THE pH OF THE CONTENTS OF THE GASTROINTESTINAL TRACT IN DOGS, IN RELATION TO DIET AND RICKETS.

By DAVID M. GRAYZEL and EDGAR G. MILLER, Jr.

(From the Laboratory of Biological Chemistry of Columbia University, College of Physicians and Surgeons, New York.)

(Received for publication, November 28, 1927.)

That the reaction of the intestinal contents may be an important factor in certain abnormalities of inorganic metabolism is evident from the researches of several workers. Zucker and Matzner (41) and, later, Jephcott and Bacharach (16), found that the feces of albino rats were alkaline under a rachitic régime, but were acid normally and with a rickets-producing diet plus curative procedures. Abrahamson and Miller (1) found the same type of changes in the contents of the intestines of albino rats. Recently Redman, Willimott, and Wokes (24) confirmed these findings on rats and guinea pigs, and Yoder (37) on rats.

In the experiments reported in the present paper, dogs were used. The diets provided were as follows, used *ad libitum*.

Diet 1.—125 gm. of lean meat, 100 gm. of cracker meal, 15 gm. of lard, 30 gm. of bone ash, 300 cc. of water. This is a routine normal diet.

Diet 2.—25 gm. of meat, 150 gm. of cracker meal, 30 gm. of lard, 30 gm. of bone ash, 350 cc. of water. This diet is relatively high in carbohydrate and low in protein.

Diet 3.—Lean meat—a high protein diet.

Diet 4.—50 gm. of meat, 50 gm. of cracker meal, 180 gm. of lard, 30 gm. of bone ash. This provides a high fat diet.

Diet 5.—175 cc. of whole milk, 200 gm. of white bread, 2 gm. of NaCl. This is Mellanby's Diet 2 (21) with which he produced rickets in puppies.

Diet 6.—10 cc. of cod liver oil added to Diet 5.

The dogs were fed the diet for several days previous to the experiment. In the cases of the Mellanby diet, and the modifications of this diet, the feeding was continued for from 3 to 6 weeks.

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The irradiation by ultra-violet light was by means of a quartz mercury vapor lamp (for the use of which we are indebted to Dr. A. F. Hess and the Department of Pathology of Columbia University) for periods as indicated in the discussion below.

Observations made previously led us to the practice of killing the dogs from 4 to 6 hours after the last feeding. At this time the intestinal digestion is at its height. When the canal is empty of food, the reaction of the secretions (2) may give a very different result. The gastric reaction varies with the stage of digestion, and, to a lesser degree, the duodenal reaction is shifted by the ingress of the material from the stomach. But in the intestine, during the time when the food mass is present, there is no sharp cyclic change in reaction.

The dogs were killed by the injection of a solution of Lilly's amytal (isoamylethylbarbituric acid) into the jugular vein. It was found that about 30 cc. of a 10 per cent solution when so injected produced instantaneous death, the heart and respiration stopping together. The amytal was used as a freshly prepared solution.

Upon the death of the dog a median abdominal incision was immediately made and the alimentary tract exposed. The various parts of the tract were then quickly ligated and removed. For our purpose we used the stomach, duodenum, cecum, colon, and 2-foot sections of the ileum and jejunum. The contents of each section were expressed gently into centrifuge tubes, several drops of pure mineral oil added, and the tubes centrifuged for half an hour. The pH was then determined. The colorimetric procedure was carried out by the use of the Clark and Lubs (6) indicators and the Sörensen standard buffer solutions (citrate-HCl and phosphate mixtures) which had been checked electrometrically. The electrometric method was also used for 156 of the determinations (twenty-one dogs), the Clark electrode and the Leeds and Northrup laboratory hydrogen ion potentiometer being employed.

Whenever the volume of fluid was insufficient for direct determination of pH, distilled water was added before treating the tubes as described above. A series of determinations on diluted and undiluted samples showed that the dilution did not materially alter the pH. This confirmed the observations of Davidsohn (7)

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and others that in the case of heavily buffered solutions of low acidities dilution affects the pH very little.

