were DDT, malathion, and parathion. In every case neither the chemical tested, the deactivator, nor the heptachlor was affected.

Discussion

In 1955, deactivated mineral carriers were used in heptachlor formulations with excellent results. When the deactivator is used in the correct amount and applied properly, heptachlor formulations with excellent storage stability can be prepared. Additional time and labor required in formulation are practically negligible, and the cost of the deactivator is in the range of 0.1 cent per pound of finished formulation when diluents such as talcs and pyrophyllites are used.

Literature Cited

(1) Johnson, O., *J. Phys. Chem.* 59, 827–31 (1955).
(2) Polen, P. B., Silverman, Paul, *Anal. Chem.* 24, 733 (1952).
(3) Walling, Cheves, *J. Am. Chem. Soc.* 72, 1164–68 (1950).

Received for review April 20, 1956. Accepted June 27, 1956.

MODE OF ACTION OF PESTICIDES

Reversal of Fungitoxicity of Copper-8-Quinolinolate

S. S. BLOCK

University of Florida, Gainesville, Fla.

Antimicrobial activity of copper oxinate (copper-8-quinolinolate) is reversed in the presence of excess metal ions and excess oxine (8-quinolinol). By employing a simplified experimental system, clear-cut demonstrations of the reversal phenomenon have been demonstrated. The conversion of the 2 to 1 (moles of oxine to copper) chelate to the 1 to 1 chelate in the presence of excess copper ions has been indicated by partition of the chelates between oil and water. Results support the mechanism of Albert and coworkers that the 1 to 1 chelate is the toxic entity and that equilibrium and cell penetration relationships account for the reversals of antimicrobial action.

The practical applications of 8-quinolinol (oxine) and copper-8-quinolinolate (copper oxinate) in industry and agriculture were previously reviewed (*6*). The present report considers one interesting aspect of the activity of oxine and its chelates—namely, the "reversal" phenomenon. This describes the condition where the inhibition of microbiological activity produced by the toxic chemical is antagonized by other materials and the microbiological activity is restored.

In 1943, Zentmyer (*21*) introduced the concept that oxine might be antifungal by virtue of its ability to precipitate from solution traces of metals necessary for fungus growth. As evidence, he cited an experiment in which a fungus that requires zinc in its metabolism was first poisoned by adding oxine to its growth medium and then restored to active growth by the further addition of a zinc salt (*22*).

Other workers reported reversal of oxine toxicity with additional metals (Table I). Gale (*7*) distinguished between the reversal of the assimilatory and fermentation processes of the microorganism with different metals. The precipitation concept, however, was soon confronted with data (*10, 11, 16, 18*) showing that the chelates formed from the reaction of oxine with metals were themselves as toxic as, or even more strongly antimicrobial than oxine. Moreover, the toxicity of the metal chelates could be reversed with metal ions. Thus, some other explanation for these interesting reversals was required.

Rubbo, Albert, and Gibson (*13*) proposed the theory that copper and iron chelates of oxine cause the oxidation of vital cell constituents such as thiol groups, and that in the case of gram-positive bacteria the metal cobalt prevents this oxidation. According to Vicklund and coworkers (*19*) the reversal of inhibition of metal ions results from the workings of the mass action law in balancing the dissociation equilibrium so as to reduce the concentration of oxine ions. The oxine ion, they maintained, was the functional portion of the chelate in exerting toxic effects. Manten, Klöpping, and van der Kerk (*10*) failed to get reversals except under certain conditions. They attributed reversal phenomena to a lowering of the toxicant solubility below the inhibitory threshold, owing to the formation of a metal chelate which has a lower solubility in the growth medium than the free oxine. Anderson and Swaby (*5*) failed to get reversals with cobalt, zinc, manganese, iron, or copper, and got only a partial reversal with molybdenum. Albert, Gibson, and Rubbo (*2*) proposed an explanation of reversals based upon the dissociation of the 2 to 1 chelate (made up of 2 moles of oxine to 1 mole of metal) to the 1 to 1 chelate (made up of equimolar concentrations of oxine and metal). The latter chelate, said to be produced

