ized very simply in the laboratory. A round-bottom fused quartz flask (about 60 cc) was imbedded in magnesium oxide powder; two small holes were left for admitting monochromatic light and for viewing the interior with a 1P21 photomultiplier tube.

A few very simple experiments served to establish the essential properties of the device. (i) When the flask contained a pure absorber (dilute aqueous $KMnO_4$), the response of the phototube as a function of wavelength I, compared with that obtained with pure water I_0, had the maxima and minima characteristics of the $KMnO_4$ absorption. The number n, representing the effective path length, was a function of the extinction, and was of the order of magnitude 10. (ii) With a colorless turbid material (Dow polystyrene latex), the relative phototube response I/I_0 was not very different from unity and decreased only threefold for a 10^{15}-fold decrease of transmission in a conventional absorption cell. (iii) Mixtures of Dow polystyrene latex and hemoglobin gave the same absorption curve as pure hemoglobin. (iv) Suspensions of intact red blood cells, that gave a very flat wavelength response in the Beckman spectrophotometer were qualitatively indistinguishable from the laked cells when they were observed in the diffuse-light absorption vessel; close quantitative agreement was obtained, except for relatively small differences, which require further investigation. (v) Measurements on a suspension of bacterial cells (*Escherichia coli*) in the presence and absence of free oxygen gave good spectra of the bacterial cytochrome system, particularly of the powerful Soret band at about 420 mμ; this band was completely masked in the Beckman absorption curve. (vi) A clear red colloidal gold gave the same absorption spectrum in the diffuse-light absorption vessel as in the Beckman cell, with a maximum at about 520 mμ. Increasing the average particle size by addition of sodium chloride made the sol turn blue, with a strong Tyndall sheen, and caused the apparent absorption observed in the Beckman cell to be displaced to some wavelength beyond 600 mμ. The maximum observed in the diffuse-light absorption vessel remained between 540 and 580 mμ, at wavelengths that appeared to shift somewhat with the sol concentration.

The foregoing experiments deal with several examples of spectral absorption associated with light scattering, including, at the one extreme, the case of an optically "clear" absorber associated with colorless scattering elements and, at the other extreme, a colloidal suspension of particles that themselves absorb strongly. The results of these experiments give some indication of the scope and limitations of the new method. It has proved reliable, at least as far as first-order effects are concerned, in determining the spectral absorption of a continuous phase that separates nonabsorbing scattering particles and the spectral absorption of a continuous phase (concentrated hemoglobin solution) enclosed within a scattering envelope.

The method has also made it possible to observe the same spectral absorption of bacterial cells and the same changes associated with removal of molecular oxygen as those already demonstrated in more elaborate researches (*4*). It shares with these researches the limitation that the absorbing material in the bacterial cell is not necessarily manifested in its totality; in the new method, as in others, the absorbing material either may be opaque or may be shielded by scattering components.

The nature of the association between absorbing and scattering structures in living cells is undoubtedly complex; even when all scattered light is collected, the contributions of different substances to the total absorption may depend not only on their quantity but also on their location within the cell. We presume that the observations on a polydisperse colloidal gold illustrate how complex the situation may become when scattering and absorption are interdependent and when the absorption index is so high that at least some of the particles in the system are opaque.

J. B. Bateman
G. W. Monk

Camp Detrick, Frederick, Maryland

References

1. D. Burk, personal communication, and *Federation Proc.* **12**, 611 (1953).
2. O. Warburg and G. Krippahl, *Z. Naturforsch.* **9b**, 181 (1954).
3. See for example J. W. T. Walsh, "Photometry and Illumination," in R. Glazebrook, *A Dictionary of Applied Physics*, vol. 4 (Peter Smith, New York, 1950), pp. 410–464.
4. L. Smith. *Bacteriol. Revs.* **18**, 106 (1954).

27 December 1954.

Nutritional Changes in Diets Exposed to Ethylene Oxide

Ethylene oxide has been employed as an insecticide and bactericide for many years. It has been used in the food industry (*1*), and recent publicity (*2*) suggests that it may be a suitable sterilizing agent for a variety of products. This paper presents evidence that when diets are treated with ethylene oxide the nutritional properties are impaired.

The animals were female rats of the Osborne and Mendel strain. Except as otherwise noted, litter-mate controls were used and group mean starting weights were within the 38- to 40-g range. Each animal was housed individually on wide-mesh wire screen and given free access to food and water. The purified diet contained (in percentages) Labco casein 20, sucrose 69, Wesson oil 5 (*3*), Wesson salts 4 (*4*), and a vitamin mixture 2 (*5*). The stock diet was ground dog meal pellets (*6*).

For treatment with ethylene oxide, 500 g of diet was spread on large Petri dishes to a depth of 1 cm or less. The dishes were then stacked in a 10-lit desiccator (without desiccant). A small vent at the top prevented pressure accumulation and glass rods between the dishes assured free access of the gas to the diets. Ten milliliters (8.84 g) of liquid ethylene oxide

Table 1. Weight changes of rats fed diets exposed to ethylene oxide. The numbers in parentheses show number of rats dead at indicated time.

