those competing with it from other laboratories. Scientists' motivations being what they are (including those at DBS), they cannot help but compromise situations where DBS scientists are asked to choose between two "alternatives," one developed by them and the other by outside scientists. We find just such a situation unfolding now, that is, monkey cell populations developed under contract to DBS, and quite naturally advocated by them, as compared to WI-38 developed by others and for which DBS even 10 years later still has "reservations" and "theoretical objections." It is by just such activities that the credibility gap between DBS and its constituency widens as they abrogate the very confidence on which their control authority rests.

As Petricciani *et al.* quite rightly point out in respect to passaged monkey cells, "further evaluation by other independent investigators will be necessary to increase the level of confidence in the safety of these cells." It is to be hoped that these important studies will be done exhaustively and that the decade of WI-38 vaccine testing required by DBS to increase their level of confidence in WI-38 will be equally applicable to vaccines prepared in DBS-FCL-1 and DBS-FRhL-2.

LEONARD HAYFLICK
Stanford University School of Medicine, Stanford, California

References and Notes

1. J. C. Petricciani, H. E. Hopps, D. E. Lorenz, *Science* **174**, 1025 (1971).
2. S. A. Plotkin, *Amer. J. Epidemiol.* **94**, 303 (1971).
3. D. B. Merchant, Ed., *Nat. Cancer Inst. Monogr.* **29**, 583 (1968); F. T. Perkins, Ed., *Minutes of the First through Seventh Meeting of the Cell Culture Committee of the Permanent Section on Microbiological Standardization, 1963–71* (Medical Research Council Laboratories, Hampstead, London, England, 1971).
4. L. Hayflick and P. S. Moorhead, *Exp. Cell Res.* **25**, 585 (1961).
5. C. M. Southam, A. E. Moore, C. P. Rhoads, *Science* **125**, 158 (1957); L. Hayflick, S. A. Plotkin, T. W. Norton, H. Koprowski, *Amer. J. Hyg.* **75**, 240 (1962).
6. F. T. Perkins and P. N. O'Donoghue, Eds. *Hazards of Handling Simians, Laboratory Animals Handbook No. 4* (Laboratory Animals Ltd., 7 Warwick Court, London, W.C.1, 1969).

27 December 1971; revised 16 February 1972

In 1967 a conference on cell cultures for virus vaccine production was held at the National Institutes of Health to review the state of the art so that a basis could be established for future progress.

One of the conclusions of that conference was expressed by the chairman, Donald Merchant, as follows: "A number of lines similar to WI-38 should be developed from human, nonhuman primate and other animal sources so that, as more information is obtained and the need for a wider variety of vaccines is apparent, we will have an ample number and variety of systems with which to work. A number of participants pointed out that we should not have all our eggs in one basket" (*1*).

One year later (1968) there was still no research activity in this area by any groups. The Division of Biologics Standards felt that, on the basis of the discussions and conclusions of the 1967 cell culture conference, it had an obligation to support studies to develop nonhuman diploid cell lines and began two such projects.

In our report (*2*) we were attempting to make available the information that diploid cells from nonhuman primates are now available to those who would study them. The eventual application of these cells depends on the results of a great deal of research by experienced investigators in cell biology and vaccine technology.

JOHN C. PETRICCIANI
HOPE E. HOPPS, DOUGLAS E. LORENZ
Laboratory of Pathology, Division of Biologics Standards, National Institutes of Health, Bethesda, Maryland 20014

References

1. D. B. Merchant, Ed., *Nat. Cancer Inst. Monogr.* **29**, 583 (1968).
2. J. C. Petricciani, H. E. Hopps, D. E. Lorenz, *Science* **174**, 1025 (1971).

12 April 1972

Turbidity of the Atmosphere: Source of Its Background Variation with the Season

In a recent report concerned primarily with the long-term trend in atmospheric turbidity indicated by data from the Mauna Loa Observatory, Ellis and Pueschel (*1*) raised another issue. Considering only "control" days (of atmospheric uniformity in addition to clearness), they found annual cycles in the intensity of solar radiation recorded at the Mauna Loa Observatory between 1958 and 1970. They interpreted these as indicating reduced atmospheric transmissivity during the summer months, "most likely the result of increased worldwide photochemical aerosol formation caused by the oxidation of volatile organic materials of plant origin in the atmosphere . . . or the result of the seasonal variations in general atmospheric circulation, or both." They then concluded: "From the time scales of recovery it can be concluded that such an aerosol is confined, for the most part, to the troposphere."

