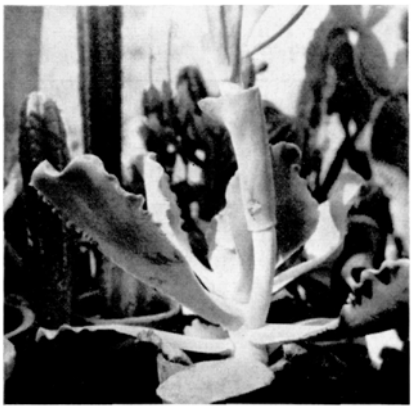
A Rare Type of Ascidium

Ascidia (scyphia) are of very rare occurrence in succulent plants. WOLTHUYS[1] reviews the literature and records his own observations. He quotes slightly more than a dozen examples recorded in almost a hundred years (the first by MORREN in 1852 and the last by himself in 1948). Seven of his examples are confined to the Crassulaceae whilst the remainder were noted in Agavacea and Liliaceae. The latter are recorded in HARRIS' *Missouri Botanical Garden Bulletin of 1906*. It is thus worth noting an ascidium on *Echeveria gibbosa*, var. *crispa*, Hort., grown in a private collection of succulent plants in Barry, Glamorganshire, in 1963. The connation has, once more, appeared in a member of the Crassulaceae. The ascidium, as shown in the photograph, is of the diphyllous type, goblet in form and open at the top as described by WOLTHUYS for other ascidia in the family. Here, however, the similarities end. The diphyllous ascidia described by WOLTHUYS terminated the shoot, the normal growing points aborted and thus further growth was impossible from within the goblet. Nearby dormant buds gave rise to side branches but nothing ever grew within the ascidium. The connation now under consideration, as can be clearly seen in the photograph, is borne on a side shoot which continues to grow from within the ascidium. Thus the growing points within the ascidium are not aborted. The shoot continues to grow from within the ascidium to end in a floral head. The flowers in this head were self-pollinated, and the seeds planted in 1964. Up to date, all the seedlings obtained are normal in every respect.



Résumé. Un type d'ascidie peu commun et jusqu'ici non enregistré dans les plantes charnues est décrit. Le point de croissance à l'intérieur du gobelet n'avorta pas, de sorte que le rejeton continua à croître à partir de l'intérieur de l'ascidie pour aboutir à une tête florale.

J. R. EVANS

Department of Chemistry and Biology, Welsh College of Advanced Technology, Cardiff (Wales, Great Britain), March 15, 1965.

[1] J. J. VERBEEK WOLTHUYS, *The Enigma of the Origin of Monstrosity and Cristation in Succulent Plants* (1948).

Acid Phosphatases and Hemoglobin in Normal and G-6-PD Deficient Erythrocytes Incubated with Acetylphenylhydrazine under Various Conditions

One of the features of the G-6-PD[1] deficient red cells is a low GSH content; moreover, when these cells are treated in vitro or in vivo with particular substances (primaquine, acetylphenylhydrazine, etc.) a further drastic decrease, indeed almost the disappearance, of the GSH occurs. This is followed in vivo by a hemolytic crisis.

The connection, if any, between the hemolytic crisis and the GSH disappearance has not yet been elucidated. The GSH is supposed to protect the cells mainly through a specific protection of the sulphydrilic groups of their proteins. The GSH decrease could then result in the inactivation of a number of enzymes and in some modification of other proteins essential for the erythrocytes.

In fact, mixed disulphides of glutathione with hemoglobin appear when GSH is oxidized to GSSG; the resulting pigment (which shows a faster anodic mobility than the normal Hb at pH 8.6) is especially susceptible to oxidation to methemoglobin followed by denaturation and precipitation[2,3].

On the other hand, a decrease of activities in a number of enzymes, such as hexokinase[4-6], pyrophosphatase[7,8], glyoxalase[9], catalase[10], and acid phosphatases[11], has been observed in erythrocytes obtained from G-6-PD deficient individuals during the hemolytic crisis induced in vivo by various drugs (primaquine) or foodstuff (fava

[1] The following abbreviations have been used: glucose-6-phosphate dehydrogenase, G-6-PD; reduced glutathione, GSH; oxidized glutathione, GSSG; hemoglobin, Hb; acetylphenylhydrazine, APH.

[2] D. W. ALLEN and J. H. JANDL, J. clin. Invest. *40*, 454 (1961).

[3] J. H. JANDL, L. K. ENGLE, and D. W. ALLEN, J. clin. Invest. *39*, 1918 (1960).

[4] G. J. BREWER et al., report in a recent paper[5] that the hexokinase activity did not appear to be reduced during the primaquine-induced hemolytic crisis in the G-6-PD deficient individuals.

[5] G. J. BREWER, R. D. POWELL, S. H. SWANSON, and A. S. ALVING, J. lab. clin. Med. *64*, 601 (1964).

[6] N. S. KOSOWER, G. A. VANERHAFF, and I. M. LONDON, Nature *201*, 684 (1964).

[7] P. BRUNETTI, F. GRIGNANI, and G. ERNISLI, Acta haemat. *27*, 146 (1962).

[8] P. BRUNETTI, F. GRIGNANI, and G. ERNISLI, Acta haemat. *27*, 246 (1962).

[9] G. J. BREWER, R. D. POWELL, A. R. TARLOV, and A. S. ALVING, J. lab. clin. Med. *63*, 106 (1964).

[10] A. R. TARLOV and R. W. KELLERMEYER, Fed. Proc. *18*, 156 (1959).

[11] F. A. OSKI, N. T. SHAHIDI, and L. K. DIAMOND, Science *139*, 409 (1963).