Diet	Exposure time (hr)	No. of rats	Weight gain (g) and standard error* at day			
			2	14	28	56
Purified	0	10	7 ± 1	64 ± 1	120 ± 3	
Purified	18	10	1 ± 1	22 ± 1	8 ± 2	
Stock	0	10	8 ± 1	63 ± 2	123 ± 4	172 ± 8
Stock	6	5	2 ± 1	41 ± 5 (1)	79 ± 8 (1)	120 ± 10 (1)
Stock	12	5	−2 ± 1	28 ± 3	62 ± 3	117 ± 7 (1)
Stock	18	5	−7 ± 1	5 ± 1	34 ± 3	86 ± 5
Stock	24	10	−6 ± 1	2 ± 3 (3)	15 ± 6 (3)	57 ± 10 (5)

* Standard errors determined by method of N. Mantel, *Am. Stat.* 5, 26 (1951).

(cold) was introduced, and the lid was rapidly replaced. After standing for the specified length of time at room temperature, the Petri dishes were removed and exposed to air overnight to dissipate residual gas before the diet was stored in metal cans. As an added precaution, some batches were subjected to a vacuum (pressure about 20 mm-Hg) for two 20-min periods with an intervening admission of air. This practice was discontinued when no difference was found in the growth response of rats fed the evacuated and non-evacuated diets.

The results summarized in Table 1 indicate severe damage to both purified and stock diets, but the effects produced by feeding the two differed in important respects. Growth was impaired after 2 days on the purified diet that had been treated for 18 hr. Thereafter the animals grew slowly for about 2 wk and then lost weight until they died in about 3 more weeks. The initial effect was more severe in the case of the 12-, 18-, and 24-hr treated stock diets. By the end of the first week 14 of the 20 animals had started to gain slowly and were still growing after 8 wk.

The deleterious effect of the purified diet treated with ethylene oxide was also seen in approximately 10-wk old animals (*7*) that had been fed the untreated diet since weaning. When these older rats were placed on the diet treated for 18 hr, they continued to grow for 7 to 10 days and then started losing weight, with the first death occurring 5 wk later. Food consumption was essentially unchanged for the first 2 days and thereafter declined to about 25 percent of the original intake. Animals that died on this regime had completely depleted their visible fat stores. Animals raised on the stock diet for approximately 7 wk and then changed to the stock diet treated in a similar manner with ethylene oxide immediately lowered their food consumption to about 70 percent of their original intake. Their body weights leveled off at that point.

That the treated purified diet might be deficient in thiamine was suggested by the fact that growth failure started at 2 wk and death followed 3 wk later. Analyses of this diet by the thiochrome method indicated that the thiamine was almost completely destroyed. Animals that had lost considerable weight on this diet gained an average of 38 g during the first week of oral supplementation with thiamine. Supplements of a complete vitamin mixture produced essentially the same results.

Thiamine hydrochloride dispersed in starch treated with ethylene oxide in the afore-mentioned manner retained its full activity. However, it was destroyed by similar treatment in the presence of choline chloride but not in the presence of choline dihydrogen citrate. When the starch mixture containing choline chloride was treated with ethylene oxide and then mixed with water, the resulting suspension was quite alkaline. This was not the case when choline dihydrogen citrate was substituted for the chloride. These findings indicate that the ethylene oxide does not react directly with thiamine. A partial answer to the mechanism whereby thiamine is destroyed may be the alkalinity produced under ethylene oxide treatment when choline chloride is added to the thiamine starch mixture.

A number of observations suggest that thiamine is not the only factor that is affected by ethylene oxide. Rats fed the treated purified diet with added untreated thiamine exhibited the initial growth inhibition. Thereafter they gained an average of 16 to 18 g/wk for 4 wk compared with an average of 30 g/wk for the controls on the untreated diet. At that time they appeared stunted but in reasonably good condition, whereas rats on the treated diet without thiamine were losing weight and were obviously near death. In addition, feeding an ethylene-oxide treated thiamine-deficient purified diet produced the initial growth inhibition, whereas the same diet without ethylene oxide treatment allowed continuous growth for the first 10 days. Thus far supplementation with thiamine or a complete vitamin mixture has not significantly stimulated growth of rats fed the treated stock diet.

It should be noted that the conditions that we employed in treating diets with ethylene oxide are comparable to some of those proposed for commercial use (*1*). The effectiveness of ethylene oxide in sterilizing our diets was tested with *B. globigii* spores (*8*). When diets were seeded with these spores, viable organisms could be recovered after 18-hr exposure to

ethylene oxide. Although all of the organisms were not destroyed by this treatment, there was a marked reduction in the count.

