values of the heat and temperature of essentially isothermal phase changes; and an estimate of the accuracy uncertainty of the results. These primary experimental data should be supplemented, but never supplanted, by a tabulation of smoothed values of thermodynamic properties at selected temperatures. Where applicable, such tabulations should include values of Gibbs free energy function, $(F-H°_0)/T$, enthalpy (heat content) function, $(H-H°_0/T$, entropy, S, heat capacity, C_p (or C_{sata}), and enthalpy, $H-H°_0$, at 5° intervals from 0 to 50°K, 10° intervals from 50 to 300°K or slightly higher temperatures, and 50° to 100° intervals at higher temperatures, with appropriately smaller intervals in regions of thermal anomaly. Values at the two important reference temperatures, 273.15 and 298.15°K, and at the temperatures of phase changes should be included in the tabulations. Graphical or analytical representation of the results is sometimes worth while for the convenience of the reader, but such representations seldom are a satisfactory substitute for tabular presentation of accurate experimental results.

Calculated thermodynamic functions. As the usefulness of calorimetric data often is extended by giving calculated thermodynamic functions based on them, recommendations for presenting this kind of thermodynamic data are included here. The following information, with sources, is needed to characterize the results of statistical thermodynamic calculations: Details of the molecular model used, including bond distances and angles, specification of the exact conformation, moments of inertia or rotational constants, and symmetry number; complete vibrational assignment; parameters used for calculating contributions of internal rotation, anharmonicity, centrifugal distortion, etc.; citation, usually by reference, of formulas and special tabulations used; comparison with experimental thermodynamic data when available; and tables of thermodynamic functions at selected temperatures. The functions tabulated should include all of the following: Gibbs free energy function, $(F° -H°_0)/T$; enthalpy (heat content) function, $(H° -H°_0)/T$; entropy, $S°$; heat capacity, $C_p°$; and enthalpy, $(H° -H°_0)$. In addition, values of enthalpy of formation, $\Delta Hf°$, Gibbs free energy of formation, $\Delta Ff°$, and common logarithm of the equilibrium constant of formation, $\log_{10}Kf$, may be published if warranted. The values should be reported at temperatures so spaced that no serious loss of accuracy will result by interpolation with a formula equivalent to 5-point Lagrangian interpolation; for example, at 50° intervals to 300°K, 100° intervals to 1500°K, 200° intervals to 2500°K, and 500° intervals at higher temperatures. Values also should be tabulated at the reference temperatures, 273.15 and 298.15°K.

Nondefinitive data. This resolution is concerned primarily with the publication of precise and accurate data taken by definitive techniques, but rough measurements often are made for technical purposes and these data occasionally are submitted for publication. As such measurements are sometimes made on materials of undefined composition or by techniques substantially inferior to those accepted as definitive, they clearly do not merit space in scientific journals on the same basis as definitive studies, and the foregoing recommendations do not apply in full. The same is true of calculated thermodynamic functions that are based on unsubstantiated or estimated molecular data and that are not verified by comparison with experimental thermodynamic data. However, even rough data or calculated values *may* be better than empirical estimates and, so, have some value, but they are of doubtful significance as a basis for many theoretical deductions or for incorporation in critical tables of scientific data. Therefore, it is recommended that minimum journal space be allotted to such results and that the presentation clearly recognize their lack of reliability. The use of the American Documentation Institute supplement may be appropriate for the bulk of such data, with the location attested only by a brief note in a journal.

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Note

1. The use herein of symbols and terminology accepted generally by American scientists is not intended as a recommendation of the Calorimetry Conference. Appropriate international organizations are now developing a uniform system of symbols and terminology for thermodynamics. Calorimetrists are urged to follow the international system when it is officially adopted.

21 October 1960

Action of 1-Benzyl-2-methyl-5-methoxytryptamine on Monoamine Oxidase

In their report on monoamine oxidase, psychoenergizers, and tranquilizers, A. Feldstein, H. Hoagland, and H. Freeman [*Science* **130**, 500 (1959)] cast "some doubt on the hypothesis that Marsilid [iproniazid] exerts its central stimulatory action by virtue of its ability to inhibit monoamine oxidase." Their objection to this hypothesis stems from the assumption that 1-benzyl-2-methyl-5-methoxytryptamine (BAS), a tranquilizing agent, and iproniazid, a psychoenergizer, both block monoamine oxidase. The authors interpret their data by referring to the work of D. W. Woolley *et al.* (*1*), who observed an increase of urinary serotonin excretion in mice after the administration of BAS and who ascribed this phenomenon to monoamine oxidase inhibition. However, no data have as yet been published regarding the influence of BAS on this enzyme.

