viewed as a narcotic (Ebbecke, *Pflüg. Arch. ges. Physiol.*, 1935, **236**, 648). In our experiments the reversal of drug narcosis was always manifested before harmful effects of pressure became apparent. Following compression at 5,000 psi, gradual recovery in tap water at normal pressure often occurred, but some of the individuals died. Narcotized animals that were not subjected to pressure always recovered after transfer to tap water.

The basic mechanism through which temperature, pressure, and various drugs act on luminescence has been considered at some length (reviewed by Johnson, *Adv. Enzymol.*, 1947, **7**, 215). Heretofore, there has been no direct evidence that the same theory, specifically with reference to the influence of hydrostatic pressure on narcosis, applies in higher organisms, although other parallels have been found with reference to the influence of temperature. These relations support the general implications of the fundamental theory, and invite further study with various aquatic animals and narcotic agents.

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Erratum

In my recent paper "Concerning the Theory of Photoconductivity in Infrared-sensitive Semiconducting Films" (*Science*, 1950, **111**) page 685, line 7, column 2, should read ". . . to essentially pictures (1) or (2). . . ." rather than ". . . pictures (*10*) or (*5*). . . ." On page 687, line 5, column, 1, the words "fixed," "positive," and "electrons" should be deleted.

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Pancreatic Changes after Injection of Intermediary Fat Metabolites

It has already been reported (Nath, M. C., and Brahmachari, H. D. *Nature*, Lond., 1944, **154**, 487; and Nath, M. C., and Brahmachari, H. D. *Nature*, Lond., 1946, **157**, 335) that intermediary fat metabolites are responsible to a great extent for the onset of diabetic symptoms. Recently it has been found (Nath, M. C., and Brahmachari, H. D. *Nature*, Lond., 1948, **161**, 18; and Nath, M. C., and Brahmachari, H. D. *Indian J. med. Res.*, 1949, **37**, 71) that the guinea pigs injected with these substances show hypersecretion of insulin in the first stage. The animals begin to lose the activity of their pancreatic insulin after treatment with intermediary fat metabolites for about two months. A stage is reached in 70 days when the potency of the pancreatic insulin comes down to half the normal value.

This hypothesis has found support from A. Lazarow, who believes the effect of injected ketone bodies might account for the increased fasting blood sugar levels observed when men are placed on a high fat diet (*Physiol. Rev.*, 1949, **29**, 48); and the increasing demand for more and more insulin which results might increase the sensitivity of beta-cells to degeneration.

This prompted us to undertake histological examination, at different stages, of pancreatic cells from animals injected with β-hydroxy butyric acid (Na salt) in gradually increasing doses as mentioned hereafter.[1] The results of preliminary observations are indicated here:

First stage—The animals (rabbits) were killed on the 27th day.

1. The area of the islets of Langerhans increases; there are relatively fewer islets in a particular microscopic field in comparison with the normal pancreas.
2. There seems to be an increase in the number of cells in the islets as shown by their being very tightly packed.
3. The nuclei of these cells are large and appear to be active, as shown by staining with hemotoxylin.
4. However, the cells of pancreatic acini do not show any deviation from the normal.

Second stage—The animals were killed on the 53rd day of the experiment.

1. Islet cells do not show the close packing present in the first stage. There is a great amount of intercellular space.
2. The nuclei of these islet cells show distinct signs of degeneration. They take less stain and are therefore less clearly defined. The islet as a whole appears dull in contrast to the deep-staining cells of the pancreatic acini.
3. In at least one islet there is an invasion of the pancreatic blood capillary.
4. A curious feature is that the acinar cells do not seem to be affected at all and are normal.

These findings confirm the hypothesis, started by two of the authors in 1944 (Nath, M. C., and Brahmachari, H. D. *Nature*, Lond., 1944, **154**, 487) that keto acids might first stimulate the pancreatic islet cells and later cause lesions after fatigue through excessive work.

Further studies on detailed investigations are in progress.

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[1] The total number of animals used in the experiment was 12, of which 2 were killed after the 1st stage and an equal number after the 2nd stage. The remaining animals were used for glucose tolerance tests and other observations. The weight range of the animals selected was between 1.8 and 2.1 kg each, and the injections were given intramuscularly in the leg muscle every day after giving food. The initial daily dose of injection was about 50 mg per kg, which was increased by 7.5 mg per kg per week.

[2] The authors are grateful to Drs. K. Krishnamurti and M. A. Moghe for their kind interest and for the facilities they offered.

The Donora Episode—A Reply to Clarence A. Mills

There are many misleading statements in Dr. Mills' note regarding the U. S. Public Health Service's Bulletin 306 on the Donora episode (*Science*, 1950, **111**, 67). Neither of us has any connection whatever with the steel mill and zinc plant in the Donora area, or with the Public Health Service, but we wish to reply to Dr. Mills.

