. Kolb. 1938.
Sensory neuron degeneration in vitamin deficiency. Jour. Expt. Med. 68: 207-220.

Wintrobe, M. M., M. Samter and H. Lisco. 1939.
Morphological changes in the blood of pigs associated with deficiency of water-soluble vitamins and other substances contained in yeast. Bul. Johns Hopkins Hosp. 64: 399-424.

Wintrobe, M. M., H. J. Stein, R. H. Follis, Jr., and S. Humphreys. 1945.
Nicotinic acid and the level of protein intake in the nutrition of the pig. Jour. Nutr. 30: 395-412.

Wise, G. H., M. J. Caldwell, D. B. Parrish, R. J. Flipse and J. S. Hughes. 1947.
Changes in cell volume and in concentration of hemoglobin and of several inorganic constituents of the blood of calves during early postnatal development. Jour. Dairy Sci. 30: 983-993.

Wise, G. H., T. W. Gullickson and W. E. Peterson. 1939.
Value of various vitamins and inorganic salts for overcoming symptoms manifested in calves restricted to a whole milk ration. Jour. Dairy Sci. 22: 573-582.

Wise, G. H., W. E. Peterson and T. W. Gullickson. 1940.
Comparative physiological responses of dairy calves fed rations having different levels of milk proteins. Jour. Dairy Sci. 23: 91-102.

Woolley, D. W. 1942.
Some new dietary essentials required by guinea pigs. Jour. Biol. Chem. 143: 679-684.

Woolley, D. W. and H. Sprince. 1945.
The nature of some new dietary factors required by guinea pigs. Jour. Biol. Chem. 157: 447-453.

Wright, L. D. and A. D. Welch. 1944.
Folic acid, biotin and pantothenic acid deficiency and the liver storage of various vitamins in rats fed succinylsulfathiazole in highly purified rations. Jour. Nutr. 27: 55-66.

APPENDIX

TABLE XVII. WEEKLY WEIGHTS OF CALVES

Calf Number	Number of Weeks on Experiment								
	0	1	2	3	4	5	6	7	8
Group 1									
C657	71	77	70	69	65	--	--	--	--
C659	75	78	77	76	71	72	68	--	--
C660	77	75	72	--	--	--	--	--	--
Group 2									
C661	91	87	87	87	83	75	--	--	--
C663	91	91	78	--	--	--	--	--	--
C665	83	72	--	--	--	--	--	--	--
C671	77	71	--	--	--	--	--	--	--
C672	63	65	--	--	--	--	--	--	--
Group 3									
C662	78	75	74	75	--	--	--	--	--
C664	97	98	99	103	--	--	--	--	--
C667	45	46	46	47	48	--	--	--	--
C668	86	87	86	90	86	73	74	74	73
C669	106	109	103	--	--	--	--	--	--
Group 4									
C673	73	78	79	74	75	79	72	70	69
C675	72	72	63	62	55	--	--	--	--
C676	87	92	98	80	71	--	--	--	--
C677	96	92	81	80	79	79	73	--	--
C679	67	70	73	63	68	71	70	72	--
Group 5									
C678	85	86	81	82	86	87	94	100	106
C680	70	65	70	74	77	84	93	98	103

TABLE XVIII. PERCENT OF NORMAL WEIGHTS OF CALVES

Calf Number	Number of Weeks on Experiment								
	0	1	2	3	4	5	6	7	8
Group 1									
0657	88.8	85.3	78.4	72.4	64.1	--	--	--	--
0659	83.3	82.0	76.8	72.1	64.3	61.0	53.8	--	--
0660	82.1	68.5	61.7	--	--	--	--	--	--
Group 2									
0661	94.7	79.5	74.6	70.2	62.5	52.9	--	--	--
0663	95.7	84.7	68.0	--	--	--	--	--	--
0665	114.5	94.7	--	--	--	--	--	--	--
0671	83.5	73.5	--	--	--	--	--	--	--
0672	101.9	99.4	--	--	--	--	--	--	--
Group 3									
0662	86.7	78.2	73.3	70.6	--	--	--	--	--
0664	103.2	97.8	92.2	89.8	--	--	--	--	--
0667	75.0	71.1	67.2	69.6	63.0	--	--	--	--
0668	124.1	119.2	110.7	108.3	97.3	76.8	72.3	67.5	62.5
0669	112.8	104.5	94.1	--	--	--	--	--	--
Group 4									
0673	81.1	80.4	79.8	69.8	67.5	63.8	55.8	51.1	47.6
0675	120.0	112.5	92.6	84.9	71.6	--	--	--	--
0676	92.6	91.1	90.7	69.1	59.3	--	--	--	--
0677	106.6	96.8	80.2	75.5	71.2	66.4	57.5	--	--
0679	71.2	68.6	66.9	54.3	54.8	53.4	49.3	47.7	--
Group 5									
0678	90.4	83.5	74.6	69.5	68.8	64.9	65.7	65.8	67.9
0680	98.6	87.8	90.9	91.4	88.5	92.3	95.9	94.2	92.8