Since the authors' argument against the monoamine oxidase hypothesis has already been repeated elsewhere (*2*) it seemed necessary to test directly the effect of BAS on monoamine oxidase. Recently this was done in my laboratory (*3*) with various preparations—for example, beef liver mitochondria and human and mouse liver homogenates which were suspended in tris-buffer. According to our standard procedure (*4*), the inhibitory power of BAS turned out to be very low ($pI_{50} \geqq 3$). It seems, therefore, not very likely that BAS acts by way of monoamine oxidase, and the biological effects of this substance, as described by Feldstein *et al.*, may have to be explained differently.

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References and Notes

1. D. W. Woolley and P. M. Edelman, *Science* **127**, 281 (1958).
2. Editorial, *Brit. Med. J.* **1959**, **II**, 1238 (1959); L. Rees, *Nature* **186**, 114 (1960).
3. I am indebted to Dr. D. W. Woolley for a sample of BAS. Most of the experiments were carried out by Sama E. Sama.
4. E. A. Zeller, J. Barsky, E. R. Berman, M. S. Cherkas, J. R. Fouts, *J. Pharmacol. Exptl. Therap.* **124**, 282 (1958).

16 June 1960

We reported (*1*) the administration of 100 mg of DL-5-hydroxytryptophan (DL-5-HTP) to patients pretreated with 1-benzyl-2-methyl-5-methoxytryptamine (BAS). We found, unexpectedly, small amounts of 5-hydroxyindoleacetic acid (5-HIAA) determined quantitatively by the Udenfriend extraction procedure and semiquantitatively by two-dimensional paper chromatograms of the neat urine. We also found major spots corresponding in R_F value and Ehrlich color to 5-hydroxytryptophan (5-HTP), 5-HIAA, and serotonin. We interpreted the data as meaning that BAS was a monoamine oxidase inhibitor which blocked the formation of 5-HIAA and caused an accumulation of serotonin.

Recently we investigated the intravenous administration of 5 μc of DL-5-HTP-C^{14} to five untreated chronic-schizophrenic patients. The recovery of urinary 5-HIAA-C^{14} based on administered counts was 22.0 percent.

Autoradiographs prepared from two-dimensional paper chromatograms (solvent systems: isopropanol-ammonia-water and n-butanol–acetic acid–water) of the neat urine again revealed three major spots corresponding in R_F values to 5-HTP, 5-HIAA, and serotonin. These results, obtained with untreated patients, are similar to those obtained with the BAS-treated patients; obviously the action of BAS on monoamine oxidase cannot be the explanation.

A comparison of the ability of BAS and iproniazid to block the conversion of 10 mg of serotonin to urinary 5-HIAA, administered intraparitoneally to rats, indicated that iproniazid at 100 mg/kg effectively blocked monoamine oxidase, whereas BAS up to 200 mg/kg did not. Similarly, the formation of endogenous 5-HIAA in the rat was blocked by iproniazid but not by BAS.

Zeller (*2*) has shown that BAS is not a monoamine oxidase inhibitor. Evidence we have obtained since our initial publication (*1*) indicates that Zeller is right and that we misinterpreted our earlier data (*3*, *4*).

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References and Notes

1. A. Feldstein, H. Hoagland, H. Freeman, *Science* **130**, 500 (1959).
2. E. A. Zeller, *Science*, this issue.
3. We wish to thank Marilyn George and William Connors for technical assistance. We also wish to acknowledge grants from the Ford Foundation and the Scottish Rite Committee on Research in Schizophrenia.
4. S. Spector, P. A. Shore, and B. B. Brodie, [*Science* **132**, 735 (1960)] have also noted that BAS is not a monoamine oxidase inhibitor. Their article appeared after we submitted for publication this reply to Zeller, and our answer also applies to their report.

19 August 1960

Virus Isolated from Inclusion Conjunctivitis of Newborn (Inclusion Blennorrhea)

Abstract. An infant developed acute conjunctivitis 7 days after birth. Smears of the mucopurulent discharge contained many typical elementary-body inclusions. From conjunctival scrapings a virus resembling trachoma was grown in eggs. When instilled into monkey eyes, it produced an acute conjuctivitis resembling the human disease. Nine other patients with inclusion conjunctivitis of similar intensity failed to yield viruses.