In work on the pH of gastric contents, Kahn and Stokes (17) reported the variations between the colorimetric and the electrometric results to be sufficiently large and inconstant to make the colorimetric results unreliable. In work on twenty-one dogs involving 156 different comparisons of the results by the two methods, we found the agreement quite close and consistent between the colorimetric and the electrometric results. The colorimetric result was, in every case except one, a lower pH than the electrometric, the average difference being 0.2 of a pH unit, the maximum difference being 0.39, and the minimum 0.1; if we omit the stomach (as the greatest error has been shown to be in the higher ranges of acidity) the maximum difference was 0.33. In 155 of the 156 determinations, it should be noted, the errors are minus. We therefore believe that we are justified in drawing conclusions from relatively large changes in pH, under different conditions in the dog, as determined colorimetrically.

The averages are recorded as the pII values for the corresponding average C_H. In the operations on the dogs, as we wished to avoid too much mixing of the intestinal contents and delay in removal, we could not measure the length of the whole gut before ligating; we therefore divided it into lengths of approximately 2 feet, thus obtaining a varying number of sections from dogs of different sizes. In recording averages, we have arbitrarily grouped the sections to represent approximately the upper, middle, and lower ranges of the small intestine below the duodenum.

Ten dogs were put on Diet 1, the routine normal diet as used in this laboratory. As seen from Table I, the reaction of the intestinal contents is on the acid side of neutrality practically throughout the entire length of the tract, the cecum and colon not showing the same constancy of reaction as do the upper levels of the tract. The intestinal contents of Dog 1 are somewhat more alkaline in reaction than those of the other dogs on the same diet, perhaps due to the fact that this dog was operated upon 18 hours after the last feeding, whereas in the case of the other dogs the operations took place from 4 to 6 hours after they were last fed.

Robinson (25) in his work on the reaction of stools under various conditions found that a preponderance of fat or carbohydrates

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gave an acid stool, whereas with heavy meat diets the reaction of the stools tended to be alkaline. Howe and Hawk (12), however, observed that the reaction of the stools was uniformly alkaline and that pronounced changes in the dietary régimes, such as high

TABLE I.

pH of Gastrointestinal Contents of Dogs on Normal Diet.

Dog No.	Sex.	Weight.	Stomach.	Duodenum.	First 2 ft.	Second 2 ft.	Third 2 ft.	Fourth 2 ft.	Fifth 2 ft.	Sixth 2 ft.	Cecum.	Colon.
		kg.										
1	M.	12.0	3.6	6.4			6.6				6.8	7.0
2	"	12.0	3.8	5.9	5.9	6.0	6.2	6.4			6.8	7.2
3	"	9.3	3.6	5.8	5.9	6.2	6.2				6.8	7.0
17	"	12.0	3.9	6.0	6.2	6.0	6.2	6.2	6.5		6.8	7.2
22	F.	8.7	4.3	6.0	5.9	5.7	6.1				6.5	6.7
23	"	9.0	4.2	6.2	6.0	6.2	6.4				6.5	6.7
24	"	8.6	4.3	5.8	6.1	6.0	6.2	6.2			6.6	7.0
28	M.		3.2	5.8	6.2	6.0	6.4	6.2	6.2	6.4	6.8	7.2
29	"		3.5	5.8	6.2	5.9	6.1	6.2			6.2	6.4
30	"		3.0	5.8	5.9	6.0	6.4	6.4			6.4	6.8

Averages: stomach 3.68, duodenum 5.91, upper level, small intestine 5.99, middle 6.27, lower 6.36, cecum 6.57, colon 6.84.

TABLE II.

pH of Gastrointestinal Contents of Dogs on High Carbohydrate Diet.

Dog No.	Sex.	Weight.	Stomach.	Duodenum.	First 2 ft.	Second 2 ft.	Third 2 ft.	Fourth 2 ft.	Cecum.	Colon.
		kg.								
4	F.	12.1	3.7	5.9	5.9	6.0	6.2	6.2	6.6	7.0
5	M.	12.2	3.9	5.9	5.9	6.0	6.2		6.4	6.8
6	F.	10.0	4.2	5.8	5.9	6.0			6.6	7.2

Averages: stomach 3.93, duodenum 5.87, upper level 5.90, middle 6.03, lower 6.20, cecum 6.53, colon 7.00.

or low proteins and fasting, did not affect it much. Cannon and McNease (4) and Hudson and Parr (13) found that on a high protein diet the feces had a pH of 7 to 7.1, and on a high lactose diet a

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pH of 6.2. Abrahamson and Miller (1) found no notable variation in the intestinal contents of albino rats.

Three dogs were put on Diet 2, two on Diet 3, and two on Diet 4. The results of the experiments are tabulated in Tables II, III, and IV. As seen from the tables the reactions under these conditions fall practically within the normal range.

