Table 2. The effects of immune lysis on the aerobic glycolysis of chicken erythrocytes. "Sensitized" cells were first incubated with an active human serum in the presence of 0.01*M* EDTA. "Native" cells were first incubated in the absence of active human serum but in the presence of 0.01*M* EDTA. GPS, guinea pig serum.

Addition	Free hemoglobin (g/100 ml)	Lactate change (mg/100 ml)
Native cells		
None	0.02	−0.35
GPS (undiluted)	.00	+0.46
Sensitized cells		
None	0.01	+0.06
GPS (undiluted)	.47	+4.08
GPS (diluted 1 : 2)	.43	+2.15
GPS (diluted 1 : 3)	.34	+1.33

tive glycolysis than did disruption by rapid freezing in the absence of added nucleotides. If the incubation mixtures were fortified with adenosine triphosphate, diphosphopyridine nucleotide, and nicotinamide, freezing and thawing exerted as great a stimulatory effect as the sound treatment did.

Cellular loss of potassium began immediately upon mixing human serum with chicken erythrocytes at 37°C (Fig. 2*a*). The early, rapid leak of potassium also suggests that the initial lesion occurs at the plasma membrane.

Saponin causes the formation of discrete plasma membrane lesions (*7*). In a concentration that causes progressive, though not complete, hemolysis of a 33-percent suspension of chicken erythrocytes during a 1-hour incubation, saponin induced loss of potassium ion, hemolysis, and glycolysis qualitatively similar to that induced by active human serum (Fig. 2*b*).

In order to determine whether complement was required for hemolysis (*8*), chicken erythrocytes were incubated with human serums that had been heated at 56°C for 30 minutes to inactivate complement. Under these conditions, the serums did not induce K^+ loss, hemolysis, or glycolysis. However, activity could be partially restored by the addition of guinea pig complement. In other experiments, complement was inhibited by carrying out the incubation in the presence of 0.01*M* ethylenediaminetetraacetate (EDTA) (*11*). Under these conditions, hemolysis and glycolysis did not occur. The antibody, however, appeared to be active because the cells agglutinated. The agglutinated cells, washed free of serum and EDTA, could be hemolyzed by the addition of guinea pig complement. In the presence of glucose, hemolysis was accompanied by active glycolysis (Table 2). Complement had no effect on cells not previously sensitized.

The appearance of active aerobic glycolysis after damage to tissues capable of the oxidative metabolism of glucose has been interpreted to be release from the Pasteur effect (*10*). Intact chicken erythrocytes are impermeable to glucose, and one might postulate that the appearance of glycolysis after damage to the plasma membrane is a "permissive" release of glycolysis rather than true stimulation or activation (*11*). Our data suggest that human plasma or serum damages the cell membrane of chicken erythrocytes and subsequently causes increased permeability to glucose. It appears likely that these effects are due to the reaction of a heterogenetic antigen of chicken erythrocyte plasma membrane with an antibody of human serum and complement. The distribution of the antibody in populations of mental patients and control normal subjects is still undetermined (*12*).

JAMES W. RYAN*
JAMES D. BROWN
JACK DURELL

Laboratory of Clinical Science, National Institute of Mental Health, Bethesda, Maryland

References and Notes

1. C. E. Frohman, N. P. Czajkowski, E. D. Luby, J. S. Gottlieb, R. Senf, *A.M.A. Arch. Gen. Psychiat.* **2**, 263 (1960).
2. C. E. Frohman, G. Tourney, P. G. S. Beckett, H. Lees, L. K. Latham, J. S. Gottlieb, *ibid.* **4**, 404 (1961).
3. E. E. Ecker and L. Pillemer, *J. Immunol.* **40**, 73 (1941).
4. This serum was obtained from Suburban Serological Laboratories, Silver Spring, Maryland.
5. D. L. Drabkin, *Am. J. Med. Sci.* **215**, 110 (1948).
6. G. F. Olson, *Clin. Chem.* **8**, 3 (1962); J. A. Gloster and P. Harris, *Clin. Chim. Acta* **7**, 206 (1962); G. R. Kingsley and G. Getchell, *Clin. Chem.* **6**, 466 (1960).
7. R. R. Dourmashkin, R. M. Doughtery, R. J. C. Harris, *Nature* **194**, 1116 (1962).
8. W. M. Karshner, *J. Lab. Clin. Med.* **14**, 346 (1928–29).
9. L. Levine, K. M. Cowan, A. G. Osler, M. M. Mayer, *J. Immunol.* **71**, 359 (1953).
10. A. C. Aisenberg, in *The Glycolysis and Respiration of Tumors* (Academic Press, New York, 1961), p. 16.
11. C. E. Shields, Y. F. Herman, R. H. Herman, *Nature* **203**, 935 (1964).
12. J. W. Ryan, J. D. Brown, R. Green, H. R. Steinberg, J. Durell, in preparation.
13. We thank Dr. C. Frohman and his collaborators for their assistance in developing the methodology for these studies and Drs. R. Green and H. R. Steinberg for psychiatric evaluations of patients for whom data is included in this manuscript.
* Present address: Radcliffe Infirmary, Oxford, England.

1 February 1966

"Respirable" Dust

In "Airborne particulates in Pittsburgh: association with *p,p'*-DDT" (*1*), Antommaria and co-workers refer to a paper of mine (*2*) as a source for the term "respirable" dust. According to their account, they measured by a two-stage sampler the dust which could penetrate and deposit in the lower respiratory tract (terminal airways and alveoli) and thereby that which constituted the greatest risk to health. This dust, they say, corresponds to the "respirable" fraction. Their use of this expression does, in fact, conform to the original use by the British Medical Research Council in discussing the risk of pneumoconiosis from coal dust (*3*); however, the MRC description cannot be applied to dust generally.

The study of *p,p'*-DDT aerosol is a case in point. If DDT, alone or associated with a vector aerosol, is absorbed reasonably well from mucous membranes and has important systemic toxic effects—and both these characterizations appear appropriate (*4*)—then the deposition site of the dust becomes relatively unimportant, because all the DDT will be absorbed and potentially injurious.

The authors do not discuss the toxicity of DDT aerosols, and it is conceivable that they have evidence of an important pulmonary effect; nevertheless, it must be stressed that "respirable" dust and "respirable" fraction are ambiguous and changeable terms. At best, they are used to describe the dust which constitutes the greatest risk to health; the site of the injury can vary, as will concern over the sites of dust deposition. The terms, therefore, connote different things for different dusts. To complicate matters further, occasionally the expressions will pertain to the dust which is literally respirable, that is, the dust which accompanies the inspired air into the respiratory tract.

P. E. MORROW

Department of Radiation Biology and Biophysics, University of Rochester, Rochester, New York

References

1. P. Antommaria, M. Corn, L. DeMaio, *Science* **150**, 1476 (1965).
2. P. E. Morrow, *Am. Ind. Hyg. Assoc. J.* **25**, 213 (1964).
3. R. J. Hamilton and W. H. Walton, *Inhaled Particles and Vapours* (Pergamon, New York, 1961), p. 465.
4. L. Goodman and A. Gilman, *The Pharmacological Basis of Therapeutics* (Macmillan, New York, ed. 3, 1965).

27 December 1965