Inclusion conjunctivitis is an acute nonbacterial eye disease most commonly observed in newborn children. It begins in the first 2 weeks of life with redness, edema, infiltration of the conjunctiva, and a mucopurulent exudate. It does not involve the cornea, and the conjunctiva heals spontaneously in weeks or months without scarring. With the application of sulfonamide or tetracycline drugs to the conjunctiva, the infection regresses in a few days. Fifty years ago it was shown that in smears from infected conjunctiva some epithelial cells contained inclusions resembling those of trachoma (*1*). Similar inclusions were demonstrated in epithelial cells from the mother's cervix (*2*). While it is evident that in the newborn the infection is acquired from the mother's genital tract, direct-contact transmission to the adult eye is occasionally observed (*3*).

The epithelial cell inclusions, composed of elementary or initial bodies, are morphologically similar to those of the psittacosis-lymphogranuloma venereum-trachoma group of viruses. However, in spite of the profusion of viral particles seen in smears from inclusion conjuctivitis of the newborn, the virus has not been grown in many attempts made since 1910. When a successful method for growing trachoma viruses became available (*4*), it was soon applied to studies on inclusion blennorrhea, and a successful isolation of such a virus was reported (*5*). During the past 2 years we have been engaged in growing trachoma viruses from patients in the United States and studying the characteristics of these viruses (*6*, *7*). We included in this study ten newborn children with the clinical and microscopic diagnosis of inclusion conjunctivitis. It is the purpose of this communication to report the first isolation of an inclusion conjunctivitis virus in the United States and to confirm and extend the observations of Jones *et al.* (*5*, *8*).

The infant, a normal full-term boy, developed redness and discharge of the right eye 7 days after birth. Three days later the left eye became involved. When treatment with boric acid drops had no effect, the child was examined (on the 16th day of life) by one of us (D.G.V.). There was hyperemia of the upper and lower palpebral conjunctivae, and there was a moderate amount of mucopurulent discharge. The cornea was clear. Conjunctival smears showed many neutrophils and some lymphocytes and monocytes; at least 10 percent of the epithelial cells contained typical inclusions in all stages of development. There were also many scattered free elementary bodies, but no bacteria. After treatment with sulfisoxazole ointment, the boy recovered promptly.

Conjunctival scrapings obtained on the 12th day after onset were suspended in broth-saline containing 10 mg of streptomycin and 0.1 mg of polymyxin B per milliliter; the suspension was kept on ice for 2 hours, inoculated into the yolk sacs of 7-day embryonated eggs, and incubated at 35°C. Blind passage of yolk-sac suspensions was carried out at 7-day intervals, as described elsewhere (*7*). In the fifth passage some eggs died, and yolk-sac smears revealed many free elementary bodies with Giemsa or Macchiavello's stains. In the ninth passage the virus was well established with an egg LD_{50} of $10^{-6.7}$. On two occasions the virus was reisolated from stored frozen yolk-sac material of first and third passage. Six and seven passages, respectively, were necessary before smears from eggs that had died revealed abundant visible virus. In morphology, infectivity for eggs, behavior toward antibiotics, and serologic reaction with antipsittacosis serum, the virus appeared to be indistinguishable from trachoma viruses isolated in this laboratory (*6*, *7*).

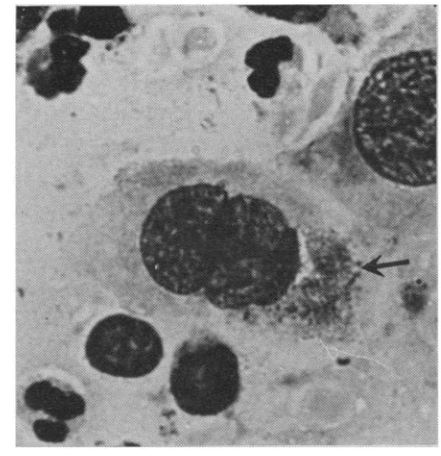

Fig. 1. Conjunctival smear from *Cynomolgus* monkey on the 8th day after infection of the eye with virus. The arrow points to an elementary-body inclusion in an epithelial cell (Giemsa stain).

Two *Cynomolgus* monkeys and one baboon were infected by instilling 0.2 ml of a 10^{-1} dilution of ninth-passage yolk-sac suspension into each eye. On the 5th day these animals developed intense conjunctival hyperemia, resulting in bleeding upon light touch. There was a mucopurulent exudate consisting largely of neutrophils and lymphocytes, and some epithelial cells contained typical inclusions (Fig. 1) indistinguishable from those seen in the newborn infant. Later the monkeys developed conjunctival infiltration and follicles which persisted for over 3 weeks. On the 12th day of infection, virus was reisolated from a conjunctival scraping of a *Cynomolgus* monkey by egg inoculation. An attempt to adapt