We wish to emphasize that we have not determined the effects of ethylene oxide on foods intended for human consumption or on practical animal foods other than our stock diet. Processing frequently is detrimental to the nutritional qualities of foods, and desirable changes must be weighed against the damage done. Nevertheless, in view of the afore-described results, it is suggested that foodstuffs, particularly those that may be major sources of essential nutrients, should not be subjected to ethylene oxide treatment until its effects have been established.

EDGAR A. HAWK*
OLAF MICKELSEN
Laboratory of Biochemistry and Nutrition
National Institutes of Health, Bethesda, Maryland

References and Notes

1. C. R. Phillips and S. Kaye, *Am. J. Hygiene* **50**, 270 (1949).
2. Anon. *Chemical Week* (2 Oct. 1954), p. 96.
3. Containing alpha-tocopherol acetate 10 mg and Natola (Parke, Davis and Co.) to supply vitamin A 550 USP units and vitamin D 110 USP units.
4. Salt mixture W, obtained from Nutritional Biochemicals Corp., Cleveland, Ohio.
5. Containing the following in milligrams per 100 g finished diet: thiamine HCl 0.4, riboflavin 0.6, pyridoxine HCl 0.5, nicotinic acid 4.0, Ca pantothenate 4.0, folic acid 0.2, biotin 0.02, choline chloride 200, inositol 20, and menadione 0.2 in cornstarch. Vitamin B_{12} (0.02 mg) was added separately as a water-alcohol solution.
6. Hunt Club dog meal, manufactured by Animal Foundation, Inc., Sherburne, N.Y.
7. These animals were divided into groups having comparable average weights when the treated diet was started.
8. The *B. globigii* spores were supplied through the courtesy of Charles R. Phillips and Saul Kaye, Camp Detrick, Frederick, Md. We also wish to acknowledge the valuable technical assistance of Ruth Clary and Samuel M. Takahashi.

* Present address: Medical Department, Upjohn Company, Kalamazoo, Mich.

23 November 1954.

On "Improving Scientific Communication"

It is refreshing to read the clear description that S. M. Garn [*Science* **121**, 7A (21 Jan. 1955)] has given of the diversification in requirements, specifications, and style in the imposing forest of scientific journals. The problem has been in existence a long time. It has been aggravated by the rapid increase in the number of journals in print. Authors have been unduly put upon to adhere to often silly differences among journals. Industrial and government laboratories have found it expedient to maintain files of journal specifications and assign personnel to become familiar with the journal idiosyncrasies. Academic authors must rewrite and check style mechanics with copies of a given journal before submitting an article for publication. Publication of valid research is in jeopardy unless these details are scrupulously adhered to in writing.

I urge that AAAS implement the wise suggestion made by Garn. A small steering committee should outline the items of confusion and invite 10 or 20 editors from the journals of largest circulation to study the problem and meet in plenary session to establish unanimity among the many mechanical impedimenta to manuscript preparation. Perhaps several of the larger trade journals should be included.

With such problems out of the way, authors, editors, and reviewers could devote increased attention to the more difficult problems of clarity and conciseness of expression, accuracy and originality of presentation.

W. H. WALDO
Kirkwood, Missouri

3 February 1955.

Science and Poetry

I have read with great interest the excellent editorial entitled "Science and poetry" and also several stimulating communications on the same subject [*Science* **120**, 17A and 951 (3 Dec. 1954)].

I was delighted to find that Phyllis McGinley was cited as holding the threads of the matter in the palm of her hand. I think she does. Of all her *Love Letters*, her "In praise of diversity" (which Louis Untermeyer, in the *Atlantic Monthly* for December, correctly called an "essay") seems most relevant to the point of issue.

I suggest that no discussion of this subject would be complete without reference to James B. Conant's Franklin Medal lecture of 19 Nov. 1943 before the American Philosophical Society on the general subject "The advancement of learning in the United States in the post-war world" [*Proc. Am. Phil. Soc.* **87**, 291 (1944)]. Among other things, Conant had this to say:

> Let me now turn from the first category—accumulative knowledge—to the other two which, following Bacon closely, I shall designate as poesy or, if you prefer, poetry and philosophy. Whereas the idea of progress is both valid and significant in the first category, accumulative knowledge, in the other two the concept is not only invalid but a positive deterrent to relevant undertakings. And at this point, lest all but scientists, mathematicians, and archaeologists leave the room in protest, I hasten to assert that I place no halo over the word progress. There is no hierarchy implied in my classification.
>
> Indeed, anyone who wished to give poetry or philosophy an inferior place as compared to accumulative knowledge would soon find himself in an untenable position. For it is obvious that poesy or poetry on the one hand and philosophy on the other together hold the keys to man's immediate future, including the future of the advance of accumulative knowledge. That this is so, current history provides ample proof. Nazism triumphed in Germany not because the Germans were lacking in power to advance learning but because bad poetry and a wrong philosophy prevailed. . . .
>
> One of the chief ends of education is surely to develop the capacity for making civilized judgments on all those matters of value which are involved in so many vital human decisions. Such judgments can be