Table I. Literature on Reversal of Fungitoxicity of Oxine and Metal Oxinates with Metal Ions

Workers	Year	Reference	Reversal	Metals	Organism	Medium	Conditions[a]
Zentmyer	1944	(22)	Yes	Zn	*Fusarium oxysporum* (Var. *lycopersici*)	Peptone-maltose broth	3 equivalents for reversal, 0.7 partial reversal
Viferri and Baldacci	1946	(20)	No	Zn	*Alternaria tennuis; Fusarium oxysporum, F. moniliforme, F. solani, Penicillium notatum*	Maltose broth for first two fungi; glucose broth for all	No reversal with 1 equivalent
Albert and coworkers	1947	(3, 13)	Yes	Co^{++}	Gram-positive bacteria	Beef extract peptone broth	2 equivalents
Rubbo and coworkers	1950		No	Ca, Mg, Mn^{++}, Cd, Fe^{++}, Zn, Ni^{++}, Cu^{++}	Gram-positive bacteria	Beef extract peptone broth	2 equivalents
			Yes	Zn, Fe^{++}, Cu^{++}; lesser with Mn, Ni, Co^{++}, Cd	Gram-negative bacteria	Beef extract peptone broth	4 equivalents
Gale	1949	(7)	Yes	Mn^{++}; lesser with Mg, Co^{++}, Fe^{++} Fe^{+++}	*Staphylococcus aureus*	Glucose + glutamic acid broth	For assimilation, 1 equiv. of Mn gave 100% reversal, lesser with Co^{++}, Fe^{++}, Fe^{+++}
			Yes	Mn^{++}, Fe^{++}, Mg^{++}	*Staphylococcus aureus*	Glucose + glutamic acid broth	For fermentation, reversals not as complete as for assimilation
Schraufstätter	1950	(14)	Yes	Co^{++}	*Staphylococcus aureus*	...	...
			No	Zn, Mg, Cd, Cu^{++}, Mn^{++}, Ni^{++}, Fe^{++}, Fe^{+++}	*Staphylococcus aureus*	...	...
Schuler and Meier	1950	(15)	Yes	Co^{++}	*Escherichia coli*	Beef extract broth	1:0.6 (oxine: Co) molar ratio if mixed before adding cells; 1:0.9 ratio if cells and oxine together before Co addition
			Yes	Co^{++}	*Staphylococcus aureus*	Beef extract broth	1:0.6 ratio if mixed before adding cells
			No	Co^{++}	*Staphylococcus aureus*	Beef extract broth	When oxine and cells together before Co addition
Vicklund and Manowitz	1950	(18)	Yes	Fe^{++}, Al	*Aspergillus niger*	Mineral salts-dextrose agar	Complete reversal effected with 129 equiv. of Fe^{++} and 51 equiv. of Al for oxine, and 116 equiv. of Fe for copper oxinate
Anderson and Swaby	1951	(5)	Yes	Mo	*Aspergillus niger*	Mineral salts-sucrose broth	Only partial reversal with 1 mg./l. Mo (less than 2 equivalents)
			No	Zn, Mn^{++}, Fe^{++}, Cu^{++}	*Aspergillus niger*	Mineral salts-sucrose broth	1 mg./l. metals to 0.7 mg./l. oxine (less than 2 equivalents)
Manten and coworkers	1951	(10)	Yes	Co^{++}	*Aspergillus niger*	Mineral salts-sucrose agar	Complete reversal with 1000 equiv. Co^{++} but only with low concn. of oxine
			No	Zn, Mo, Mn^{++}, Cu^{++}, Fe^{++}	*Aspergillus niger*	Mineral salts-sucrose agar	500, 1500, 500, 600, 1000 equiv. of Zn, Mo, Mn, Cu, Fe, respectively
			Yes	Fe^{++}, Co^{++}	*Aspergillus niger*	10% malt sugar	Complete reversal with 2 equiv. Fe, incomplete with 0.5 equiv. Co
			No	Zn, Mo, Mn^{++}, Cu^{++}, Fe^{++}	*Aspergillus niger*	10% malt agar	5, 15, 5, 6 equivalents of Zn, Mo, Mn, Cu, respectively
Teitell	1952	(17)	Yes	Cu^{++}, Zn	*Trichoderma* sp.	Mineral salts-dextrose broth	50 equiv. Cu, 20 equiv. Zn
Greathouse and coworkers	1952	(9)	No	Fe^{++}, Cu^{++}, Co^{++}, Al	*Aspergillus niger*	Mineral salts-dextrose agar	No reversal of $Cu(Ox)_2$ with 100 equiv. of metals
Albert and coworkers	1953	(2)	Yes	Co^{++}, Zn, Ni, Fe^{++}, Fe^{+++}, Cd	*Staphylococcus aureus*	Beef extract peptone broth	1 equiv. Co, 200 equiv. Zn, Ni, Fe^{+++}, Cd. Partial reversal with 200 equiv. Fe^{++}
			No	Ca, Mg, Mn	*Staphylococcus aureus*	Beef extract peptone broth	200 equiv.