TABLE XIX. PERCENT OF CHANGE FROM STARTING WEIGHT

Calf Number	Number of Weeks on Experiment								
	0	1	2	3	4	5	6	7	8
Group 1									
C657	0	-1.4	-2.8	-8.5	--	--	--	--	--
C659	0	+4.0	+2.7	+1.3	-5.3	-4.0	-9.3	--	--
C660	0	-2.6	-6.5	--	--	--	--	--	--
Group 2									
C661	0	-4.4	-4.4	-4.4	-8.8	-17.6	--	--	--
C663	0	0	-14.3	--	--	--	--	--	--
C665	0	-13.3	--	--	--	--	--	--	--
C671	0	-7.8	--	--	--	--	--	--	--
C672	0	+3.2	--	--	--	--	--	--	--
Group 3									
C662	0	-3.8	-5.1	-3.8	--	--	--	--	--
C664	0	+1.0	+2.1	+6.2	--	--	--	--	--
C667	0	+2.2	+2.2	+4.4	+6.7	--	--	--	--
C668	0	+1.2	0	+4.7	0	-15.1	-14.0	-14.0	-15.1
C669	0	+2.8	-3.8	--	--	--	--	--	--
Group 4									
C673	0	+6.8	+8.2	+1.4	+2.7	+5.6	-1.4	-4.1	-5.6
C675	0	0	-12.5	-13.9	-23.6	--	--	--	--
C676	0	+5.7	+12.6	-8.0	-18.4	--	--	--	--
C677	0	-4.2	-15.6	-16.7	-17.7	-17.7	-23.9	--	--
C679	0	+4.5	+9.0	-6.0	+1.5	+6.0	+4.5	+7.5	--
Group 5									
C678	0	+1.2	-4.7	-3.5	+1.2	+2.4	+10.6	+17.6	+24.7
C680	0	-7.1	0	+5.7	+10.0	+20.0	+32.9	+40.0	+47.1

