globulin neutralized both agents and was approximately 65 percent effective in decreasing the mean symptom score caused by either one. Apparently these agents are antigenic in the human being, and they or related agents have caused rather widespread infections and antibody response. Nonspecific inhibition of the infectious agents is a possibility, but other experiments have discounted this (*3*).

One possible explanation of the lack of resistance against the common cold is that circulating antibody can protect against a systemic infection but is of little importance in the prevention of a local infection of the mucous membranes of the upper respiratory tract. We have shown that the common cold in adults is an afebrile and presumably localized respiratory infection (*1*), whereas children have more systemic symptoms. There is some evidence that local infections with other viruses also might occur in the presence of circulating antibody (*9*).

Prior studies in our laboratory have demonstrated that gamma globulin is consistently found in nasal secretions. It is not clear why the antibody is ineffective in the nose. The concentration of specific antibody might be critical, or inhibitory substances might block the antibody effect. Possibly cellular resistance is independent of antibody. Finally, antigenic variants of the agents that cause common colds might produce recurrent infection.

The demonstration of protective antibodies against the common cold in human gamma globulin is noteworthy with respect to the problem of immunity and immunization against this illness. Further exploration of some of the factors discussed is necessary before it will be known whether it is feasible to protect against common colds by the administration of vaccines.

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References and Notes

1. G. G. Jackson *et al.*, *A.M.A. Arch. Internal Med.* 101, 267 (1958).
2. C. H. Andrewes, *Brit. Med. J.* 1953, No. 9, 206 (1953); Commission on Acute Respiratory Diseases, Armed Forces Epidemiology Board, *J. Clin. Invest.* 26, 957 (1947); A. R. Dochez, G. S. Shibley, K. C. Mills, *J. Exptl. Med.* 52, 701 (1930); G. B. Foster, *J. Infectious Diseases* 21, 452 (1917); W. Kruse, *Münch. med. Wochschr.* 61, 1547 (1914).
3. G. G. Jackson *et al.*, unpublished observations.
4. G. F. Badger *et al.*, *Am. J. Hyg.* 58, 31 (1953).
5. W. H. Price, *Proc. Natl. Acad. Sci. U.S.* 43, 790 (1957).
6. These studies were carried out under the sponsorship of the Commission on Acute Respiratory Diseases of the Armed Forces Epidemiology Board and were supported in part by the Office of the Surgeon General, Department of the Army, and in part by grants from the Lederle Laboratories Division of the American Cyanamid Co. and from Charles Pfizer and Co., Inc. We gratefully acknowledge the assistance of Delores Seketa, Louise Jensen, and Marvin Turck, who participated in conducting the experiments.
7. The solution of pooled human plasma proteins was kindly furnished by Lederle Laboratories Division of the American Cyanamid Co., Pearl River, N. Y., and by Armour Laboratories, Chicago, Ill.
8. Neutralization tests for 2060 and JH viruses were performed by W. J. Mogabgab of Tulane University School of Medicine.
9. J. A. Bell *et al.*, *J. Am. Med. Assoc.* 165, 1366 (1957); M. J. Lipson, F. C. Robbins, W. A. Woods, *J. Clin. Invest.* 35, 722 (1956).

6 March 1958

In vitro Decomposition of 1-Adrenochrome

1-Adrenochrome has been reported to produce psychotomimetic effects following intravenous injection (*1, 2*), and it has also been reported to be markedly elevated in the blood of normal subjects receiving lysergic acid diethylamide (*3*). In view of the reported instability of adrenochrome (*2, 4*) in the solid state and in solution prior to its injection, a study of the variables affecting decomposition was made (*5*).

