sium on germination and growth is due in part to its radioactivity. It is an interesting speculation, therefore, that the enhanced K^{40} content may have been a contributing factor to the carboniferous age.

The writer is especially indebted to Dr. R. C. Wells, of the U. S. Geological Survey, for suggesting the necessity for these calculations, and to Dr. A. Bramley and Dr. W. E. Deming for suggestions and for checking the calculations.

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A POSSIBLE ACID SEED SOAK FOR THE CONTROL OF BACTERIAL CANKER OF THE TOMATO

THE discovery that bacterial canker of tomato (*Aplanobacter michiganense* E. F. S.) may be controlled by fermenting the fruit pulp prior to seed extraction[2] has led to a study of the toxicity of fermenting pulp to the causal organism. Toxicity tests made by a method corresponding to the one used by McCown[3] demonstrated an unquestionable toxic action of the fermenting fruit pulp upon the bacterial canker pathogen. In several tests the pathogen maintained its viability in unfermented juices for from 60 to 100 hours and in 96-hour fermented juices for only one half to two and one half hours.

Distillates obtained from juices fermented for 240 hours and lethal to the pathogen in less than one hour were neutralized by the addition of barium hydroxide and their toxicity tested. The pathogen remained viable in the neutralized juices for 2,180 hours. In a similar experiment the barium salts were acidified with sulfuric acid, using methyl orange indicator and the barium sulfate removed. The resulting freed acids, when adjusted to the original volume, were as toxic as the untreated distillates.

Analysis of the fermented juices revealed that acetic and lactic acids were the acids formed most abundantly during fermentation. From .35 to .58 per cent. acetic acid and from .45 to .72 per cent. lactic acid were usually found in fruit juices which had fermented for a 96-hour period. A preliminary test of the effectiveness of those acids as seed soaks in the control of bacterial canker was undertaken.

Seed was extracted from fruit picked from plants infected with bacterial canker and aliquot parts soaked in .15 per cent., .3 per cent. and .6 per cent. acetic acid solutions for 3, 6, 12, 24, 48 and 96 hours. A similar series was soaked in .3 per cent., .6 per cent. and 1.2 per cent. lactic acid solution and in combinations of the respective concentrations of the two acids for the same periods of time. For comparison, aliquots of seed from the same lot were treated with copper sulfate 1 pound to 8 gallons for 21½ hours, with mercury bichloride 1–1,000 for ten minutes and with hot water 54 degrees centigrade for one hour. In order to compare the effectiveness of the seed treatments with fermentation, a portion of the pulp of the same infested fruit material was set aside to ferment for 96 hours and the seed then extracted. All treatments were applied immediately following extraction and before the seed had an opportunity to dry. Germination tests revealed that none of the treatments were particularly injurious. The seed was not milled and cleaned before the germination tests were made and for that reason there was a greater variation in the germination of different samples and a generally lower germination than is usually observed in first grade seed.

Representative amounts of seed of each treatment were planted in the field during the summer of 1936, and records taken of the number of diseased plants which developed from the seed of each treatment. The results are summarized in Table I. The various concentrations of each acid and the combination of acids for the various treatment durations are grouped together to condense the table.

TABLE 1

THE EFFECT OF ACID, CHEMICAL, HOT WATER AND FERMENTATION SEED TREATMENTS ON SEED GERMINATION AND THE CONTROL OF BACTERIAL CANKER

Treatment	Plants		Per cent.	
	Tested	Diseased	Diseased	Germination
Untreated, immediate extraction	1,336	1,086	81.28	90.0
Acetic acid soaks (all treatments)	3,751	3	0.08	87.1
Acetic and lactic acid soaks (all treatments)	4,003	9	0.225	83.38
Lactic acid soaks (all treatments)	4,140	26	0.62	82.97
$CuSo_4$ 1 lb.—8 gal. 21½ hours	418	26	6.22	81.0
$HgCl_2$ 1–1000—10 minutes	517	32	6.19	91.5
Hot water 54° C.—1 hour	427	33	7.73	93.5
Fermentation 96 hours	532	1	0.188	90.0

Two of the three plants which developed the disease following the acetic acid treatment were from the seed lot treated with an acid concentration of .6 per cent. for 6 and 96 hours, respectively, and the other one from seed treated with a .15 per cent. acid concentration for 96 hours. In the lactic and combination lactic and acetic acid series, the greatest amount of disease developed from seed lots treated with the lower acid concentrations for shorter durations of time. No canker developed in any of the 1,147 plants grown from seed treated with a combination of .6 per cent. acetic acid and 1.2 per cent. lactic acid for any duration.

[1] Authorized by the director of the Bureau of Plant Industry on March 6, 1937.

[2] H. L. Blood, *Proc. Utah Acad. of Sci.*, 10: 19–23, 1933.