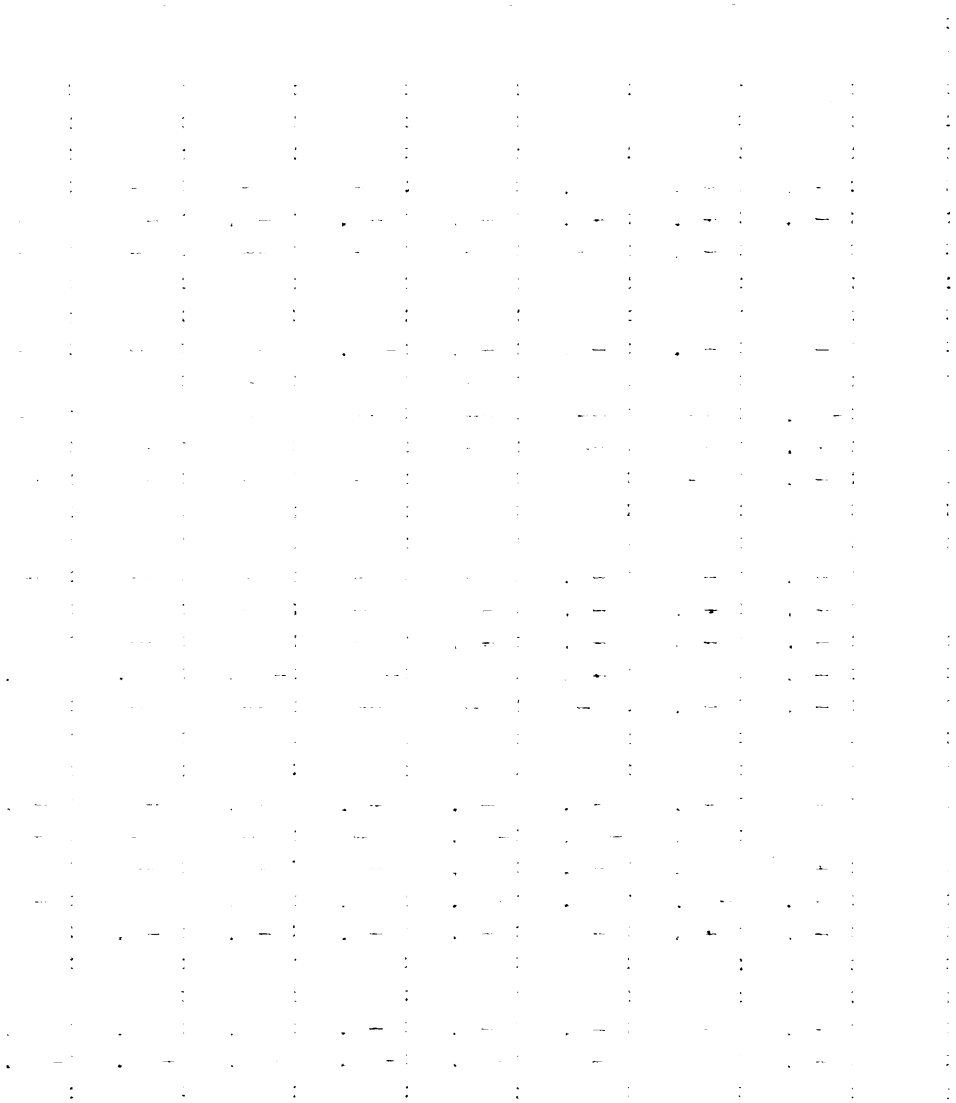


TABLE XX. WEEKLY BLOOD ANALYSIS OF C657

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	12.2	6.28	2.15	12.90	34.0	0.272
1	10.0	4.84	2.04	11.30	28.0	0.290
2	11.3	5.34	2.43	11.60	31.0	0.346
3	10.0	6.48	2.08	11.15	22.5	0.367
4	10.6	4.28	2.04	8.47	23.0	0.157

TABLE XXI. WEEKLY BLOOD ANALYSIS OF C659

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	12.3	5.90	2.40	10.13	27.0	0.389
2	11.9	6.01	1.90	10.50	28.0	0.214
3	10.3	6.19	1.75	13.25	32.0	0.278
4	9.1	5.48	1.18	9.70	24.0	0.328
5	8.9	5.02	1.63	8.40	22.0	0.271
6	9.8	5.12	1.87	9.20	22.0	0.235

TABLE XXII. WEEKLY BLOOD ANALYSIS OF C660

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	11.1	6.62	1.98	7.93	21.0	0.451
1	10.2	6.51	1.85	8.27	22.5	0.288
2	10.5	5.12	1.87	9.15	24.0	0.309

TABLE XXIII. WEEKLY BLOOD ANALYSIS OF C661

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	10.5	5.66	1.81	14.75	36.0	0.311
1	10.0	6.51	1.90	15.50	41.0	0.461
2	9.6	5.51	1.73	14.20	36.0	0.226
3	--	--	--	--	--	--
4	8.1	6.48	2.07	12.23	29.5	0.215
5	10.5	4.72	1.84	11.00	28.0	0.190

TABLE XXIV. WEEKLY BLOOD ANALYSIS OF C663

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	12.2	6.54	2.28	8.33	23.0	0.474
1	10.7	5.63	2.08	9.04	24.5	0.162
2	12.4	10.68	3.52	8.97	23.5	0.326

TABLE XXV. WEEKLY BLOOD ANALYSIS OF C665

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	10.7	6.48	2.06	9.33	25.0	0.442

TABLE XXVI. WEEKLY BLOOD ANALYSIS OF C671

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	10.8	6.41	2.02	11.67	31.0	0.440

TABLE XXVII. WEEKLY BLOOD ANALYSIS OF C 672

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	11.7	6.41	2.62	8.97	24.5	0.424

TABLE XVIII. WEEKLY BLOOD ANALYSIS OF C662

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	11.2	6.07	1.79	7.53	22.0	0.267
2	11.5	6.28	2.05	7.33	20.0	0.153
3	11.0	6.01	2.38	6.87	19.0	0.243

