cent. of the animals inoculated intracutaneously with this strain were infected. Later (after the seventh passage) this property seemed to decrease and about 25 per cent. were infected. But, among the seven other established strains, cutaneous infectivity was not at all pronounced; in fact, it was quite uncommon. In 40 tests there are only two instances (5 per cent.) in which monkeys were infected by these seven other strains from our so-called established group.

Somewhat in contrast to this is the action of the fresh strains. The number of animals tested is small but large enough to indicate a substantial degree of intracutaneous infectivity of not only human material (*i.e.,* prior to its first passage) but also of the virus during its first two passages. In fact, all the few fresh strains which we so far have tested have shown this property in at least a third of the inoculated animals, and the total percentage of animals cutaneously infected in this particular series is about 60 per cent. This appears all the more remarkable when one recalls the difficulties of getting human poliomyelitis virus established in the monkey by the intracerebral route in its earliest passages, and when one also considers that in the present experiments almost the same dose has been used for these intracutaneous as for the intracerebral inoculations. It remains to be seen whether this apparent difference between the behavior of fresh and established strains is a coincidence or not.[8]

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THE EFFECT OF COPPER IN THE PRODUCTION OF NUTRITIONAL ANEMIA IN RATS

DISCREPANCIES in the bioassay results from different laboratories relative to the availability of iron in foodstuffs have been variously explained by Elvehjem and coworkers[1,2,3] and by Smith and Otis.[4] Elvehjem believed that failure to effect complete depletion of iron storage in the experimental animals was the reason for these discrepancies, while Smith and Otis postulated a sex difference in the ability of anemic rats to utilize iron, and attributed differences in results from various laboratories to ignorance of this fact. Mitchell and Hamilton[5] concluded from their paired-feeding studies that the sex difference noted by Smith and Otis was partially, or entirely, the result of a greater intake of the anemogenic basal diet by the male rats. Smith and Otis[6,7] in recent papers have suggested and attempted to prove that copper administration during the depletion period is necessary to assure complete exhaustion of the iron reserves. They believe that failure to completely eliminate these potentially available iron reserves in the depletion period has often resulted in invalid conclusions being drawn from subsequent curative data. They contend that part, at least, of the hemoglobin response was due to the copper of the food under assay rather than to its available iron content.

In this laboratory we have had occasion to deplete large numbers of rats for hemoglobin regeneration studies. Two small groups of these were depleted in the usual way, except that whole milk powder (Klim) was substituted for whole fresh milk, while other groups received in addition to this same basal diet a daily supplement of 0.05 mg copper as copper sulfate. The results are shown in Table I. Whereas Smith

TABLE I

SHOWING COMPARATIVE HEMOGLOBIN DECREASE IN RATS FED WHOLE MILK POWDER WITH AND WITHOUT COPPER

Supplement to basal diet	No. of rats used	Sex	Initial hemoglobin (gms/100 cc)	Decrease in hemoglobin (gms/100 cc)		
				In 3 weeks	In 5 weeks	In 7 weeks
None	10	M	12.26	5.59 ± .55	8.44 ± .20	9.08 ± .20
None	10	F	12.18	3.85 ± .58	8.31 ± .31	9.03 ± .36
0.05 mg Cu as $CuSO_4$.	10	M	11.77	4.84 ± .33	8.01 ± .27	8.48 ± .30
0.05 mg Cu as $CuSO_4$.	10	F	11.77	5.18 ± .40	8.25 ± .27	8.78 ± .21

and Otis found that by supplementing the basal diet with copper, beginning at the fourth or at the sixth week of depletion, an immediate and sustained increase in hemoglobin occurred, we have found that the trend of hemoglobin values during the depletion period is not significantly affected by the presence of copper in the basal depletion ration.

To determine the effect of subsequent iron feeding upon anemic rats depleted with and without copper, five animals from each of the previous groups were fed a daily curative supplement, consisting of 0.1 mg Fe as $FeCl_3$, 0.05 mg Cu as $CuSO_4$ and 0.04 mg Mn as $MnCl_2$, for a period of six weeks.

According to Smith and Otis,[8] at this level of iron

[8] Aided by grants from the President's Birthday Ball Commission for Infantile Paralysis Research.

[1] C. A. Elvehjem and A. R. Kemmerer, *Jour. Biol. Chem.,* 93: 189–195, 1931.

[2] C. A. Elvehjem, E. B. Hart and W. C. Sherman, *Jour. Biol. Chem.,* 103: 61–70, 1933.

[3] W. C. Sherman, C. A. Elvehjem and E. B. Hart, *Jour. Biol. Chem.,* 107: 383–394, 1934.

[4] M. C. Smith and L. Otis, SCIENCE, 85: 125–126, 1937.

[5] H. H. Mitchell and T. S. Hamilton, SCIENCE, 85: 364–366, 1937.

[6] M. C. Smith and L. Otis, *Jour. Nutrition,* 13: 573–582, 1937.

[7] M. C. Smith and L. Otis, *Jour. Nutrition,* 14: 365–371, 1937.