[a] Weight concentrations converted to equivalents in order to have all results on same basis.

Table II. Effect of Increasing Copper Concentration on Toxicity of Oxine

(Organism. *Curvularia lunata*)

Oxine (10^{-4}M), Ml.	$Cu(Ac)_2$ Ml.	$Cu(Ac)_2$ Concn., M	% Germination of Spores	Length of Hyphae[a]
1	0	10^{-5}	100	20X
1	1	10^{-5}	100	20X
1	2	10^{-5}	100	20X
1	3	10^{-5}	96	1.5X
1	4	10^{-5}	85	0.6X
1	5	10^{-5}	44	0.2X
1	6	10^{-5}	46	0.2X
1	7	10^{-5}	24	0.2X
1	8	10^{-5}	12	0.1X
1	9	10^{-5}	2	0.1X
1	1	10^{-4}	2	0.1X
1	5	10^{-4}	5	0.1X
1	1	10^{-3}	10	0.1X
1	2	10^{-3}	21	0.2X
1	4	10^{-3}	5	0.5X
1	7	10^{-3}	1	0.5X
1	1	10^{-2}	0	0
0	2	10^{-3}	63	5X
0	4	10^{-3}	6	0.2–4X
0	1	10^{-3}	0	0
0	0	...	100	20X

[a] Estimated number of times longer than diameter of spore.

from the former in the presence of excess metal ions, was stated to be unable to penetrate the cells sufficiently to exert its toxicity. The reversal of oxine inhibition of gram-positive bacteria with cobalt, however, was considered to be due to a more specific reaction within the microbial cell.

The data in Table I show a great deal of difference in the results obtained by different workers. Some reported reversals; others employing the same organisms did not get reversal with the same metals.

Experimental

Reversal of Toxicity with Metals. The experimental procedure employed in this work was the fungus spore germination test. By employing this simple test method (*4*), it was possible to eliminate the use of agar and complex media which might influence the experiments, and to observe microscopically the effect of treatment on individual fungus spores. *Curvularia lunata* spores were employed because these were found to be able to germinate in pure water. A small quantity (0.1%) of orange juice was added to the water, however, for this provided more complete germination and vigorous growth than pure water alone. The spores were taken from potato dextrose slants and were washed and centrifuged before being used. After 24 hours of incubation the percentage germination was recorded. Although it is customary to count as germinated only those spores whose hyphae are longer than the diameter of the spores, in this work all germinated spores were recorded as well as the length of the hyphae. Valuable information would have been missed, had not the length of the hyphae been noted. These lengths were estimated and are expressed in the tables as the number of times they were greater than the spore diameter.

