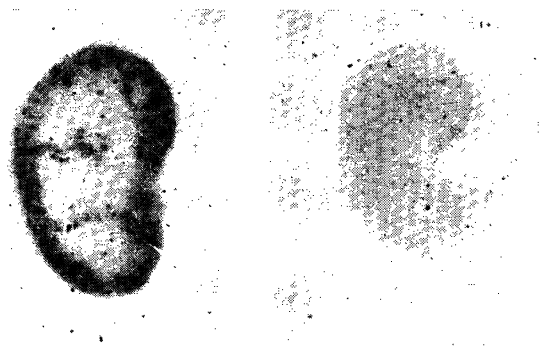
ney from the mouse receiving the radioantiplasma serum showed no such accumulation (Fig. 1B). No accumulation was observed with either the liver or the spleen of the animals receiving the antikidney serum or the antiplasma serum. Fig. 2 shows the radioautographs of the kidney sections. In the sections from the animal receiving the radioantikidney serum there was a punctate accumulation of the radioactivity (Fig. 2A), while this was not the case for the kidney of the mouse receiving the radioantiplasma serum (Fig. 2B). Also, there was no such accumulation of radioactivity in the spleens or livers of the animals receiving either radioantikidney or radioantiplasma serum. With these tissues, very faint and diffuse radioautographs were obtained due to the radioactivity content of the blood in the organ.



A B

FIG. 2. Radioautographs of sections of kidney tissue. A—from mouse injected with radioanti-mouse-kidney serum; B—from mouse injected with radioanti-mouse-plasma serum.

Upon comparing the microphotograph of the kidney from the animals receiving the radioantikidney serum with an enlargement of the radioautograph, it was quite clear that the localization of the radioactivity and presumably the radioantibody was taking place in the glomeruli of the kidney. This point will be discussed more fully elsewhere (*3*).

The concentration of the antikidney serum in the glomeruli is not due to a nonspecific pickup by the kidney of foreign substances or to the fact that the radioactive material may pass through the kidney in its excretory path. The evidence for this is the fact that the radioantiplasma serum control contains substances of the same nature as radioantikidney serum, except for the specific kidney antibodies, and does not show any localization in the kidney. Similarly negative results were shown by a radioantiovalbumin serum. The radioactivity concentration in the various tissues for the two sera described here were quite similar except in the case of the kidney (Table 1). There the concentration of the radioactivity in the kidney of the animal receiving the radioantikidney serum was about three times that in the kidney of the animal receiving the control serum. The radioactivity in the kidney of the mouse receiving the control serum was essentially all due to the blood in the kidney, since mouse kidneys contain about 30–35% blood (*1*). The greater amount of radioactivity in the kidneys of the animal which received the antikidney serum must have been due to the kidney antibodies.

The localization of I^{131} in the glomeruli as shown by the radioautographs is probably a result of the corresponding localization of the specific antibodies to glomerular tissue.

These experiments were repeated with other preparations of radioantikidney serum with similar results.

References

1. KALISS, N., and PRESSMAN, D. To be published.
2. MARINELLI, L. D., and HILL, R. F. *Amer. J. Roentgenol. rad. Therap.*, 1948, **59**, 396.
3. PRESSMAN, D. To be published.
4. PRESSMAN, D., and KEIGHLEY, G. *J. Immunol.*, 1948, **59**, 144.
5. SMADEL, J. E. *J. exp. Med.*, 1936, **64**, 921.

Growth of Potato Sprouts Retarded by 2,4,5-Trichlorophenoxyacetic Acid[1]

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Guthrie (*1*) first discovered that methyl ester of α-naphthalene acetic acid, when applied to potato tubers, retards the growth of sprouts. Many potato growers are now using this chemical on a commercial scale to prevent sprouting of potatoes in storage. Smith, Baeza, and Ellison (*2*) found during the 1945 season that this chemical also retards sprout growth of potatoes in subsequent storage when it is applied as a spray to the potato plants during the growing season. During the 1946 season the authors found that two spray applications of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), 25 ppm on July 19 and 50 ppm on August 20, retarded subsequent sprout growth of potatoes to the same degree as two applications of methyl ester of naphthalene acetic acid (MENA), 2,000 ppm on July 19 and 2,000 ppm on August 20.

In the spring of 1947 tubers of the Sebago variety were treated separately with MENA and 2,4,5-T in isopropyl alcohol and water and applied as a spray at the rate of 1 gm of chemical/bushel of potatoes. Although both chemicals significantly retarded sprout growth as compared with untreated tubers, sprouts of those treated with MENA had significantly less weight than those of tubers treated with 2,4,5-T.

During the 1947 growing season spray applications of sodium naphthalene acetate (sodium NA), 500, 1,750, and 3,000 ppm, compared with applications of sodium 2,4,5-T at 50, 175, and 300 ppm, were made to plants of the Sebago variety. The effect on sprout growth during subsequent storage at 50° F from October 25 to February 26 is shown in Table 1.