Dyer and Hicks (*2*) analyzed solar radiation data on "clear" days for the period 1961 through 1965 and found, subsequent to the eruption of Mount Agung, Bali, in 1963, maxima in turbidity moving toward the poles with a period of approximately 1 year. Near the equator the maxima occurred in summer (in agreement with data from Mauna Loa Observatory) but successively later with increasing latitude so that the maxima appeared in winter at mid-latitude stations. They attributed this phenomenon to an annual cycle or equatorial stratospheric dust (presumably from Mount Agung) being fed alternately into each hemisphere.

Three points appear clear: (i) atmospheric turbidity, determined predominantly by the composition of the troposphere, has a pronounced summer maximum (*3*); (ii) measures of stratospheric turbidity, particularly those attributed to volcanic dust, display winter maxima in mid-latitudes (*4, 5*); and (iii) the stratosphere is capable of significant variations in composition on a time scale of 1 year (*6*). What remains unclear is whether the use of solar radiation data on "clear" or "control" days reveals atmospheric turbidity variations reflecting changes in tropospheric composition as postulated by Ellis and Pueschel (*1*) or changes in stratospheric composition as claimed by Dyer and Hicks (*2*).

The available data appear to admit both claims. At Mauna Loa the stratospheric and tropospheric variations are presumably in phase and could produce the single summer maximum observed by Ellis and Pueschel (*1*). Subsequent to the Mount Agung eruption, the stratospheric variations could predominate (on "control" days), producing the single winter maximum in mid-latitudes claimed by Dyer and Hicks (*2*). Prior to the Mount Agung eruption, the two variations could be of comparable magnitude (on "control" days) producing in mid-latitudes either a biannual or an ill-defined seasonal variation as suggested by the analysis of Dyer and Hicks (*2*) for this period.

Volz rejected the analysis by Dyer and Hicks (*2*) because "optical soundings of the stratosphere indicated no stratospheric dust amounts and no seasonal variations of such magnitude" (*7*) and because Dyer and Hicks failed to detect an increase in stratospheric dust in summer after the Mount Agung eruption (*8*). However, less violence is done to the literature on this subject by rejecting Volz's (*7*) data for the Northern Hemisphere since they reveal neither the general rise in optical thickness after the Mount Agung eruption (*1*, *4*) nor its seasonal fluctuations (*4*, *9*).

HUGH W. ELLSAESSER
Lawrence Radiation Laboratory, University of California, Livermore 94550

References and Notes

1. H. T. Ellis and R. F. Pueschel, *Science* **172**, 845 (1971).
2. A. J. Dyer and B. B. Hicks, *Quart. J. Roy. Meteorol. Soc.* **94**, 545 (1968).
3. E. C. Flowers, R. A. McCormick, K. R. Kurfis, *J. Appl. Meteorol.* **8**, 955 (1969).
4. F. E. Volz, *J. Geophys. Res.* **75**, 1641 (1970); F. Burdecki, *Weather* **1964**, 113 (1964).
5. W. M. Irvine and F. W. Peterson, *J. Atmos. Sci.* **27**, 62 (1970).
6. Most unambiguously documented in the case of O_3 but also apparent in measurements of stratospheric H_2O and dust [see (*4*, *5*)].
7. F. E. Volz, *J. Geophys. Res.* **75**, 5185 (1970).
8. ———, personal communication.
9. G. Grams and G. Fiocco, *J. Geophys. Res.* **72**, 3523 (1967).
10. Performed under the auspices of the U.S. Atomic Energy Commission.

15 November 1971; revised 22 February 1972

We have subjected the data from Mauna Loa Observatory to a harmonic analysis with the following results: (i) Since about 1970, the annual average amount of atmospheric turbidity is the same as it was prior to 1963; (ii) monthly averages of turbidity data for the period 1970–1971 agree in amplitude and phase with the annual periodicity observed for 1958–1962; (iii) the annual average as well as the amplitude of the periodic turbidity function are significantly reduced for a 5-year period of strong volcanic activity (*1*) following the eruption of Mount Agung in March 1963.