1-Adrenochrome (*6*) was prepared as follows: To 176.5 mg of synthetic 1-epinephrine in 5 ml of redistilled methanol there was added 0.1 ml of 98-percent formic acid. One gram of freshly prepared silver oxide was added, with stirring. A deep red color developed immediately, and the temperature rose to 42°C. The reaction mixture was stirred for 2 minutes and centrifuged at high speed, and the supernatant solution was allowed to stand at −20°C for ½ hour. The precipitate was filtered and washed with a small volume of cold methanol-ether (1:1) followed by methanol-ether (1:3) and, finally, with ether to remove all traces of methanol. The yield of 1-adrenochrome was 54.3 mg (32.5 percent).

The absorption spectra showed three maxima, at λ-220, λ-300, and λ-480 mμ. The 480-mμ peak was used to follow the decomposition of 1-adrenochrome in 0.9-percent saline (*p*H 7.0), redistilled water (*p*H 7.0), and methanol solutions. It was found that the stability at room temperature (22°C) was greatest in sodium chloride solution and least in methanol solution. The decomposition was approximately 2.5 percent in saline, 5 percent in water, and 10 percent in methanol in 12 hours in the concentration range of 15 to 25 μg/ml and was approximately proportional to the time for at least 48 hours. It also was found that concentration affected the rate of decomposition. Thus, in saline solution, 3.8 mg of 1-adrenochrome per milliliter decomposed to the extent of 34 percent in 12 hours at room temperature, whereas, at 3.4 μg/ml, decomposition was not measurable even after 96 hours. Temperature also affected the decomposition. A solution of 1.9 mg of 1-adrenochrome per milliliter in 0.9-percent sodium chloride solution decomposed to the extent of more than 60 percent in 24 hours at room temperature but only to the extent of 4 percent at 3°C or at −20°C. The *p*H is also an important variable. Zambotti and Moret (*7*) reported that in 0.9-percent saline solution at 37°C in 2 hours there was a loss of 25 percent at *p*H 5.91 and of 90 percent at *p*H 7.38.

The decomposition of 1-adrenochrome in the solid state stored for 3 months at room temperature in an ordinary capped glass vial was also studied. No changes were observed in the infrared spectra obtained in the solid state in a potassium bromide pellet. The spectrum of the 1-adrenochrome synthesized by the method described was identical in all respects with the spectrum of a sample of 1-adrenochrome synthesized by the method of Sobotka and Austin (*8*), which was obtained from the Research Division of Armour and Co.

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References and Notes

1. A. Hoffer, H. Osmond, V. Smythies, *J. Mental Sci.* 100, 29 (1954).
2. H. Osmond, in *Neuropharmacology* (Josiah Macy, Jr. Foundation, New York, 1956), p. 183.
3. A. Hoffer, paper presented at Sci. Conf. Brain Research Foundation, New York, N.Y., 27 Jan. 1958.
4. D. E. Green and D. Richter, *Biochem. J.* 31, 596 (1937); D. J. Ingle, D. A. Shepherd, W. V. Haines, *J. Am. Pharm. Assoc. Sci. Ed.* 37, 375 (1948); F. M. Bacq, *Pharmacol. Revs.* 1, 1 (1949).
5. I wish to acknowledge the technical assistance of Jerry Murphy, precollegiate science summer student, St. Mark's School. This project was completed under grants from the Ford Foundation and the U.S. Public Health Service (B-713).
6. C. L. McCarthy, *Chem. & Ind. (London)* 55, 435 (1946).
7. V. Zambotti and V. Moret, *Arch. sci. biol. (Bologna)* 33, 522 (1949).
8. H. Sobotka and J. Austin, *J. Am. Chem. Soc.* 73, 3077 (1951).

3 February 1958

Cytochemical Demonstration of Masked Lipids

There are several methods for the cytochemical or histochemical *in situ* demonstration of free lipids, or of lipids which give general or special color reactions (*1*). However, the larger fraction of lipids in ordinary cells (excluding fat cells or similar tissues) belongs to the class of, until now, cytochemically nondemonstrable or masked lipids, which should form lipoprotein complexes or complexes of a higher order. For instance, in calf-liver cells it has been possible, by extraction with suitable salt