apparently in the whole world."[2] Announcement of the find was followed at once by a rush of prospecting, resulting in the discovery of other workable beds of colemanite, including the large Callville Wash deposit south of the Muddy Mountains. In 1921 the Pacific Coast Borax Company constructed the automobile road connecting the White Basin deposits with a point on the Union Pacific Railway. This road was laid out through rugged topography along the route of an old Indian trail, and crosses the large window in which the thrust is so well exposed.

The foregoing recitation of facts and dates leads to the following summary: When I studied the Muddy Mountains in 1919 there was not so much as a prospect pit in White Basin, and the site of the present borax road was marked only by a rough and obscure trail. Therefore in the first of the articles mentioned above, Keyes claims to have inspected mine workings more than ten years before the first of those workings was begun or even conceived; in the second quotation he practically repeats and elaborates this remarkable claim. That he wrote his statements with full knowledge of the date of my field work is indicated by his references to my publications on the Muddy Mountains, including my original paper, which appeared in January, 1921.

I refrain from stating obvious conclusions, and also from commenting at length on the general merits of the two papers by Keyes. One additional fact is somewhat illuminating. In the more recent article Keyes gives the appearance of supporting his claims regarding the Muddy Mountain thrust by printing footnote references (without titles) to several of his own earlier publications. Some of these (supposed) articles can not be found in the places cited (perhaps because of grievous errors in the printing of the citations), and the others make no mention of a thrust in the Muddy Mountains but discuss the efficacy of wind as a geologic agent.

Before this article was submitted for publication, the writer wrote to Keyes, pointing out that some of his printed statements appear to be contrary to fact and asking him for an explanation. His reply contains additional grave discrepancies and does not explain any of those discussed above.

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THE SPONTANEOUS OXIDATION OF CYSTEINE

IN a note recently published,[1] Elvehjem criticizes my conclusion[2, 3] that cysteine is autoxidizable, stating that the oxidation observed can not be called autoxidation until the possible presence of metals other than iron and especially copper and manganese has been considered. He states also that, since copper is a catalyst at least sixteen times stronger in cysteine oxidation than is iron, the oxidation could be explained by the presence of traces of copper.

The possibility of traces of contaminants other than iron has been considered and tests, carried out both at the time the reported studies were made and since their publication, have proved as conclusively as a negative can be proved the absence of the metals mentioned. Some of the cysteine used in the experiments had been recrystallized by the method described by the author[4] as often as sixteen times. The rate of oxidation of cysteine thus prepared was identical with that recrystallized but six times, indicating that the additional crystallization did not affect the purity of the product with respect to possible contaminating metals which may act as active catalysts. Further, the samples of the cysteine thus prepared were ignited in two-gram samples and tested for both iron and copper. In no instance was there a visible residue nor any evidence of the presence of either of these metals. The method employed for testing for traces of copper was that described by Yoe,[5] which makes use of the purplish brown color produced when a solution containing copper ions is added to potassium ferrocyanide. This method is sensitive to 1 part in 2,500,000.

Further evidence of the absence of copper was manifest in the failure of cyanide to reduce the oxidation rate of the purified cysteine. Since cyanide is such a powerful poison to both iron and copper catalysis, and since its addition failed to affect the rate of oxidation, it was concluded that both iron and copper were absent.

That manganese, too, was absent was shown by the results of preliminary tests not included in the papers published. The oxidation of cysteine was carried out in the presence of 0.2 molar pyrophosphate which, as Warburg has shown,[6] completely inhibits the activity of iron and manganese but not of copper. The oxidation rate was neither increased nor decreased, the average of several runs being very nearly that obtained for a series of runs on cysteine alone.

In view of the considerations outlined above, it must be inferred that Elvehjem's criticisms are unwarranted and that the oxidation rate observed represents approximately the basic rate of oxidation

[2] Another account of the deposits and their discovery was published by L. F. Noble in "Colemanite in Clark County, Nevada," U. S. Geol. Surv. Bull. 735-B, 1932.

[1] C. A. Elvehjem, SCIENCE, 74: 568–9, 1932.

[2] E. G. Gerwe, *J. Biol. Chem.*, 92: 399–411, 1931.

[3] E. G. Gerwe, *J. Biol. Chem.*, 92: 525–33, 1931.

[4] E. G. Gerwe, *J. Biol. Chem.*, 91: 57, 1931.

[5] J. H. Yoe, "Photometric Chemical Analysis," New York, 1: 182, 1928.