To find how the reaction of the intestinal contents of dogs varied along the length of the tract on a rickets-producing diet,

TABLE III.

pH of Gastrointestinal Contents of Dogs on High Protein Diet.

Dog No.	Sex.	Weight.	Stomach.	Duodenum.	First 2 ft.	Second 2 ft.	Third 2 ft.	Fourth 2 ft.	Fifth 2 ft.	Cecum.	Colon.
		kg.									
15	M.	10	4.2	6.1	6.4	6.4	6.3	6.4	6.6	7.0	7.4
16	F.	10	3.7	5.9	6.2	6.0	6.2	6.2		6.8	7.2

Averages: stomach 3.95, duodenum 6.00, upper level 6.30, middle 6.23, lower 6.45, cecum 6.90, colon 7.30.

TABLE IV.

pH of Gastrointestinal Contents of Dogs on High Fat Diet.

Dog No.	Sex.	Weight.	Stomach.	Duodenum.	First 2 ft.	Second 2 ft.	Third 2 ft.	Fourth 2 ft.	Cecum.	Colon.
		kg.								
18	M.	9.5	3.5	5.8	5.8	5.9	6.2		6.4	6.8
19	"	10.2	3.5	5.7	5.8	5.6	5.9	6.2	6.4	6.8

Averages: stomach 3.50, duodenum 5.75, upper level 5.80, middle 5.85, lower 6.20, cecum 6.40, colon 6.80.

six dogs were put on Diet 5. This corresponds to Mellanby's Diet 2 with which he produced rickets in puppies. That the reaction tends to go to the alkaline side of neutrality is evident from Table V.

According to Mellanby (21), 10 cc. of cod liver oil when added to his rickets-producing diet were sufficient to prevent rickets in puppies. It was interesting to note whether or not the reaction

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of the intestinal contents would also change to its normal value. For this purpose we put four dogs on Diet 6, which is Diet 5 (the Mellanby diet) plus 10 cc. of cod liver oil. The results of this experiment are given in Table VI. Here we note that with the

TABLE V.

pH of Gastrointestinal Contents of Dogs on Rickets-Producing Diet.

Dog No.	Sex.	Weight.	Stomach.	Duodenum.	First 2 ft.	Second 2 ft.	Third 2 ft.	Fourth 2 ft.	Fifth 2 ft.	Cecum.	Colon.
		kg.									
7	F.	12.6	3.4	6.6	7.0	6.8	7.0	7.0	7.2	7.4	7.4
8	M.	11.8	3.6	7.0	7.4		7.2	7.4		7.5	7.7
25	F.	8.6	3.9	6.4	6.6	6.8	7.0			7.2	7.4
26	"	4.3	3.7	6.6	6.8	7.2	7.0			7.2	7.4
31	"	7.5	3.9	6.8	7.0	6.6	7.2			7.2	7.4
32	M.	8.0	4.2	6.4	6.6	7.0	6.8			7.2	7.4

Averages: stomach 3.71, duodenum 6.59, upper level 6.81, middle 6.88, lower 7.05, cecum 7.27, colon 7.44.

TABLE VI.

pH of Gastrointestinal Contents of Dogs on Rickets-Producing Diet Plus Cod Liver Oil.

Dog No.	Sex.	Weight.	Stomach.	Duodenum.	First 2 ft.	Second 2 ft.	Third 2 ft.	Fourth 2 ft.	Fifth 2 ft.	Cecum.	Colon.
		kg.									
9	M.	10.2	3.0	5.9	6.0	6.0	6.2			6.6	6.8
10	"	11.2	3.4	6.0	5.9	5.9	6.2	6.2	6.4	6.4	6.6
35	F.	7.0	3.9	5.9	5.8	6.2	6.0			6.2	6.4
36	M.	7.5	4.2	6.1	6.0	5.8	6.0			6.2	6.4

Averages: stomach 3.40, duodenum 5.97, upper level 5.92, middle 6.02, lower 6.11, cecum 6.32, colon 6.52.

preventive value of the cod liver oil we have the reaction of the intestinal contents again becoming more acid.

That irradiation with ultra-violet light cures rickets has been known for some time, especially through the work of Hess (10) and of Steenbock and Black (31). Six dogs were put on Diet 5

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and kept on it for several weeks, after which they were subjected to irradiation with ultra-violet light, two dogs being subjected to a single irradiation of 1 hour, and four to a daily irradiation of 1 hour for 3 consecutive days.