In Table II data are presented on the fungitoxicity produced by the progressive increase in the concentration of copper, while keeping the concentration of oxine constant. In all instances the solutions were made to constant volume with water in order to maintain these concentration relationships.

Table II shows that 1 or 2 ml. of cupric acetate had no effect on the germination of the spores or the length of the hyphae. The third milliliter, however, produced marked inhibition, as evidenced by the length of the hyphae, although there was little effect on percentage of germination. The fungitoxicity was progressively increased with added copper until 9 to 10 ml. of cupric acetate were added, whereupon some further increase appeared to diminish the toxicity. Still further increase in the copper concentration increased the fungitoxicity, but this effect could be attributed to copper toxicity, as is shown by the data where no oxine is present.

Unfortunately, the toxicity of the copper ion occurred at a point where it obscured any clear-cut demonstration of reversal of fungitoxicity with increasing concentration of the metal. For this reason, the experiment was repeated, employing the less toxic metal, nickel, in place of copper. Each tube contained 1 ml. of 10^{-4} *M* oxine, as in Table II, plus 1 ml. of 10^{-4}*M* copper acetate and was diluted to 10 ml.

These proportions of copper and oxine were employed because, as observed from Table II, greater fungitoxicity was demonstrated when 1 ml. of 10^{-4}*M* copper acetate was present, giving copper and oxine in equimolar proportions, than when 5 ml. of 10^{-5}*M* copper acetate was added, to give proportions of the 2 to 1 chelate. In order to maintain pH 5.5, as in the experiment in Table II, 0.5% acetate buffer was added. Nickelous chloride was added in the same concentrations as cupric acetate in Table II. The pertinent data, which are given in Table III, show the reversal of the copper oxinate by nickel. Since the nickel was not toxic in the range employed, it is possible to witness the progressive decrease in fungitoxicity as the concentration of nickel was increased.

Because the stability constant of ferric oxinate is considerably greater than cupric oxinate (*2*), ferric iron should be a better reversing agent than copper. In order to keep iron buffered and in solution at pH 4.6 it was necessary to employ 1% citric acid in the iron solution. This, in turn, made it necessary to employ higher concentrations of copper oxinate in order to obtain toxicity because of the competing chelating properties of the citric acid. It is shown in Table IV that ferric iron produces a reversal of the copper oxinate toxicity and that a further increase in concentration of copper oxinate is counteracted by a similar increase in the concentration of iron.

As shown, both nickel and iron in sufficient concentration can reverse the toxicity of copper oxinate. It should be recognized, however, that the lowered toxicity with increasing metal concen-

Table III. Effect of Nickel on Fungitoxicity of Copper Oxinate

(Organism. *Curvularia lunata*)

$NiCl_2$ Ml.	$NiCl_2$ Concn.	% Germination of Spores	Length of Hyphae
0	...	0	0
1	10^{-3}	0	0
1	10^{-2}	10	8X
2	10^{-2}	20	20X
4	10^{-2}	90	10X
7	10^{-2}	100	20X

Table IV. Effect of Iron on Fungitoxicity of Copper Oxinate

(Organism. *Curvularia lunata*)

Copper Oxinate, Concn., M	$FeCl_3$, Concn., M	% Germination of Spores	Length of Hyphae
4×10^{-5}	0	4	1X
	7×10^{-4}	28	4X
	7×10^{-3}	98	20X
6×10^{-5}	0	18	20X
	7×10^{-3}	90	20X
	1.75×10^{-2}	98	20X
8×10^{-5}	0	6	20X
	7×10^{-3}	17	3X
	1.75×10^{-2}	91	20X
0	0	100	20X
	7×10^{-3}	100	20X
	1.75×10^{-2}	100	20X

ıration might be interpreted as the displacement of copper in the chelate ınd the formation of the iron and nickel oxinates. If the latter are less toxic han copper oxinate (*6*), a diminution f toxicity might result from the addition f excess iron or nickel to a solution of opper oxinate. To be sure of the opper oxinate reversal phenomenon, t should properly be produced with opper ions. As *Curvularia* was shown n Table II to be sensitive to inorganic opper, an organism that is resistant to opper, *Aspergillus niger*, was employed. The results employing *Aspergillus* and everal concentrations of copper oxinate ith copper acetate are found in Table . As with iron and nickel, the toxicity f the copper oxinate is reduced with ncreasing quantities of copper ion.