TABLE XXIX. WEEKLY BLOOD ANALYSIS OF C664

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma MG	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	11.5	6.07	3.00	12.80	35.0	0.407
2	11.3	5.37	1.51	12.00	31.5	0.364
3	10.5	6.41	1.70	11.07	27.5	0.397

TABLE XXX. WEEKLY BLOOD ANALYSIS OF C667

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	12.1	5.66	2.91	12.45	35.0	0.589
1	11.4	4.18	1.79	---	--	0.272
2	11.3	4.23	1.71	12.35	--	0.511
3	10.5	5.46	2.46	12.83	35.0	0.523

TABLE XXXI. WEEKLY BLOOD ANALYSIS OF C668

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	12.1	6.16	1.60	10.87	31.5	0.272
2	11.2	6.83	1.80	12.88	36.0	0.413
3	11.1	7.58	2.19	12.83	35.0	0.357
4	10.7	8.99	1.85	11.67	30.5	0.538
5	10.5	6.69	2.31	15.00	42.0	0.412
6	11.1	5.43	2.16	12.65	34.0	0.206
7	10.0	5.27	1.87	12.27	32.0	0.198
8	9.7	6.48	1.83	11.50	31.0	0.275

TABLE XXXII. WEEKLY BLOOD ANALYSIS OF C669

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	10.3	6.44	3.24	11.60	31.0	0.600

TABLE XXXIII. WEEKLY BLOOD ANALYSIS OF C673

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	10.3	5.00	2.32	16.00	43.0	0.266
2	9.3	5.27	2.05	15.20	42.0	0.268
3	--	--	--	--	--	--
4	10.1	5.81	2.09	14.63	45.0	0.156
5	10.2	4.75	1.81	11.93	34.0	0.198
6	9.2	6.25	1.60	11.40	33.5	0.276
7	9.9	5.95	1.96	10.80	32.5	0.235
8	9.5	6.58	1.77	9.03	25.5	0.249
9	10.5	5.93	2.21	8.97	25.0	0.364
10	9.2	5.95	1.84	8.20	22.5	0.167

TABLE XXXIV. WEEKLY BLOOD ANALYSIS OF C675

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	--	--	--	---	--	--
2	10.7	4.00	1.94	12.45	32.0	0.391
3	9.7	4.61	1.57	10.00	26.0	0.240
4	9.2	5.68	1.97	9.63	26.0	0.311

TABLE XXXV. WEEKLY BLOOD ANALYSIS OF C676

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	12.9	5.32	2.15	10.93	30.0	0.505
1	9.9	6.62	2.18	10.43	30.0	0.621
2	10.7	5.27	1.22	10.13	27.0	0.302
3	9.5	5.63	2.43	10.50	31.0	0.235

TABLE XXXVI. WEEKLY BLOOD ANALYSIS OF C677

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
1	10.5	5.74	1.10	15.90	42.0	0.455
2	8.5	5.63	2.15	16.20	44.0	0.261
3	8.9	7.67	1.86	15.50	40.0	0.249
4	9.1	6.28	2.28	16.10	42.0	0.305
5	8.2	5.68	1.97	15.40	41.5	0.271
6	8.4	5.56	1.20	14.55	36.0	0.229

TABLE XXXVII. WEEKLY BLOOD ANALYSIS OF C679

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	10.7	6.22	2.91	10.57	28.0	0.494
1	11.9	7.76	2.23	10.43	30.0	0.403
2	10.7	5.74	1.90	11.67	31.5	0.537
3	10.3	5.63	1.26	11.93	32.0	0.364
4	10.4	5.43	1.93	12.45	30.5	0.348
5	10.7	5.76	1.62	11.15	30.0	0.212
6	10.2	6.19	1.96	11.30	30.0	0.385
7	10.2	5.63	2.10	11.60	30.0	0.255
8	10.8	6.16	1.58	11.40	30.5	0.212

TABLE XXXVIII. WEEKLY BLOOD ANALYSIS OF C678

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	12.9	6.79	2.67	8.27	25.0	0.757
1	11.4	6.25	2.28	7.67	22.0	0.429
2	11.5	5.63	2.25	7.33	21.5	0.202
3	11.3	5.87	1.08	8.00	22.5	0.185
4	10.0	6.10	1.89	7.73	21.5	0.220
5	10.7	6.16	1.53	7.73	21.5	0.195
6	10.8	7.10	1.58	7.03	19.5	0.229
7	10.9	7.19	1.84	8.60	24.0	0.255
8	11.3	7.02	1.56	9.77	25.5	0.307