In all cases sodium 2,4,5-T retarded sprout growth to a greater degree than sodium NA, although the latter was applied in concentrations 10 times the former. There

[1] Paper No. 301, Department of Vegetable Crops, Cornell University.

were no significant differences in yields of potatoes between any of the treatments.

TABLE 1

WEIGHT OF SPROUTS PER TUBER
(Concentration of spray in ppm)

Date of application	Sodium NA 500	Sodium 2,4,5-T 50	Sodium NA 1,750	Sodium 2,4,5-T 175	Sodium NA 3,000	Sodium 2,4,5-T 300	Sodium NA Mean	Sodium 2,4,5-T Mean
	(gm)	(gm)	(gm)	(gm)	(gm)	(gm)	(gm)	(gm)
Aug. 1	2.30	1.70	2.27	1.22	2.27	0.87	2.28	1.26
Aug. 22	3.07	2.93	2.82	2.25	3.13	1.97	3.01	2.38
Sept. 10	3.22	2.73	3.13	2.75	3.10	2.83	3.15	2.77
Means	2.86	2.45	2.74	2.07	2.83	1.89	2.81	2.14
Untreated:		3.17 gm						

Sodium NA not significantly different from untreated at 19 : 1.
Sodium 2,4,5-T significantly lower than sodium NA or untreated at 99 : 1.

During the spring of 1948 potato tubers of the Houma variety were treated in open baskets in storage with dust forms of MENA (1 gm/bushel) and isopropyl ester of 2,4,5-T (0.9 gm/bushel). Both treatments significantly reduced the weight of sprouts produced; treatment with MENA, however, resulted in greater retardation than treatment with isopropyl ester of 2,4,5-T.

It was assumed that one of the reasons for less retardation of sprout growth by isopropyl ester of 2,4,5-T compared with MENA was due to lesser volatility of the former. During the storage season early in 1948 potato tubers were treated with the chemicals in dust form indicated in Table 2 and stored at 50° F for 10 weeks in closed paper bags to confine the volatile sprout retardant in the atmosphere immediately around the tubers.

TABLE 2

EFFECT OF TREATING POTATO TUBERS WITH SPROUT RETARDANTS

Treatment	Wt. of sprouts (gm/tuber)
1.0 gm of isopropyl ester 2,4,5-T/bushel	2.91*
0.5 " " " " " "	3.70*
1.0 " " MENA " "	1.46†
Untreated	10.81

* Significantly lower than untreated lots at odds 99 : 1.
† Significantly lower than any other treatment at odds 99 : 1.

To obtain information on the penetrability of 2,4,5-T into tubers and its subsequent reaction on sprout growth, the following experiment was conducted. Ten toothpicks soaked for one week in saturated solution of sodium 2,4,5-T were inserted about 1" into each tuber. After storage for three months at 50° F, treated tubers were just beginning to sprout, whereas several sprouts 3" to 4" long developed on each of the untreated tubers (Fig. 1).

The toothpick technique was further employed to insure equal penetration of sodium NA and sodium 2,4,5-T into comparable sets of tubers. Aqueous solutions of 1,000 ppm of the two respective salts were used for soaking the toothpicks. One set of toothpicks was soaked in distilled water for comparison. After the picks were soaked 5 days they were inserted into Houma tubers of similar size and one series of untreated tubers (no toothpicks) was included as a control. Eight replications were used. Table 3 shows the effect of the above treatments on number and weight of sprouts. Single tuber plots were used and each value is the mean for 8 tubers.

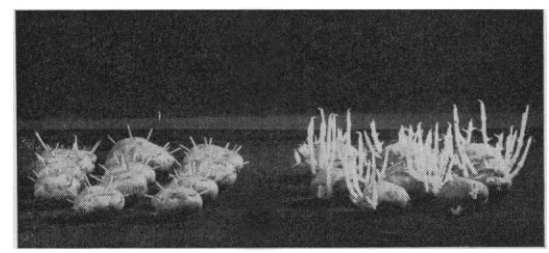

FIG. 1. Left: Tubers pierced with toothpicks which had been soaked in a saturated solution of sodium 2,4,5-T (10 toothpicks/tuber). Right: Untreated control tubers.

TABLE 3

Treatments	No. of sprouts/ tuber	Wt. of sprouts/ tuber
Toothpicks soaked in sodium NA (1,000 ppm)	3.0	2.96
Toothpicks soaked in sodium 2,4,5-T (1,000 ppm)	5.0	2.61
Toothpicks soaked in distilled water	12.1	9.24
Untreated control (no toothpicks)	11.4	10.98

No significant difference was found between the sprout growth of tubers with distilled water-treated toothpicks and tubers with no toothpicks. Sprouting was reduced very significantly by both sodium NA and sodium 2,4,5-T, but no significant difference was found between the two in their effect on number or weight of sprouts. Further work is being conducted with other more volatile forms

of 2,4,5-T to determine its practical value as a sprout retardant on a commercial scale.

References

1. GUTHRIE, JOHN D. *Contr. Boyce Thompson Inst.*, 1939, **10**, 325–328.
2. SMITH, ORA, BAEZA, M. A., and ELLISON, J. H. *Bot. Gaz.*, 1947, **108**, 421–431.