Reversal of Toxicity with Excess Oxine. Although many workers observed the reversal effect produced by xcess metals in conjunction with oxine, nly Albert and coworkers (*2*) have reorted a parallel reversal of metal xinate toxicity caused by excess oxine. Their work was done with bacteria and it was of interest to learn whether such a reversal can be demonstrated with fungus spores. The procedure employed was to add the spores to copper oxinate in water with varying amounts of oxine. At intervals, the spores were sampled, centrifuged, washed with water, and suspended in diluted orange juice solution employed as a germination stimulant. This procedure was necessary, because the high concentration of oxine employed was fungistatic and had to be removed to permit germination of the spores. In Table VI it is shown that oxine as well as metals can effect reversal of toxicity.

Discussion

Progressive reversal or counteraction of the inhibition of fungi by the highly toxic compound copper oxinate has been demonstrated to occur in the presence of an increasingly high concentration of nickel, iron, and copper ions.

Table V. Effect of Copper on Fungitoxicity of Copper Oxinate

(Organism. *Aspergillus niger*)

Copper Oxinate, Concn., M	$Cu(Ac)_2$ Concn., M	% Germination of Spores	Length of Hyphae
5×10^{-5}	0	5	2X
	2×10^{-3}	80	2X
	10^{-2}	90	3X
1^{-4}	0	5	2X
	2×10^{-3}	70	3X
	10^{-2}	90	3X
$\times 10^{-4}$	0	7	2X
	2×10^{-3}	0	0
	10^{-2}	100	20X
0	0	85	20X
	2×10^{-3}	100	20X
	10^{-2}	100	20X

The failure of Anderson and Swaby to get reversal with metals might be explained by the fact that they used less than 2 equivalents, thus lacking sufficient excess metal ions. Where an agar medium was employed (*9*, *10*), the agar might have prevented equilibrium conditions from being established. For example, the rate of solution of the highly water-insoluble oxinates might be so slow as to account for the lack of reversal where the lipophilic oxinates might directly dissolve in the cell membrane prior to dissolving in the aqueous agar medium. If the chelate solubility explanation (*9*) is accepted, why then did increasing concentrations of iron and cobalt over that quantity necessary to form the chelates give more complete remission of the inhibition? The theory of Vicklund and Manowitz (*18*) is satisfactory as far as it goes, but it does not consider the possibility of the 1 to 1 chelate nor the reversal with excess oxine.

The hypothesis offered by Albert, Gibson, and Rubbo (*2*) based upon mass law equilibria provides the most satisfactory explanation for the experimental findings. As the metal ion concentration is increased in the presence of chelate, there is a shift in concentration from the fully chelated metal species

Table VI. Effect of Oxine on Fungitoxicity of Copper Oxinate[a] (5×10^{-6}M)

(Organism. *Curvularia lunata*)

Oxine Concn., M	% Germination of Spores	Length of Hyphae
	0 Hour	
0	100	20X
10^{-4}	100	20X
2×10^{-3}	100	20X
	1 Hour	
0	50	1.5X
10^{-4}	96	20X
2×10^{-3}	98	15X
	4 Hours	
0	40	1X
10^{-4}	80	3X
2×10^{-3}	92	10X
	7 Hours	
0	40	0.2X
10^{-4}	70	3X
2×10^{-3}	79	2X
	24 Hours	
0	9	0.2X
10^{-4}	60	0.5X
2×10^{-3}	74	2X

[a] Toxicity measured at intervals following treatment.