This is not a new field for the Public Health Service. One of us was a member of a committee set up about 1925 by Lewis R. Thompson, the former chief of the Industrial Hygiene Division of the PHS. On the committee was the late F. G. Cottrell, whose air-cleaning process is used all over the world. (It is used at Donora.) The late James E. Ives, a very fine physicist from the PHS, and Harry Meller, of the Mellon Institute, were also members of this committee. The question then vexing us was the effect of pollution on sunlight. Ives organized and carried out a nice piece of work which appeared as *U. S. Public Health Service Bulletin No. 224* in 1936. The field and laboratory work actually was done in 1931–33 and included pollution surveys in several major American cities chosen for industrial and topographic reasons. Other pollution studies by the PHS have followed.

Dr. Mills grumbles a bit because a grant he requested from the PHS for a pollution study was refused. Yet such grants are going to institutions in large industrialized areas capable of performing the work needed. What in the world is wrong with that? Where else should they go?

His comment on the effect of age on people in smogged areas is no new discovery. It was featured in the reports on the Meuse Valley (Belgium) disaster. We noted with much interest that W. P. D. Logan, an English investigator, analyzed deaths (*Lancet*, January 8, 1949, page 256) in the period November 26–December 1, 1948, when a heavy fog persisted in London. Deaths from bronchitis and pneumonia were 20%–30% higher than in any of the previous four weeks. It was observed that this increase came in persons over 45 and that infant mortality increased similarly. We realize that the PHS did not treat this aspect as a new discovery—because it wasn't new—but it is obvious to anybody that PHS was thoroughly aware of the facts and their implications.

Dr. Mills' calculations on pollution from nitrogen oxides emanating from steel and zinc plants are, we think, badly in error. The present accepted limits (American Standards Association) for 8-hr daily exposures is 25 ppm, which is equivalent to approximately 47 mg/m^3. Taking our measurements from the maps in the Donora report, the volume of the valley might be estimated as 0.22 cubic miles, based on an area of $2 \times 1\frac{1}{2}$ miles and 400 ft high—this takes in the highest stacks. Based on 4 tons of nitrogen oxides daily and complete diffusion, this could give a maximum theoretical daily concentration of 2.2 mg/m^3—far below the 8-hr threshold. This involves the assumption that the oxides remain unchanged chemically and stay put—which assumption is nonsense. The oxides are highly reactive and have never been found outdoors in significant concentrations.

Dr. Mills' statement that concentrations of zinc oxide and carbon monoxide reached dangerous levels implies that the operators of the plant were not utilizing the fuel value of CO from blast furnaces and were letting go zinc oxide in amounts that were industrially valuable. Neither implication fits the facts—blast furnaces produce enormous quantities of CO that are cleaned and used as fuel. No blast furnace could run if the CO were belched into the atmosphere—it would be economic suicide (See paper by H. O. Johnson in *Blast Furnace and Steel Plant*, 1944, 32, 44, in which a modern gas cleaning installation at Donora is described.) Zinc oxide makes a white smoke but it takes a very heavy concentration, such as 15 mg/m^3 and more, to affect man. Zinc retort plants, such as those at Donora, have been operating all over the world for years, and the health record of men inside the plants and of people in the communities has not been adversely affected.

Los Angeles is worried about its smog, and in that area it is estimated that about 700 tons of sulfur dioxide is emitted daily. Inversion layers occur quite often, yet the SO_2 does not build up to toxic levels. Why? Because it is eliminated and disseminated by natural processes.

The concentration of sulfuric acid mists and of particulate matter in the air determined at Donora is less than that found in London almost every winter. There are areas in England where SO_2 pollution actually has become a serious matter because of erosion of building stone—but the health record of the communities remains good. We've heard British visitors facetiously remark that our city air doesn't have enough body!

We admit that the Donora report has some weaknesses and omissions. We think it would be timely if an attempt were made to duplicate the Donora atmosphere on a pilot plant scale. Nobody proved what was the essential etiologic agent in the Meuse Valley disaster and nobody found it at Donora, although some very able men were on the spot. We do not feel that the Public Health Service's efforts should be belittled. They are of considerable value in pointing out that under usual conditions it is impossible to get disabling smog. Furthermore, we think the suggestion that the PHS continue the studies is timely and that the pollution meeting in Washington was a logical and practical step.

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Dr. Mills' Rejoinder

I am indeed very sorry that Drs. Silverman and Drinker should have been so misled by my remarks on the Public Health Service's Donora report, for I attempted to keep my statement free of ambiguity and inconsistency. Have they done as well?

(1) They disclaim any connection whatever with the Public Health Service—and in the next paragraph detail a former collaboration.

(2) This collaboration a quarter century ago marked the only PHS activity in the *community health aspects* of air pollution, and it dealt only with smoke's exclusion of sunlight, touching not at all on pollution's respiratory tract damage, which is today recognized as of paramount community importance.

(3) Silverman and Drinker show unfamiliarity with nitric oxide gases in claiming that they are never found outdoors in significant amounts. Have they never stood in the dispersion pathway of such gases escaping from