Previous tests showed that the reaction of the stools in dogs on

TABLE VII.

pH of Gastrointestinal Contents of Dogs on Rickets-Producing Diet, with Ultra-Violet Irradiation.

Dog No.	Sex.	Weight.	Stomach.	Duodenum.	First 2 ft.	Second 2 ft.	Third 2 ft.	Cecum.	Colon.
		kg.							
11	F.	12.1	3.8	5.9	6.0	6.4	6.2	6.8	7.2
12	"	9.8	3.9	6.1	6.2	6.4	6.6	7.0	7.4
27	"	5.0	3.4	5.9	6.1	6.2	6.2	6.6	6.8
33	M.	8.4	3.5	6.0	5.9	6.0	6.2	6.4	6.8
34	"	6.5	3.7	6.1	6.0	6.0	6.2	6.6	6.8
37	F.	5.0	3.9	6.1	6.3	6.2	6.4	6.6	6.8

Averages: stomach 3.66, duodenum 6.01, upper level 5.99, middle 6.17, lower 6.28, cecum 6.63, colon 6.91.

TABLE VIII.

pH of Gastrointestinal Contents of Dogs on Normal Diet Plus Irradiation.

Dog No.	Sex.	Weight.	Stomach.	Duodenum.	First 2 ft.	Second 2 ft.	Third 2 ft.	Fourth 2 ft.	Cecum.	Colon.
		kg.								
38	M.	8	4.2	6.0	5.9	5.8	6.2	6.4	6.2	6.8
39	"	15	4.2	6.2	6.5	6.3	6.6	6.8	6.6	7.0
40	"	12	3.7	5.9	6.2	6.5	6.4		6.8	6.8
41	"	10	3.5	5.6	5.9	6.2	6.0		6.2	6.4
42	F.	7	3.4	5.3	5.9	6.2	6.4		6.2	6.8

Averages: stomach 3.68, duodenum 5.68, upper level 6.03, middle 6.14, lower 6.30, cecum 6.34, colon 6.70.

a regular mixed diet was around 7.2. An analysis of the stools of the first two dogs a few days before irradiation (after 3 weeks on the Mellanby diet), showed pH 8.0 for one and pH 8.2 for the other. The day following subjection of the dogs to the mercury quartz lamp, the reactions of the stools were pH 7.2 and pH

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7.4 respectively. The animals were therefore killed and studied on that day. That, with the curative action of the ultra-violet light, the reaction of the intestinal contents tends to be more on the acid side is evident from Table VII. Dog 12 in this experiment showed marked signs of malnutrition.

TABLE IX.

Averages of pH of Gastrointestinal Contents.

No. of dogs.	Diet.	Stomach.	Duodenum.	Upper small intestine.	Middle small intestine.	Lower small intestine.	Cecum.	Colon.
10	Normal.	3.68	5.91	5.99	6.27	6.36	6.57	6.84
3	High carbohydrate.	3.93	5.87	5.90	6.03	6.20	6.53	7.00
2	" fat.	3.50	5.75	5.80	5.85	6.20	6.40	6.80
2	" protein.	3.95	6.00	6.30	6.23	6.45	6.90	7.30
6	Rachitic.	3.71	6.59	6.81	6.88	7.05	7.27	7.44
4	" + cod liver oil.	3.40	5.97	5.92	6.02	6.11	6.32	6.52
6	" + irradiation.	3.66	6.01	5.99	6.17	6.28	6.63	6.91
5	Normal + "	3.68	5.68	6.03	6.14	6.30	6.34	6.70

TABLE X.

Average $C_H \times 10^{-8}$ of Gastrointestinal Contents.

	Normal.	Rachitic.	Rachitic + irradiation.	Rachitic + cod liver oil.
No. of dogs	10	6	6	4
Stomach	168,230	19,500	61,300	264,677
Duodenum	123±29	26±12	99±15	108±13
Upper small intestine	98±20	15±8	86±17	121±16
Middle " "	82±29	12±7	68±16	96±26
Lower " "	56±8	9±4	53±10	78±15
Cecum	27±10	4±1	24±6	48±11
Colon	14±7	4±1	12±8.6	30±6.9

In passing, it is interesting to note (though the relation to this phenomenon is only speculative) that Liechti (19) found an increased local subcutaneous acidity, lasting for several days following ultra-violet irradiation.