Table VII. Effect of Copper Ions on Partition of Copper Oxinate between Oil and Water

$Cu(OX)_2$, Concn., M	$Cu(Ac)_2$, Concn., M	Transmittance of Light by Xylene Fraction at 425 mμ, %
4×10^{-5}	0	68
4×10^{-5}	4×10^{-4}	89
4×10^{-5}	8×10^{-3}	98

$Cu(Ox)_2$, toward the half chelated metal species, Cu^+Ox, as required by the equilibrium equations. If, as proposed (*2*), this change occurs and the ionic 1 to 1 chelate that becomes the increasingly more prominent species with increasing metal ion concentration is less able to exert its toxicity by its difficulty in penetrating the fatty, cell membrane, the toxicity of the solution would indeed by suppressed by metal ions. Those metals which have a greater affinity for oxine, as measured by their stability constants, would be most efficient in reversing the toxicity. As has been seen, this is the case.

Evidence for the existence of the 1 to 1 chelate has been given by Albert (*1*) by titration and the stability constant measured. However, since the proposed mode of action is based upon this entity, a special experiment has been run to give further evidence of the presence of this form and to demonstrate whether it is produced from the 2 to 1 chelate with increasing concentration of metal ions.

Copper oxinate, 2 to 1 chelate, was dissolved in xylene and divided into three portions. Each portion was partitioned between the xylene and an aqueous medium. In the first extraction the aqueous medium was water alone; in the second portion it was water containing 10 equivalents of cupric acetate; and in the third portion it was water with 200 equivalents of cupric acetate. The quantity of 2 to 1 chelate remaining in the xylene phase was indicated by the light transmittance measured spectrophotometrically at 425 mμ. If the copper ions resulted in an increased quantity of the half chelate at the expense of the 2 to 1 chelate, and the 1 to 1 chelate were preferentially soluble in the aqueous phase, the quantity of the fully chelated copper in the xylene would be reduced. The data in Table VII show the gradual increase in transmitted light as the concentration of copper acetate was increased. Since the pH was unchanged, it can be assumed that the 2 to 1 chelate was converted to the 1 to 1 chelate, which was transferred to the aqueous extract. There is an equilibrium between the two chelates and even with 10 equivalents of copper, the data exhibit the presence of 2 to 1 chelate in the oil fraction. With 200 equivalents of copper ion, however, the 2 to 1 chelate was virtually absent. The 2 to 1 chelate cannot be considered to be dissociated to the water-soluble oxine anion and copper cation, because the oxine anion concentration is strongly suppressed by the high concentration of metal ions.

In a recent paper (*8*), Goksøyr reports different absorption bands as he adds copper ion to sodium dimethyldithiocarbamate, which he attributes to the 1 to 1 and 2 to 1 complexes of copper dimethyldithiocarbamate. From his biological studies he concludes that the 1 to 1 copper complex is the toxic entity.

The fact that metals such as nickel and aluminum, that are not known to have physiological functions in fungus metabolism, produce reversals indicates that the reversals are not related to specific chemical activity within the living tissues. It is possible that the effective reversal of oxine obtained with ferrous iron (*10, 18*) might be the result of oxidation to ferric iron in the solution, the latter having a very high stability constant.

The parallel reversal, with excess oxine, has been explained by the hypothesis (*2*) that the 1 to 1 chelate is the true toxic species, that it is produced by equilibrium from the 2 to 1 chelate, and that it is free to react with other complexing agents as exist in the living cell. Because the charge-carrying 1 to 1 chelate cannot readily penetrate the cell, it has been proposed (*2*) that the 2 to 1 chelate enters the cell where it is in equilibrium with the toxic 1 to 1 chelate. With an excess of oxine, however, dissociation of the 2 to 1 chelate is inhibited and a reversal of toxicity occurs. Similarly, an excess of other complexing agents counteract the fungitoxicity of copper oxinate, as in the case of citric acid.