[6] O. Warburg, *Biochem. Z.*, 187: 255, 1927.

which pure cysteine undergoes. The fact that small amounts of iron or copper salts accelerate the oxidation of cysteine by air does not at all prove that a slow oxidation can not take place in their absence. Nor is there reason to suppose that copper and iron and possibly manganese are the only elements having the power of combining spontaneously with oxygen when in a pure state; but on the contrary the evidence is that carbon, sulfur, silicon and a great number of other elements have these same powers. The fact, if it be such, that pyrophosphate fails to check cysteine oxidation and also fails to prevent the catalytic action of copper on the oxidation does not at all prove that therefore the oxidation of cysteine is due to traces of copper. Such a conclusion is a *non-sequitur*. All that it shows is that pyrophosphate is inert in effecting the oxidation, whether catalyzed by copper or not.

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THE IRON-DEFICIENCY HYPOTHESIS IN PELLAGRA

IT is not desired to further the controversy between Bliss[1] and Halliday[2] regarding the soundness of the hypothesis presented by Bliss that pellagra is an iron-deficiency disease, nor to enter into a discussion of the nature of the specific food factor concerned in pellagra. However, since Bliss' position is largely based upon an erroneous interpretation of the more recent literature, a correct presentation of the facts may, at least, serve to forestall misplaced confidence in iron therapy in the treatment of pellagra.

In presenting his hypothesis, Bliss[3] fails to make available sufficient working details of his own observations, depending upon his appraisal of the literature, chiefly the work of Goldberger and his associates, for its support. The following is presented as the keynote of his argument:

> "Goldberger and his associates adopted the working hypothesis that blacktongue of dogs is the analogue of pellagra in man, and they found that diets which are effective in preventing pellagra in man are also effective in preventing blacktongue of dogs—and the same is said of the curative effects of those diets. Examination of the protocols published by Goldberger reveals the fact that those diets which prevented or cured blacktongue in dogs are just those to which had been added 'syrup iodid of iron U.S.P.,' and those diets which when fed to dogs produced blacktongue or failed to cure the disease are those to which no iron had been added. Apparently the iron was not added to or withheld from those diets with any intent to affect the balance of the element, because the footnote explains that it was added to 'improve the mineral composition of the diet.' They make no further mention of iron."

The least that can be said of the above is that it is erroneous and misleading, and may be dismissed once and for all by the statement that at no time was syrup iodid of iron used by Goldberger and his associates in their studies in dogs, and no mention is made of it in any of their reports on blacktongue. It is, however, fair to state that the experimental basal diet used in the human studies at Milledgeville, Georgia, contained this substance, but it was a constant basic factor in all diets used, regardless of whether pellagra was or was not prevented.

Bliss also cites the fact that while beef, liver and yeast have been found to rate high as blacktongue and pellagra preventives, they are also rich in iron. However, he makes the mistake of including egg yolk in this group. Although very rich in iron, this substance made such a poor showing as a blacktongue preventive[4] that its test in human pellagra was not considered worth while. Milk[5] and tomato juice,[6] which, though poor in iron, were found to be pellagra preventive, are left out of consideration; and wheat and cowpeas,[7] which, like egg yolk, are high in iron but low in the pellagra preventive factor, are not mentioned. He also overlooked the fact that while yeast itself was found to be a rich source of this protective factor, its dried watery extract[8] was even better, and its charred form,[9] which retains all the iron, was entirely devoid of it. It would therefore seem that in formulating his iron deficiency theory, Dr. Bliss either overlooked the nullifying nature of the very facts by which he attempts to support it or failed to grasp their significance.

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[1] S. Bliss, "The Iron-Deficiency Hypothesis in Pellagra," SCIENCE, 75: 266, March 4, 1932.

[2] N. Halliday, "The Lack of Correlation between Anemia and the Pellagra-like Symptoms in Rats," SCIENCE, 74: 312, September 25, 1931.

[3] S. Bliss, "Considerations Leading to the View that Pellagra is an Iron-Deficiency Disease," SCIENCE, 72: 577, December 5, 1930.

[4] Goldberger and others, U. S. Pub. Health Rep., Vol. 43, No. 23, Wash., D. C., June 8, 1928 (Reprint 1231).

[5] Goldberger and others, U. S. Pub. Health Rep., Vol. 39, No. 3, Wash., D. C., January 18, 1924 (Reprint 895).

[6] Goldberger and others, U. S. Pub. Health Rep., Vol. 42, No. 19, Wash., D. C., May 13, 1927 (Reprint 1157).

[7] Goldberger and others, U. S. Pub. Health Rep., Vol. 43, No. 23, Wash., D. C., June 8, 1928 (Reprint 1231).

[8] Goldberger and others, U. S. Pub. Health Rep., Vol. 41, No. 8, Wash., D. C., February 19, 1926 (Reprint 1062).

[9] Goldberger and others, U. S. Pub. Health Rep., Vol. 43, No. 12, Wash., D. C., March 23, 1928 (Reprint 1216).