TABLE XXXIX. WEEKLY BLOOD ANALYSIS OF C680

Weeks on Expt.	Plasma Ca	Inorg. P	Plasma Mg	Hemoglobin	Cell Volume	Ascorbic Acid
	mg %	mg %	mg %	gm %	gm %	mg %
0	--	--	--	--	--	--
1	11.5	5.43	2.09	8.33	23.0	0.339
2	10.7	6.51	1.28	8.20	22.5	0.322
3	10.3	6.28	2.02	9.40	25.0	0.272
4	10.8	6.65	1.95	8.50	23.0	0.261
5	--	--	--	--	--	--
6	10.6	5.76	1.88	11.07	28.0	0.346
7	10.8	7.02	1.66	11.60	31.0	0.344
8	11.6	7.67	1.80	11.67	30.0	0.388

TABLE XXXX. SURVIVAL TIME OF CALVES ON SYNTHETIC RATIONS

Calf Number	Age When Placed on Synthetic Ration	Survival Time on Synthetic Ration
Group 1		
C657	31	45
C659	2	28
C660	8	21
Group 2		
C661	8	40
C663	5	17
C665	2	10
C671	4	8
C672	4	8
Group 3		
C662	4	25
C664	3	23
C667	4	30
C668	5	63
C669	3	14
Group 4		
C673	5	72
C675	3	28
C676	2	24
C677	2	48
C679	2	58
Group 5		
C678	2	90 +
C680	3	90 +

NOTES

Group 1

C657. This Jersey male calf was started on experiment at one month of age. The appetite was poor and the ration modified after the first week. From the start of the experiment the feces were quite soft and scouring was frequent. The calf became emaciated and died after 45 days on the experiment.

Necropsy. Stomachs and small intestine normal. Intussucepted cecum, ulcerated colitis and perforated ulcers of the large intestine; peritonitis. Glands, heart and lungs normal.

C659. Holstein female. Scoured from start of experiment. Emaciation but good appetite. Died after 28 days on experiment.

Necropsy. Large amount of feed in the rumen. Abomasum contained yellow fluid which showed a yellow precipitate. No apparent pathology of digestive tract above cecum. Cecum and large intestine were hemorrhagic on ridges. Condition most marked in cecum, decreasing down the large intestine. Thyroid, kidneys, liver and spleen normal. Bronchopneumonia of the apical, cardiac and a small area of the diaphragmatic lobes of lungs.

C660. Holstein male. Became listless and weak after two weeks, and died after 21 days on experiment.

Necropsy. Small amount of course material (hair and shavings) in the rumen. Distention and enteritis of the abomasum. Small intestine normal. Slight erosion near junction of cecum and large intestine. Slight hemorrhage on walls of cecum.

Group 2

C661. Holstein male. Developed stiffness of rear legs after two weeks on experiment, believed due to wire screen floor. Appetite poor during fourth week, and calf became emaciated. Died on 40th day.

Necropsy. Digestive tract normal with exception of slight congestion of abomasum. Kidney and liver normal. Death due to pneumonia.

C663. Holstein male. Scoured first week, intermittently thereafter. Emaciation and weakness developed at 10 days, and death followed at 17 days.

Necropsy. Rumen contained some shavings. Mucosa of abomasum showed petechial hemorrhages. Ulcer at opening between omasum and abomasum. Lower tract normal. Thyroid, brain, heart, liver, adrenals normal. Numerous white focal areas throughout both kidneys.

C665. Guernsey male. Appetite good but calf scoured and did poorly from the start. Blood in feces on 9th day. Died on 10th day.

Necropsy. Abomasum showed numerous petechial hemorrhages. Congestion of first four feet of duodenum. Remainder of tract normal. Lungs, heart, kidneys, liver, thyroid, thymus and brain normal.

C671. Deacon calf purchased at three days of age. Appetite poor; frequently ate only half feed. Died after eight days on experiment.

Necropsy. Severe hemorrhage and gastritis of abomasum. Enteritis throughout tract. Heart, lungs, brain, adrenals, kidneys and liver normal.

