from the ground (26 days after planting) and thereafter declined gradually as the plant went to seed. A part of the decrease in rutin concentration was due to the relatively faster growth of stem tissues, which contain less rutin than the leaves and blossoms, and later in the season, to atrophying of leaf tissues and replacement of blossoms by seeds. The weight of rutin per plant, however, reached a maximum in 37 to 51 days from emergence and then was 3.5 to 4 times as great as at the time of greatest concentration, the plants having increased 6 to 12 times in weight during the two- to four-week period. On an over-all yield basis, one acre of buckwheat in 26 days from planting would produce 14.2 pounds of rutin, while in 40 days the yield would be 50.25 pounds, or approximately 3.5 times as much.

Storage of the crop presents some difficulties because of the tendency of rutin to disappear as the plant dries. Buckwheat exposed to the sun as in haymaking does not dry quickly or thoroughly and loses rutin rapidly. Experiments conducted to determine the effect of drying conditions upon the rutin content showed that the loss of rutin usually increased as the drying process was prolonged, especially at moderate temperatures. Some typical cases are presented in Table 3.

TABLE 3

LOSS OF RUTIN ON DRYING BUCKWHEAT

Sample number	Manner of drying	Rutin content* of		Loss Per cent
		Undried plant Per cent	Dried plant Per cent	
32	Dried in air 4 days, then at 110° overnight	2.50	0.71	71.6
34	Dried† at 135° for 22 minutes	2.12	1.36	35.8
34	Dried† at 71° for 135 minutes	2.12	.00	100.
34	Dried at 110° for 19 hours	2.12	0.59	72.2
35	Dried† at 105° for 50 minutes	2.98	0.84	71.9
35	Dried at 92–100° for 4 hours	2.98	0.81	72.8
37	Dried at 92–100° for 6 hours	2.47	1.53	38.1
37	Dried at 105° for 40 minutes	2.47	1.52	38.1

* Moisture-free basis.
† Chopped.

When the buckwheat is thoroughly dried the rutin content appears to be stable, no loss being observed in specimens stored for six months or more.

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Thiamine Depletion of Human Subjects on a Diet Rich in Thiamine [1]

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Studies on thiamine balance in human subjects have usually been carried out with restrictions in dietary intake, with recovery or therapeutic test doses administered after deficiency symptoms have been produced, or, more recently, with enzymatic destruction of thiamine in the digestive tract.

In this laboratory it has been found that by supplementation of an adequate basal diet with certain viable fresh bakers' yeasts it is possible to produce within a period of days a strikingly low excretion of urinary thiamine. In a series of nine tests, five college women in a satisfactory nutritional state were fed a basal diet consisting of bread, pineapple, and dairy products which provided 1.6 mg. of thiamine per day. In order to further insure satisfactory body stores 2 mg. of thiamine hydrochloride were added daily to the subjects' self-selected diets for a 3-day period 24 hours prior to ingestion of the basal diet. The diet sequence was one of three periods: a yeast-supplement period during which either 15 or 150 grams of the live, fresh bakers' yeast containing 7 µg. of thiamine per gram, of a yeast type previously shown not to yield its thiamine for absorption (*4*), was ingested in addition to the basal diet; the yeast period was immediately preceded and followed by a yeast-free basal period.

The interference of the yeast with the availability of dietary thiamine from other sources and the resulting decrease in thiamine output are recorded in Table 1. The trend was obvious during the 15-gram dosage as well as on 150 grams of yeast. The sharpest decline occurred in all cases within the first 4 days after which fluctuations at the low level of approximately 50 µg. of thiamine excretion persisted in the case of the larger dosage, a decline twice as great as that produced by the smaller amount. In like manner, when the larger amount was continued for 10 instead of 6 days, the recovery of higher urinary concentrations upon resumption of the yeast-free diet was measurably slow, indicating a possible depletion of body stores. That the interference was a function of the viability of the yeast was indicated not only by the greater decrease in urinary thiamine excretion on the larger dosage, but also by the decided increases in the

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output when the fresh yeast was heated to the temperature of boiling water just before ingestion (Table 1). It should be noted that the yeast was fed immediately before each meal to permit the possibility of blending with the food. The capacity for viable yeast to withdraw thiamine from a surrounding medium has been well authenticated (*1*). Fecal thiamine concentrations observed during the various periods showed an inverse relationship to urinary thiamine concentrations, indicating that this is a withholding process by the viable yeast rather than destruction within the digestive tract.

TABLE 1

URINARY THIAMINE* EXCRETION ON YEAST-SUPPLEMENTED DIET

Period	Days of period	Grams yeast ingested		
		15 grams (3 subj.)	150 grams 3 subj.†)	150 grams (3 subj.)
		µg. thiamine/ day	*µg. thiamine/ day*	*µg. thiamine/ day*
Basal	3 or 6 days	374‡	312‡	332‡
Basal plus live yeast	6 or 10 days	217	168	161
		178	124	109
		158	107	99
		163	84	29
		163	52	49
		101	50	33
		...	...	62
		...	...	49
		...	...	42
		...	...	40
Basal	3 days	212	189	80
		227	232	141
		242	257	198
Basal plus boiled yeast	3 days		372	
			712	
			702	

* Thiochrome Assay Method.
† Two subjects, only, ingested boiled yeast.
‡ Values are daily averages for entire period.

While the low values observed in the present experiment do not approach the zero excretions reported in long-term investigations such as those of Keys (*3*) with acute deprivation of B-vitamins following a long period of mild depletion, they are within the range accepted as denoting "considerable to severe deficiency" (*2*).

This procedure may possibly have an application in certain short-term experiments, in that rapid depletion of thiamine may be achieved without the use of a quantitative or deficient diet, and thiamine stores may be quickly regained by merely discontinuing the yeast from the basal adequate diet.

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Effect of Methionine Supplements on Hepatic Injury Produced by Carbon Tetrachloride[1]

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It was first observed by Hershey and subsequently by Best and collaborators that choline exerts a lipotropic effect. Since then numerous studies have been performed on the relation of choline, methionine, and cystine to fat metabolism with special reference to hepatic changes (*6*). However, little work has been reported on the relationship of dietary choline and methionine to functional changes in the liver. It was demonstrated by Hough and Freeman (*4*) that removal of protein from the diet resulted in an increase in serum phosphatase and a decreased hepatic clearance of dye. They later reported that choline chloride prevented these changes during the first 8 weeks of the deficient diet (*5*). It is also known that such protein-depleted dogs are more susceptible to hepatic poisons. Miller and Whipple (*7*) noted that the toxicity of chloroform in protein-depleted dogs was decreased when methionine was administered either before or up to four hours after the chloroform anesthesia. The methionine-treated animals survived a period of anesthesia lethal to untreated animals. In similar studies Goodell, *et al.* demonstrated that the rise in icterus index following the administration of mapharsen to protein-depleted dogs was lessened when methionine was also given (*2*).

In the above experiments the beneficial effects of methionine were obtained in animals maintained on practically protein-free diets. It is of interest to determine if methionine supplements to a normal protein diet exert any protective action against hepatic toxins. No studies have appeared on this subject, and the following data, pertinent to this question, are reported.

Liver damage was produced by administering small doses of carbon tetrachloride to healthy, adult dogs. The CCl_4 was mixed with an equal volume of corn oil and given by stomach tube before the dogs were fed. Changes in hepatic function were studied by means of the bromsulphalein and serum phosphatase tests, as they have been shown to be sensitive methods in detecting liver damage produced by CCl_4 (*1*). Serum phosphatase was determined by the method of Bodansky (*1*) and bromsulphalein retention by the

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