[8] *Op. cit.*

feeding, standard anemic male rats gained 5.5 ± 0.14 grams hemoglobin in six weeks, while females gained 6.5 ± 0.15 grams of hemoglobin. However, when the rats had been made anemic in the presence of copper, presumably from the fourth or sixth week on, the hemoglobin gain for males and females over six weeks was only 3.00 ± 0.28 and 4.50 ± 0.23 grams, respectively. Our results (Table II) indicate that no significant lessening of hemoglobin response occurred when copper had been fed throughout the entire depletion period as compared with hemoglobin response when regular depletion had been carried out. However, we noted a continuance of sex variation in response to iron feeding regardless of depletion technique. This does not corroborate the findings of Smith and Otis that sex variation diminished when copper was furnished during depletion and who believed that it would disappear entirely if a longer period of copper feeding were carried out.

TABLE II
SHOWING HEMOGLOBIN REGENERATION DURING CURATIVE PERIOD OF RATS MADE ANEMIC WITH AND WITHOUT COPPER

Amount of iron fed daily (mg)	Supplement to basal diet during depletion	No. of rats	Sex	Initial hemoglobin (gms/ 100 cc)	Gain in hemoglobin in 6 wks. curative period (gms/ 100 cc)
0.10	None	5	M	3.28	4.72 ± .22
0.10	None	5	F	3.15	5.76 ± .19
0.10	Copper	5	M	3.30	4.60 ± .24
0.10	Copper	5	F	3.00	5.83 ± .20

It must be concluded from results obtained in this laboratory that supplementing the basal diet with copper in anemia production does not significantly affect the time nor severity of iron depletion, nor does it affect the hemoglobin response to subsequent iron feeding.

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THE ABSORPTION OF SELENIUM BY CITRUS AND BY GRAPES

PLANTS differ greatly in their ability to accumulate selenium from the soil.[1]

This situation is of importance in connection with any public health hazards which may follow from the use of insecticidal sprays containing selenium. Hence the results of analyses of citrus fruit and grapes from plots which have been treated for several years with the commercial product Selocide and from nearby untreated plots may be of interest. This material corresponds in composition to the empirical formula $(KNH_4S)_5Se$ and is sold as a 30 per cent. aqueous solution.[2]

It was used for the control of the Pacific mite, *Tetranychus pacificus,* on grapes and of the citrus red spider, *Paratetranychus citri,* on citrus. The usual dilution was 1–800 on citrus and 1–600 on grapes. Samples of fruit were picked at random. All citrus fruit was washed in dilute nitric acid in order to remove adhering selenium from the exterior. Soil samples were taken at the edge of the trees or vines where the run-off of spray was heaviest. Only composite results for 0–36 inches depth are given in Table I:

TABLE I
A. CITRUS

Plot	No. applications	Se in soil 0–36″	Se in fruit	
			skin	pulp
		ppm		ppm
A (1)*	6 (1932–36)	0.91	0.09	0.07
AA (v.o.)†	None	0.28	0.13	0.12
B (v.o.)	6 (1933–36)	0.46	0.47	0.07
BB (v.o.)	none	0.29	0.07	0.03
C (1)	5 (1934–36)	0.80	0.31	0.06
CC (1)	none	0.27	0.12	0.09
D (v.o.)	3 (1935–36)	0.61	0.22	0.12
DD (v.o.)	none	0.30	0.10	0.02
E (v.o.)	3 (1933–36)	0.27	0.17	0.07
EE (v.o.)	none	0.35	0.09	0.04
F (1)	5 (1933–35)	0.93	0.18	0.02
FF (1)	none	0.60	0.10	0.02
G (1)	1 (1936)	0.21	0.22	0.03
GG (1)	none	0.12	0.08	0.01
H (v.o.)	2 (1934)	0.53	0.09	0.03
HH (v.o.)	none	0.31	0.06	0.02

* (1) = lemons.
† (v.o.) = Valencia oranges.

B. THOMPSON SEEDLESS GRAPES

Field	No. applications	Se in soil 0–36″	Se in grapes	
			unwashed	washed
4	5 (1933–37)	0.49	1.80	0.64
4	2 (1933–34)	...	0.14	...
13–1	2 (1935–36)	...	0.23	...
4	none	0.25	0.11	...

On the basis of these results the following conclusions seem to be justified:

(1) Se occurs in all untreated soils tested at about 0.25 ppm.

(2) Se in soils of plots sprayed up to six times was always less than 1 ppm.

(3) The average Se content of the sprayed citrus fruits was: skin 0.21 ppm., pulp 0.06 ppm.

(4) The average Se content of the unsprayed citrus fruit was: skin 0.10 ppm., pulp 0.05 ppm.

(5) Grapes from vines sprayed during the current year contained over 0.6 ppm. Se, but the amount was much less when selenium was used in earlier years only.

(6) Neither citrus trees nor grape vines concentrate

[1] O. A. Beath *et al., Jour. Am. Pharm. Assn.,* 26: 394–405, 1937; H. G. Byers, *U. S. Dept. Agr. Tech. Bul.* No. 482, 48 pp., 1935.

[2] C. B. Gnadinger, *Jour. Ind. Eng. Chem.,* 25: 633–7, 1933.