summers there may be no noticeable death-rate. Parasites, overcrowding and pollution are important possible factors in the mortality observed, but the small proportion of parasitised individuals found, and the occurrence of recently dead cockles in sparsely populated areas, indicate that these two factors are not of sufficient importance to account for the high percentage of deaths observed, while pollution of a chemical kind can probably be eliminated on general biological grounds, for example, abundance of young shrimps and gobies.

The object in writing this preliminary notice of these observations is to direct attention to the need for *ad hoc* researches on the problem over a number of years in the interest of public health.

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¹ J. Johnstone. Liverpool Marine Biological Committee. Memoir No. 2, Liverpool, 1899.

² F. S. Wright. Fishery Investigations, 2, 9, No. 5, Min. Agric. and Fisheries, London, 1926 (1927).

A Scottish Occurrence of *Craspedacusta sowerbii*, Lankester

MAY I record what is, I believe, the first Scottish occurrence of the trachyline *Craspedacusta sowerbii*, which was proved by Payne¹ and confirmed later by Flower and Boulenger² to be synonymous with *Microhydra ryderi*, Potts.

The medusoid phase, previously discovered in England, France, Germany, in the *Victoria regia* tanks of the botanic gardens, and also in China, the United States and the Panama Canal zone, appeared suddenly in a tropical aquarium kept at my home in Liberton, Edinburgh.

The production of medusæ was first observed about 8 a.m. on the morning of May 20, 1933, and they continued to appear until about 2 p.m. Altogether 70-80 medusæ were counted, but in spite of careful search no trace of the polyp form was discovered. The temperature of the tank was constant (75° F.). The larger plants were *Vallisneria spiralis* and *Elodea canadensis*. The whole plant and animal stock originally came from the Derham Fish Farm, Herts, but on inquiry there Mr. Derham stated that he had never seen the medusæ in his tanks. A certain amount of the water in my aquarium, however, came from the Braids Pond, south Edinburgh, while from time to time small quantities of water and scraps of vegetation from bog pools on Balerno Moor, near Edinburgh, have been introduced while feeding the fish on *Daphnia* and *Cyclops* obtained from these pools.

It was a curious fact that the medusæ should not have been noted before during the four years I have kept the tank undisturbed, but it may be significant that on the morning on which the medusæ appeared, my pair of paradise fish laid eggs and there was an enormous increase in the numbers of a certain species of bdelloid rotifer inhabiting the tank and also of *Carchesium polytypum* in the tank.

The development of the very early stages of the medusæ would appear to have been more rapid than that observed by Boulenger (loc. cit.) and Potts³, since those I examined immediately after their appearance already had sixteen well-developed

tentacles, the eight adradial being slightly shorter than the others. During the fortnight I was able to keep the medusæ alive, however, there was no further sign of development of any sort.

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¹ *J. Morph.*, 38, 387; 1924.

² *Proc. Zool. Soc. Lond.*, 1905; 1928.

³ *Quart. J. Micro. Sci.*, 50, 623; 1906.

Influence of Iodoacetic Acid on the Blood Sugar Level

I HAVE previously reported¹ that iodoacetic acid causes a hyperglycæmia in rabbits and that it reverses the effects of insulin by causing the blood-sugar to rise after hypoglycæmia. In an endeavour to find out whether this was due to mobilisation of liver glycogen or to an inability of the tissues to utilise blood-sugar, a series of perfusion experiments has been carried out. Rabbits' hind legs were perfused with blood and the rate of fall of the blood-sugar was followed. In certain of the experiments sodium iodoacetate was present in the whole preparation in a final concentration of from 1 in 2,000 to 1 in 6,000, calculated as free acid. The utilisation of sugar was found to be impaired to a considerable extent, both in the presence, and, more so, in the absence of insulin, as these figures show. The sugar used is calculated and stated as mgm. per kgm. of legs per hour.

Normal perfusion (average of 3)	96
Perfusion with iodoacetate (average of 5)	19
Perfusion with insulin (average of 4)	164
Perfusion with insulin and iodoacetate (average of 5)	84

If a great deal of evidence did not exist showing that lactic acid in the blood stream is probably not a precursor of muscle glycogen, these experiments would not preclude the possibility that glucose was degraded to lactic acid before being utilised by muscle tissue. If this is not so, then it appears that iodoacetate can inhibit the reaction glucose → glycogen as well as that of glycogen → lactic acid.

The expenses of this work were defrayed by grants from the Royal Society and the Medical Research Council.

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¹ Irving, *J. Physiol.*, 75, 4P; 1932.

Vitamin C in Blood and Urine?

HARRIS has showed that the reducing capacity of animal tissues titrated against 2·6 dichlorophenolindophenol (as indicated by Tillmans and Szent-Györgyi) in acid solution, coincides with the biological activity.

Harris believes, however, that the reducing substance of tumour tissue is not, or not entirely, due to vitamin C (perhaps in this case a toxic effect of the tumour tissue plays a rôle). This shows that the test with the indicator must be used with due understanding. Euler showed that urine also contains a reducing substance. We have made the following experiments with blood and urine.