[3] Monroe McCown, *Phytopath.*, 19: 285–293, 1929.

The results of the preliminary test indicate that the acetic acid, when alone or in combination, offers promise as a safe and effective tomato seed soak for the control of bacterial canker. Further studies to establish limits of concentration and effective schedules for the treatment are in progress.

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VITAMIN B_1 AND THE SYNTHESIS OF FAT FROM CARBOHYDRATE

IN recent years it has become clear that vitamin B_1 is concerned with carbohydrate metabolism. Professor R. A. Peters[1] has ably reviewed the evidence which substantiates this belief, and he has provided a theory for the action of the vitamin in preventing or curing polyneuritis. As stated by Peters, "This vitamin is a catalyst used by the tissue at some stage in the combustion of carbohydrate. Defect in this stage within the central nervous system will lead readily to convulsions."

The theory of the action of vitamin B_1 as defined to date is, then, that carbohydrate metabolism proceeds to the pyruvic acid stage but is there halted in the absence of vitamin B_1. In the presence of the vitamin the pyruvic acid is oxidized and energy production from carbohydrate is normal in amount. Emphasis has been placed upon the action of vitamin B_1 as a catalyst necessary for the combustion of carbohydrates. There is no doubt that the vitamin permits the oxidation of pyruvic acid by brain tissue *in vitro* and the explanation may be completely satisfactory for that tissue. More recent evidence has indicated that the vitamin has as a principal function in the body generally the synthesis of fat from carbohydrate.

Whipple and Church[2] have shown that the main factor in the weight increases due to vitamin B_1 in rats is the laying down of fat, and, in their experiments, the only possible source of this fat was the dietary carbohydrate. Further evidence was provided by them[3] in measurements of respiratory quotients that carbohydrate is transformed into fat under the influence of vitamin B_1. The writer has confirmed[4] the production of fat from carbohydrate in the presence of the vitamin.

At this stage an hypothesis regarding the action of vitamin B_1 might be advanced, based, it is true, upon incomplete evidence. Whether or not the vitamin is supplied, it has been generally accepted that carbohydrate metabolism proceeds to the pyruvic acid stage. In the absence of the vitamin pyruvic acid accumulates as has been shown in pigeons, rats and in human subjects by a number of workers. In the presence of the vitamin fat is synthesized, presumably with pyruvic acid as an intermediary stage between carbohydrate and fat, although this is as yet unproven. It has long been felt that this possibility exists and recently Krebs and Johnson have shown[5] that hydroxy butyric acid can be formed from pyruvic acid by tissues.

This hypothesis, which attempts to correlate various pieces of published evidence, is that vitamin B_1 is necessary for the synthesis of fat from carbohydrate. It does not weaken the belief that vitamin B_1 is concerned with carbohydrate metabolism but alters the conception of the vitamin being a factor in energy production from carbohydrate to a broader view of carbohydrate utilization. An explanation is suggested for the disappearance of pyruvic acid when vitamin B_1 is supplied to avitaminous birds or animals and for the laying down of fat under such conditions. Furthermore, the action of dietary fats in sparing vitamin B_1 might be through provision of the body with necessary fat which on diets poorer in fat would be synthesized by the animal from carbohydrate with the help of vitamin B_1.

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THE EXPERIMENTAL PRODUCTION OF INTERSEXUALITY IN THE FEMALE RAT WITH TESTOSTERONE

IN a previous report[1] the observations of Hain[2] on the production of hypospadias in the female offspring of the rat by the injection of the mother with estrone,[3] before or immediately after birth, have been confirmed. We have now found that estradiol[3] injected into the mother (2.0–3.0 mg) antepartum or into the *new-born* female (0.2–0.4 mg) also produces hypospadias. The male offspring were apparently not influenced. On the basis of embryological facts, it was suggested[1] that the hypospadias was due to an hypotrophic defect. This immediately suggested the idea that testosterone[3] when given to the pregnant rat might (a) cause hypospadias in the male offspring, or (b) produce an arrest of the development of the vagina in the female, or (c) produce intersexuality (free-martin) in the female. These latter two possibilities have now been shown to be true.

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5 H. A. Krebs and W. A. Johnson, *Biochem. Jour.*, 31: 645, 1937.
1 R. R. Greene, *Proceedings Soc. Exp. Biol. and Med.*, 36: 503, 1937.
2 A. M. Hain, *Quart. Jour. Exp. Physiol.*, 25: 131, 303, 1935; *ibid.*, 26: 290, 293, 1936.
3 We desire to thank Dr. Oliver Kamm, of Parke, Davis and Company for the estrone, and Dr. E. Schwenk, of the Schering Corporation, for the estradiol and testosterone used in this work.