Five dogs were put on the normal diet (No. 1) and subjected to irradiation as in the case of the dogs on the Mellanby diet.

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The irradiation caused no apparent lowering of the intestinal pH below the normal figures (see Table VIII).

Table IX presents a summary of these data. In Table X the data for the normal, rachitic, irradiated, and cod liver oil series are presented in terms of the non-logarithmic expression $C_H \times 10^{-8}$, with probable errors.

To test the validity of our conclusion that the series on the rickets-producing diet showed a real difference from the series on the normal diet and the series on the rachitic diet plus irradiation,

TABLE XI.

Average $C_H \times 10^{-8}$ of Intestinal Contents, with Differences and Probable Errors of Differences.

	Averages.			Difference.		Probable error of difference.	
	N.	R.	I.	N.-R.	I.-R.	N.-R.	I.-R.
Duodenum	123	26	99	97	73	9.8	6.9
Upper small intestine	98	15	86	83	71	7.2	7.7
Middle " "	82	12	68	70	56	9.7	7.2
Lower " "	56	9	53	47	44	3.0	4.5
Cecum	27	4	24	23	20	3.2	2.5
Colon	14	4	12	10	8	2.3	3.6

N. = normal diet; R. = rachitic diet; I. = rachitic diet plus irradiation.

we have calculated, from the data as shown in Table X, the probable errors of the differences

$$P.\ E._{(M_1 - M_2)} = 0.6745 \sqrt{(\delta_{M_1})^2 + (\delta_{M_2})^2}$$

(Chaddock (5)) for comparison with the differences of the means of the series. These data are shown in Table XI. Similar data for the other series are not shown, since with a smaller number of animals in a series the calculation of the deviations becomes less reliable. The results with them, however, are similar throughout.

DISCUSSION.

A study of Tables I to XI shows that the pH of the contents of the intestinal tract of a dog on a normal diet, 4 to 6 hours after the last feeding, was found to be remarkably constant at the various levels studied. There was a slow but steady rise in

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average pH in descending levels. The reaction remained acid throughout the small intestine. In the large intestine the contents may be slightly acid or slightly alkaline.

When the diet was varied so as to alter the proportion of carbohydrate, fat, or protein, the reaction in the alimentary tract remained essentially normal. The high fat diet, as might be expected, tended to give reactions in the more acid range of the normal variations.

Although ordinary variations of the normal diet were not effective in altering the alimentary pH, the use of a diet which is known to produce rickets in puppies (though, as we were working with adult dogs, we obtained no clinical rickets) gave results which were strikingly different from the normal picture. The reaction of the gastric contents fell well within the same range as in the normal dogs. However, since our experiments were aimed more directly at the intestinal conditions, we had chosen a postfeeding interval at which the intestinal rather than the gastric digestion was at its height, so that we do not feel that we have ruled out the possibility of gastric alteration. In the intestines, the animals on the rickets-producing diet showed a definite and distinct tendency to a pH of contents greater than in the normal animals. The difference between the normal and the rachitic animal is evident throughout the tract below the stomach, but is rather more striking in the small intestine than in the large intestine. This variation can perhaps be explained by the fact that in the cecum and colon there is a greater superimposition of bacterial changes tending to mask, in some degree, the physiological conditions.

In the group of dogs on the rickets-producing diet, which were also given ultra-violet irradiation or cod liver oil—each of which is known to have, in some way, preventive or curative effects on rickets—the intestinal pH was found again to be very different from that obtained on the rachitic diet alone, and approaching closely or reaching the normal figures.

That the pH of the medium must have an important influence on the solubility of calcium and phosphate (and, also, of other ions, such as iron and magnesium), is well known. Also, the obvious belief that the solubility of these materials in the intestine

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influences their absorption and their ultimate loss has much direct experimental evidence to support it (3, 14, 15, 36).

That the ingestion of excess acid or alkali will affect the inorganic metabolism of the body, especially as to calcium and phosphate, is known from the work of Zucker (38). It has also been shown that the dietary ratios of amounts of mutually precipitating substances (such as calcium and phosphate, calcium and fatty acid) will affect their utilization in the body. There is evidence (40) to show that the addition of sodium carbonate or ammonium chloride to diets which are on the border line between rachitic and non-rachitic may make them, respectively, rickets-producing or not rickets-producing and that additions of ammonium chloride to the diet may prevent tetany (32, 34, 35), which is a disease frequently associated with calcium deficiency. In these instances the possible relation between pH, solubility, and inorganic metabolism seems fairly evident.