A New Dietary Factor Related to Xanthine Oxidase[1]

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Measurements of the xanthine oxidase activity in rat livers by the method of Axelrod and Elvehjem (*1*) have demonstrated that, in order to obtain normal liver xanthine oxidase levels on diets containing adequate riboflavin, two other dietary factors are essential. These are: (1) adequate protein, as originally indicated by McQuarrie and Venosa (*5*), and (*2*) an unidentified factor found in raw cream and liver, both good sources of xanthine oxidase.

Rats are born without any detectable xanthine oxidase activity in the liver, even when their mothers are on an adequate diet and have normal levels of xanthine oxidase in their own livers at the time of birth. Small amounts appear in the liver during the nursing period, and when the rats are weaned at 21 days of age, the average activity is 720 units ($C_{mm}O_2$/gm of dry liver/hr). This is less than half of the 1,550 units of activity found as an average for mature rats maintained on an adequate diet. If such weanling rats are placed on a diet containing 21% purified casein or 8% casein plus 13% peanut protein or 21% egg albumin plus biotin, the liver xanthine oxidase remains at approximately the starting level for 6 weeks. The 21% casein diet is given in Table 1; all other diets mentioned are identical except for the specific differences noted. When weanling rats are fed Purina dog chow (21% protein), the liver xanthine oxidase is brought to a normal level of 1,535 units in two weeks. If the Wesson oil and Crisco in the 21% purified casein diet are replaced by an equal amount of raw cream (6%), the liver xanthine oxidase activity of weanling rats is increased to 1,300 units in two weeks. If 5% dried whole liver replaces an equal weight of casein in the diet, the liver xanthine oxidase remains low for two weeks but is increased significantly after four weeks. Similarly, feeding a 21% crude casein diet does not affect the liver xanthine oxidase activity within two weeks, but gives levels of 1,260 units after four weeks.

TABLE 1

Casein (GBI Vit. Test)	21%
Crisco	4
Wesson oil	2
Cod-liver oil	1
Salt mix (Phillips and Hart)	4
Glucose	68
Choline chloride	100 mg%
Nicotinic acid	2.5
Ca pantothenate	1.0
Riboflavin	0.4
Thiamine	0.4
Pyridoxine	0.4

These experiments demonstrate that a 21% protein diet is adequate in providing the necessary protein for normal xanthine oxidase levels in the liver if another dietary essential is also incorporated in the diet in adequate amounts. In the relative absence of this unknown factor the starting levels of liver xanthine oxidase remain unchanged. When limited amounts of the factor are supplied, as with the liver and crude casein diets, the xanthine oxidase activity remains low for a period of time and then increases rather suddenly. Feeding a relative abundance of the factor, as with Purina dog chow and, to a lesser extent, the raw cream diet, gives a rapid increase to normal levels.

Rats fed an 8% casein diet (81% glucose) have essentially no xanthine oxidase activity in the liver after four weeks whether they were started as weanlings or were first brought to normal levels of activity by being fed Purina dog chow for two weeks. Rats brought to a zero level of xanthine oxidase activity by feeding them an 8% casein diet remain at the zero level when 6% raw cream replaces the Crisco and Wesson oil in the low-protein diet. Hence, supplying the dietary factor found in raw cream is ineffective in the absence of an adequate protein intake. Such zero levels of xanthine oxidase activity can be restored to normal by feeding dog chow; feeding the 21% purified casein diet allows a slower increase in the activity, indicating the presence of some of the unidentified factor in the diet containing purified casein.

The above experiments were carried out with animals obtained from Albino Farms. Sprague-Dawley rats have a lower xanthine oxidase level in the liver at weaning, averaging 430 units. They show considerably less individual variation at this time, but require appreciably longer dietary periods to bring the activity in the liver to normal levels.

Supplementing the 21% purified casein diet with biotin, inositol, *p*-aminobenzoic acid, pteroylglutamic acid, rutin, ergostanyl acetate, adenine, *d*-ribose, and additional riboflavin did not give normal xanthine oxidase levels in the liver. It is suggested that this dietary factor necessary for normal liver xanthine oxidase activity may be related to the unidentified component of the prosthetic group of xanthine and aldehyde oxidases (*2*, *3*, *4*).

[1] Supported by a grant from the Nutrition Foundation, Inc.

References

1. AXELROD, A. E., and ELVEHJEM, C. A. *J. biol. Chem.*, 1941, **140**, 725.
2. BALL, E. G. *J. biol. Chem.*, 1938, **128**, 51.
3. CORRAN, H. S., DEWAN, J. G., GORDON, A. H., and GREEN, D. E. *Biochem, J.*, 1939, **33**, 1694.
4. GORDON, A. H., GREEN, D. E., and SUBRAHMANYAN, V. *Biochem, J.*, 1940, **34**, 764.
5. MCQUARRIE, E. B., and VENOSA, A. T. *Science*, 1945, **101**, 493.