Summary and Conclusions

The fungitoxicity of copper oxinate (copper-8-quinolinolate) is progressively reversed with increasing concentrations of copper, nickel, and iron ions. Reversal of the fungitoxicity of copper oxinate with excess oxine (8-quinolinol) has also been shown.

The reversal with excess metals is due to the suppression of the cell-penetrating 2 to 1 chelate, whereas the reversal with excess oxine is due to the suppression of the toxic 1 to 1 chelate within the cell according to the requirement of the equilibrium equations.

References

(1) Albert, A., *Biochem. J.* 54, 646 (1953).
(2) Albert, A., Gibson, M. I., Rubbo, S. D., *Brit. J. Exptl. Pathol.* 34, 119 (1953).
(3) Albert, A., Rubbo, S. D., Goldacre, R. J., Balfour, B. G., *Ibid.*, 28, 69–87 (1947).
(4) American Phytopathological Society, Committee on Standardization of Fungicidal Tests, *Phytopathol.* 33, 627 (1943).
(5) Anderson, B. I., Swaby, R. J., *Australian J. Sci. Research* B-4, 275–82 (1951).
(6) Block, S. S., J. AGR. FOOD CHEM. 3, 229 (1955).
(7) Gale, E. F., *J. Gen. Microbiol.* 3, 369 (1949).
(8) Goksøyr, J., *Nature* 175, 820 (1955).
(9) Greathouse, G., Block, S. S. Butler, G. B., Emerson, D. L., Kovach, E. G., Barnes, D. E., Johnson, R. A., "Research Studies, and Delivery of Compounds," U. S. Corps of Engineers, Fort Belvoir, Va., Terminal Rept. June 30, 1952.
(10) Manten, A., Klöpping, H. L., van der Kerk, G. J. M., *Antonie van Leeuwenhoek J. Microbiol. Serol.* 17, 58–68 (1951).
(11) Mason, C. L., *Phytopathol.* 38, 740 (1948).
(12) Moeller, Therald, "Inorganic Chemistry," p. 301, Wiley, New York, 1952.
(13) Rubbo, S. D., Albert, A., Gibson, M. I., *Brit. J. Exptl. Pathol.* 31, 425–41 (1950).
(14) Schraufstätter, E., *Z. Naturforsch.* 5G, 190–5 (1950).
(15) Schuler, W., Meier, R., *Schweiz Z. Path. u. Bakt.* 13, 463–9 (1950).
(16) Sexton, W. A., "Chemical Constitution and Biological Activity," p. 226, Van Nostrand, New York, 1952.
(17) Teitell, L., Pittman-Dunn Laboratories, Frankford Arsenal, Philadelphia, Pa., unpublished data, 1952.
(18) Vicklund, R. E., Manowitz, M., *CADO, Tech. Data Dig.* 15, No. 5, 18–21 (1950).
(19) Vicklund, R. E., Manowitz, M., Bagdon, V. J., *Mycologia* 46, 133 (1954).
(20) Viferri, R., Baldacci, E., *Farm. sci. e tec.* 1, 250 (1946).
(21) Zentmyer, G. A., *Phytopathol.* 33, 1121 (1943).
(22) Zentmyer, G. A., *Science* 100, 294 (1944).

Received for review January 12, 1956. Accepted July 18, 1956. Part of a research contract sponsored and supported by the Corps of Engineers, U. S. Army, Fort Belvoir, Va.

Quantitative Determination of Terminal Methionine, Leucine, and Lysine in Raw and Toasted Soybean Oil Meal–Correction

In the article on "Quantitative Determination of Terminal Methionine, Leucine, and Lysine in Raw and Toasted Soybean Oil Meal" [S. W. Fox, Carol Warner, and T. L. Hurst, AG. AND FOOD 3, 704 (1955)] the headings for leucine and methionine are interchanged in Table IV.

S. W. Fox