That the various antirachitic measures affect the absorption of calcium or phosphate or both from the intestine is shown by the work of Shabad (27, 28), Lesue, Turpin, and Zizine (18), Henderson (9), Mitchell and Johnson (22), Orr, Holt, Wilkins, and Boone (23), Telfer (33), Yoder (37), and others. The relation of this absorption and rickets to intestinal reaction has been supported by the researches of Zucker (39), Zucker and Matzner (41), Abrahamson and Miller (1), Jephcott and Bacharach (16), Redman, Willimott, and Wokes (24), and Yoder (37).

The curative effect of ultra-violet light is clearly independent of any addition of material; its effect seems to be registered indirectly through some change in a body substance (probably cholesterol (11)). The question of how such a change affects the ossification is of great importance. It seems unlikely, from the researches of several investigators (26, 29, 30) that the rachitic defect lies altogether in the ossifying tissues themselves. The results reported here are, we believe, further evidence in favor of the idea that the primary defect in rickets, which is corrected under ultra-violet treatment, may be the absorption of phosphate (or calcium, or both) from the gut, with the hydrogen ion concentration as a limiting factor.

Again, the cod liver oil has a profound effect on the inorganic

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metabolism, in spite of the fact that it adds no inorganic material. The effect of the oil on the intestinal pH, and its effect on the calcium and phosphate balance of the body (3, 27, 28) lead us to the same conclusion. Here, too, however, the mechanism whereby the cod liver oil causes the change in the intestine is a matter of speculation. It is in all probability not related to the reaction of the products of digestion *per se*, since *in vitro* digestion of the various normal, rachitic, and curative diets by alimentary enzymes gives mixtures of approximately the same pH, and since other fats do not give the same physiological result *in vivo*. Furthermore, neutral concentrates have the same curative action.

It is probable, though not now demonstrable, that the pH factor is to a certain extent, at least, primary in these phenomena. The speed with which the irradiation alters the reaction of the intestinal contents after a long period on a rickets-producing diet suggests that this change comes early among the changes associated with the cure. The apparent universality of the pH relationship in the cases studied also tends to support this view.

It is perhaps idle at present to speculate as to the probable physiological mechanism by which these changes occur. It may be a change in the reaction of the digestive secretions in the stomach or intestine or both; it may be an altered absorption of acidic or basic materials from the gut. These changes may be due to nerve control or to direct action of some substance on the epithelial cells involved. The fact that the intestinal tract seems to be concerned to some degree in the maintenance of the acid-base equilibrium of the body (8, 20) suggests that systemic conditions may possibly be involved, changing the acidic or basic excretions into the gut. Yoder (37), testing a small number of rats and finding that irradiation of the rachitic rats affected the intestinal pH only below the duodenum, suggests this as evidence of excretion of acid or acid-producing substance into the gut. We have found, however, in the dog, that the duodenum shows the same type of change as do the lower parts of the tract.

SUMMARY.

1. The reaction of the intestinal contents of dogs on a normal mixed diet is acid practically throughout the entire length of the

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tract, the cecum and colon showing somewhat less constancy in the results than the other levels of the tract. The pH of the small intestine varies between 5.7 and 6.6.

2. The reaction of the intestinal contents of dogs fed high fat, protein, or carbohydrate diets is practically within the normal range for each level of the tract.

3. On Mellanby's diet, known to produce rickets in puppies, the reaction of the intestinal contents of dogs tends to be more alkaline. The pH of the small intestine varied from 6.4 to 7.4.

4. The addition of cod liver oil to Mellanby's diet changes the intestinal reaction back to its normal range.

5. Irradiation with ultra-violet light tends to bring the reaction of the intestinal contents of dogs on a rickets-producing diet back to the normal values.

6. Irradiation of dogs on a normal diet with ultra-violet light causes no apparent change in the alimentary pH.

7. These facts are offered as further evidence that changes in the reaction of the alimentary contents are (through the effect on solubilities of various materials and hence on their absorption) an important factor in alterations of inorganic metabolism and especially rickets.

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David M. Grayzel and Edgar G. Miller, Jr.

J. Biol. Chem. 1928, 76:423-436.

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