These results substantiate what we reported earlier (*2*). In particular, they contradict the finding by Dyer and Hicks (*3*) of an increased annual turbidity amplitude after 1963, which is the basis of Ellsaesser's argument that the seasonal turbidity fluctuations observed on Mauna Loa reflect changes in the stratospheric aerosol load. Confined to the stratosphere or troposphere, these fluctuations apparently are characteristic of the variations in the atmospheric background aerosol. Any perturbation in the amplitude or phase of the annual periodicity, or both, such as was the case during 1963–1969, are indicative of a change in the colloidal composition of the atmosphere. We wish to emphasize again that such perturbations should be utilized to evaluate man's impact on our atmospheric environment.

R. F. PUESCHEL
H. T. ELLIS
National Oceanic and Atomspheric Administration, Mauna Loa Observatory, Hilo, Hawaii 96720

References

1. J. F. Cronin, *Science* **172**, 847 (1971).
2. H. T. Ellis and R. F. Pueschel, *ibid.*, p. 845.
3. A. J. Dyer and B. B. Hicks, *Quart. J. Roy. Meteorol. Soc.* **94**, 545 (1968).

30 March 1972

Galactose Toxicity in the Chick: Hyperosmolality or Depressed Brain Energy Reserves?

In a recent report Malone *et al.* (*1*) described the close correlation of hyperosmolality with elevated galactose concentrations in the serum of chicks fed water containing 10 g of galactose per 100 ml. Since observations were made over a period during which galactose exerted an extremely toxic effect on the chicks and during which high rates of mortality occurred (3 to 5 days after the initiation of the experiment), Malone *et al.* stated that severe hyperosmolar dehydration could be responsible for the entire galactose toxicity syndrome. However, we have concluded, as a result of recent experiments, that hyperosmolality per se is not the major factor responsible for the galactose toxicity syndrome in the chick. We have observed both physical and biochemical recovery from the neurotoxicity upon elevation of blood glucose concentrations. On the basis of previous studies (*2–4*) and evidence presented here, we propose that galactose interferes with the supply of glucose to the brain, thus affecting brain energy metabolism.

Two-day-old male Leghorn chicks were obtained from Klager Hatcheries, Bridgewater, Michigan and were maintained in a brooder at 32°C. The animals were fed a semisynthetic diet (*5*), 40 percent (by weight) of which was replaced with D-galactose at the expense of Cerelose. Osmolality was determined with an osmometer (Fiske G-66) on plasma samples obtained by heart puncture. Both preparation and analysis of brain metabolites were performed as previously described (*2*).

After chicks had been fed their respective diets for 48 hours (control diet, group A; galactose diet, groups B, C, and D), animals in group C were injected intraperitoneally with 1 ml of a 1*M* D-glucose solution and those in group D were injected intraperitoneally with 1 ml of 0.5*M* NaCl solution. The chicks in group C no longer exhibited convulsions and tremors, whereas the appearance of those chicks in group D was unchanged. The duration of the physical recovery correlated with an increase in the concentrations of plasma glucose (Table 1). Similarly, during the recovery phase, the brain concentrations of adenosine triphosphate (ATP), phosphocreatine, glucose, fructose-1,6-diphosphate, and lactate returned virtually to normal values (Table 2, group C), whereas injection of saline had essentially no effect on the concentrations of these metabolites (Table 2, group D).

As previously reported (*2*, *4*), the inclusion of galactose in the diet resulted in depressed concentrations of brain glucose and glucolytic intermediates (Table 2, group 8) without affecting the blood glucose concentrations (Table 1, group B). However, when the plasma glucose concentration was significantly elevated, the concentrations of brain metabolites approached those of normal chicks. Thus, these experiments support our contention that galactose interferes with glucose transport across the blood brain interface and that this interference may be the major cause of the neurotoxicity in chicks.

The suggestion by Malone *et al.* (*1*) that hyperosmolality may be the leading factor in galactose neurotoxicity in the chick cannot be inferred from our experiments. Osmolalities were determined on plasma simultaneously analyzed for glucose and galactose concentrations, and are listed in Table 1. Severely disabled chicks (group B) had plasma osmolality values (in milliosmoles per kilogram) of 335 ± 10 as compared with 309 ± 7 for controls. The injection of glucose, which temporarily reversed the neurotoxicity, further elevated the osmolality by 12 (347 ± 7) after 20