With this in mind, other reducing agents were used. Both sodium thiosulphate and ascorbic acid (Table 1) protected mice against death by ozone poisoning. These compounds may react with ozone before it has a chance to come into contact with lung tissue, or afford protection by decreasing capillary permeability in the lungs, and by sustaining the alveolar capillary cells during conditions of anoxia. Sodium nitrite at 2·5 mgm. per mouse (Table 1) did not protect mice but made ozone more toxic. At a level of 1·75 mgm. per mouse there was no effect on mortality due to ozone. The apparent increase in death-rate of mice injected with 2·5 mgm. of sodium nitrite can be explained by the induction of methæmoglobinæmia[7] by this compound. A sharp reduction in available hæmoglobin would handicap an animal severely injured by ozone and would hasten death due to asphyxiation.

Glutathione and *p*-aminopropiophenone[9], which have had some success in protection against death due to radiation, were not successful in protecting against death induced by ozone poisoning. Gerschmann and colleagues[10] pointed out the similarities between oxygen poisoning and injury due to radiation and found that glutathione protected against oxygen poisoning. It appears that compounds which protect against death by radiation and oxygen poisoning are not effective against ozone poisoning. The difference in protection is due to the fact that damage in ozone poisoning appears to be limited to the respiratory tract only, whereas in radiation and oxygen poisoning the whole body is involved.

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[1] Mittler, S., King, M., and Burkhardt, B., *Arch. Indust. Health*, **15**, 191 (1957).
[2] Mittler, S., Hedrick, D., King, M., and Gaynor, A., *Indust. Med. Surgery*, **25**, 301 (1956).
[3] Balagot, R. C., Reyes, R. M., Sadove, M. S., *Amer. Med. Assoc.*, **163**, 630 (1957).
[4] Sarnoff, S. J., and Sarnoff, L. C., *Circulation*, **6**, 51 (1952).
[5] Avaido, jun., D. M., and Schmidt, C. F., *Physiol. Rev.*, **35**, 247 (1955).
[6] Cole, C. J., Bond, V. P., and Fishler, M. C., *Science*, **115**, 644 (1950).
[7] Rao, P. V., *Madras Med. J.*, **3**, 1 (1953).
[8] Chapman, W. H., and Cronkite, E. P., *Proc. Soc. Exp. Biol. and Med.*, **75**, 318 (1950).
[9] Storer, J. B., and Coon, J. M., *Proc. Soc. Exp. Biol. and Med.*, **74**, 202 (1950).
[10] Gerschmann, R., Gilber, D. C., Nye, S. W., Dwyer, P., and Fenn, W. O., *Science*, **119**, 623 (1954).

Isolation of β-Sitosterol-D-Glucoside from Groundnut Phospholipids

Naturally occurring sterol glycosides (sterolins) were first characterized in 1913 by Power and Salway[1], since which time these compounds have been isolated from a variety of plant materials[2,3].

In the course of experiments on the lipids of groundnuts, a sterol glycoside has been isolated from a crude phospholipid fraction and has been identified as β-sitosterol-D-glucoside.

The initial phospholipid was produced on an industrial scale; in order to remove small amounts of triglyceride the material was washed several times with three times its weight of acetone, dissolved in ether and reprecipitated with acetone. The precipitate was filtered off on a Buchner funnel and final traces of solvent then removed *in vacuo*.

129 gm. of this material was ground with about 300 gm. of anhydrous sodium sulphate and the mixture extracted with acetone in a large Soxhlet extractor for 22 hr. The acetone extract on cooling gave a yellow waxy solid which after washing with ether yielded a white powder, insoluble in most of the usual solvents but soluble in pyridine. Further quantities of this material were obtained by concentrating the acetone extract, and in all 2·3 gm. (1·8 per cent of the crude phospholipid) was obtained.

The product was recrystallized from pyridine–ethanol as a loose white solid giving positive Liebermann–Burchard and Molisch tests, m.p. 290–292° (dec.) and $[\alpha]_D^{20} - 45{\cdot}7$. These values correspond with those reported by other workers[4,5] for β-sitosterol-D-glucoside, and the identity of the solid was confirmed by the melting points and specific rotations of the acetyl and benzoyl derivatives, and by identification of the sterol and sugar components following hydrolysis of the glycoside by the method of Thornton and co-workers[6]. Chromatographic analysis showed glucose to be the only carbohydrate component in the water-soluble portion of the hydrolysis products, and the presence of glucose was confirmed by osazone formation. Recrystallization of the ether-soluble hydrolysis products yielded a sterol, m.p. 135–136°, $[\alpha]_D^{20} - 37{\cdot}17$, in 75 per cent yield. The acetate (m.p. 126° $[\alpha]_D^{20} - 43{\cdot}01$) and benzoate (m.p. 142–143°, $[\alpha]_D^{20} - 15{\cdot}1$) correspond to β-sitosterol derivatives.

In further experiments the crude phospholipid was fractionated with alcohol; the alcohol-insoluble material corresponded to 75 per cent of the initial phospholipid and contained 87·5 per cent of the total sterol glycoside.

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[1] Power, F. B., and Salway, A. H., *Proc. Chem. Soc.*, **29**, 63 (1913); *J. Chem. Soc.*, **103**, 399 (1913).
[2] Jantzen, E., and Gohdes, W., *Biochem. Z.*, **272**, 167 (1934).
[3] Matlack, M. B., *J. Amer. Pharm. Assoc.*, **18**, 24 (1929).
[4] Swift, L. J., *J. Amer. Chem. Soc.*, **74**, 1099 (1952).
[5] Ma, R. M., and Schaffer, P. S., *Arch. Biochem. Biophys.*, **4**, 419 (1953).
[6] Thornton, M. H., Kraybill, H. R., and Mitchell, J. H., *J. Amer. Chem. Soc.*, **62**, 2006 (1940).

Site of Catabolism of Serum Albumin

The relative importance of the liver, kidneys and intestines for the catabolism of serum albumin has been examined in Swiss albino mice, 5–6 weeks of age. Mouse serum albumin and human serum albumin were labelled with iodine-131 as described elsewhere[1] and given to the mice intravenously, about 0·5 μc. or 5–15 μgm. iodoalbumin per mouse. The biological half-lives of the radioiodinated albumins were determined by one or more of three methods: (1) the living whole mouse was counted at intervals in a crystal scintillator; (2) pooled serum was obtained by exsanguination of two or three mice at a time