C672. Deacon calf purchased at three days of age. Died on 8th day without previous indication of malfunction.

Necropsy. Rumen contained about one pint of feed. Abomasal mucosa showed numerous petechial hemorrhages and marked edema. Small intestine normal. Mucosa of cecum thickened. Congestion of upper colon. Heart, liver, lungs, kidneys, adrenals and spleen normal.

Group 3

C662. Holstein female. Bloated on 7th day. Gas removed by stomach tube. Paralysis of rear and fore legs on 8th day. Also some bloat. Treated with 100 micrograms of biotin injected subcutaneously. Paralysis disappeared in 12 hours. Bloated again on 14th day. Rear and fore legs paralyzed on 15th day. Neck also involved. Treated with 20 grams potassium chloride orally. Some improvement in three hours. Complete recovery in 36 hours. Died on 25th day.

Necropsy. Digestive tract normal throughout. Organs normal. Death apparently due to progressive pneumonia.

C664. Holstein male. This calf did not scour except at time of paralysis. Developed paralysis of rear legs on 23rd day of trial. Died two hours after being given 20 grams of potassium chloride orally.

Necropsy. Localized congestion at numerous points in the small intestine, in general becoming more severe posteriorly. Cecum and colon normal. Hypertrophic catarrh in duodenum. Heart, lungs, liver, kidneys, rumen and abomasum normal.

C667. Jersey male. Scoured slightly first few days. Developed paralysis, involving rear and fore legs and neck, on 27th day. Also scours. Treated with oral administration of 15 grams of potassium chloride and subcutaneous injection of 100 micrograms of biotin. Recovery apparently complete in 24 hours. Calf died on 30th day of trial.

Necropsy. Moderate petechial hemorrhages of abomasum. Remainder of gastrointestinal tract normal. No other abnormalities except large amount of peritoneal fluid.

C668. Deacon calf received at about four days of age. Poor appetite first several days. No scours, but some emaciation after fourth week. Developed paralysis on 63d day, involving legs, head and neck. At time of discovery the limbs and nose were cold. Calf was sacrificed for autopsy.

Autopsy. Rumen well developed but contents dry. Slight enteritis of abomasum. Duodenum and lower tract in good condition with no abnormalities. Heart, lungs, thyroid, kidneys, liver and adrenals normal. Open foramen ovale.

C669. Holstein calf. Calf normal until overall weakness and paralysis of rear legs developed on 5th day. Anorexia accompanied paralysis. Treated by three 10 gram doses of potassium phosphate at 12 hour intervals. Recovery complete in 36 hours. Bloated and died on 13th day.

Necropsy. Severe gastroenteritis throughout with exception of rumen and duodenum. Severe hemorrhage of abomasum. Apparently normal outside gastrointestinal tract.

Group 4

C673. Holstein female. Excellent condition throughout the first month. Occasionally bloated slightly after feeding. Mild scours during the sixth week. Severe scours on 71st day. Found dead on morning of 72nd day of trial.

Necropsy. Gastrointestinal tract normal throughout. Rumen functional and contained about one gallon of semisolid feed. Some edema of tissues, otherwise normal throughout.

C675. Jersey male. Rather severe scours the fourth day on the experiment. Feces normal by the sixth day. Scoured one day of second week and two days of third week. Gradually became dehydrated and emaciated. Died on the 28th day.

Necropsy. The rumen contained about one pint of very dry material, largely hair and shavings. Slight enteritis in the abomasum. Lower tract showed only very slight irritation. Lungs, heart, brain, thyroid, adrenals, kidneys and liver normal.

C676. Holstein female. Became weak and emaciated after five weeks. Progressively worse until death occurred on 48th day.

Necropsy. The only abnormality found was the presence of fluid in the peritoneal and pleural cavities.

C679. Holstein male. Normal feces. Calf did well first several weeks, then began gradual decline ending in death at 58 days.

Necropsy. No abnormalities whatsoever.

Group 5

C678. Holstein male. This calf developed anorexia and became weak and unable to stand during the second week. Thiamin therapy resulted in a return to normal, although the growth rate was slow for several weeks. Removed from experiment at 90 days. Normal in all respects although rather light in weight for a calf of that age.

C680. Guernsey male. Failed to grow the first three weeks, then showed normal growth curve until removed from experiment at 90 days. No scours or other abnormalities encountered during the experimental period.