Wood *et al.*[1] have previously reported a diatom cell with the characters of *Coscinodiscus lineatus* and *C. radiatus* in opposite valves, and inter-specific variants have been seen by other workers (N. I. Hendey *in litt.*), but such variations could be explained as inter-specific crosses. Several Antarctic *Actinocyclus* and *Schimperiella* species have two dissimilar valves, but this character is not characteristic of either *Coscinodiscus* or *Asteromphalus*. It is perhaps fortunate that the aberrant cells were observed as further division could have resulted in apparently normal *Coscinodiscus* and *Asteromphalus* cells, in which their derivation would not have been apparent. The fact that one cell of the pair was a normal *Coscinodiscus* makes an explanation of the phenomenon difficult.

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[1] Wood, E. J. F., Crosby, L. H., and Cassie, U. V., *Trans. Roy. Soc. N.Z.*, **86**, (1959).

A Rare Hydromedusa

In June of this year a small medusa appeared in a salt-water aquarium tank at Aberdour, Fife. Stones placed in this tank had been gathered on the shore and dredged from below low-water mark at Aberdour. These stones had unidentified hydroids attached. Other medusæ appeared during June and July.

These medusæ have been identified as *Gonionemus*, probably *murbachi*, which Dr. F. S. Russell suggests may possibly be the same as *vertens*.

It is interesting to note that *Gonionemus* has only been recorded in Great Britain from aquarium tanks, once from the Laboratory at Cullercoats, once from the Laboratory at Port Erin and now from a private aquarium at Aberdour.

Specimens of this medusa have been sent to the British Museum (Natural History) and registered as No. 1959.9.2.1–9, and others are held by me.

Efforts are now being made to identify the hydroid.

Another medusa of interest appeared off Aberdour in June this year, when several specimens of *Eutonina indicans* were taken in a hand net from the sea at low tide.

I am indebted to Dr. F. S. Russell for identifying the *Gonionemus*.

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Aug. 31.

PATHOLOGY

Inhibition of Rous I Sarcoma Metastases by a Copper-Catalyst Blocking Agent

A connexion has previously been demonstrated between the activation of copper-enzyme catalysis and the production of tanned lipoprotein surface membranes[1]. These same membranes, under various experimental conditions, were found to be non-adhesive[2], and since tumour cell surfaces display this property[3], it suggested the possibility that cellular adhesion may depend on the integrity of a proportion of the protein moiety of the lipoprotein complex of the surface membrane.

When a tanned (sclerorized) membrane is treated with Mallory stain, it takes up the orange *G* dye to the exclusion of the other competitive aniline blue dye. Naturally occurring untanned membranes, or tanned membranes pretreated with chlorodioxyacetic, an agent which breaks down aromatic bonds in tanned structures[4], take up the aniline blue of the same stain. It is well known that the scleroprotein group, which includes keratin, myosin, and fibrinogen besides tanned protein, has an affinity for the orange *G* of Mallory stain, whereas the collagen and secretory group of proteins react with the aniline blue.

The Mallory staining of cells revealed that dried and methanol fixed smears of Rous 1 solid sarcoma and ascites cells, as well as red blood cells, show an affinity for orange *G*. These same cells after momentary pretreatment with the above agent, very much diluted, took up the aniline blue. The counterparts of the Rous cells, connective tissue cells and white blood cells (monocytes) respectively, show an affinity for the aniline blue. This differential staining has also been tried on hamster kidney tumour cells and their normal counterparts. The staining time for these cells, however, seems to be very critical, and variations in the results are liable to occur. The tumour cells, however, show a readiness to take up the orange *G*, the normal cells aniline blue. The surfaces of the cells described stained intensely. These results recall the differentiation of tumour cells from normal ones by their cytoplasm, unlike that of normal cells, failing to take up fluorescein-globulin stains, the reason given for this being that the protein that interacts with the globulin fraction is absent in tumour cells[5]. In the case of Mallory staining, it would seem that a protein of the scleroprotein group may be responsible for the tumour cells taking up the orange *G* dye. There remained also the possibility of the protein being tanned in some way, and that such a protein might comprise a proportion of the tumour cell surface.

Accordingly, the amelanotic Rous 1 sarcoma tissue was tested for the presence of tyrosinase, the tanning enzyme[6], and the result was positive. It may be recalled that melanotic melanomas provide a rich source of the enzyme[7], and reference has also been made to its presence in amelanotic tumour tissues[8]. Incubation of the same tissue with dihydroxyphenylalanine as the substrate resulted in melanin being intensely developed. The tumour tissue also mediated the conversion of the diphenol to melanin more readily than equivalent wet weights of normal tissues such as connective tissue from a growing cockscomb, or those of the various parts of chick embryos. Rous 1 and MH_2 tumour tissue induced by virus infection to grow on the chorio-allantoic membrane of the chick embryo also displayed an ability to mediate the conversion of the diphenol to melanin more readily than the normal membrane tissue from which the tumours were derived. These tests involved immersing the material in a saline solution of the diphenol. The process was completely blocked by phenylthiourea at $10^{-3}M$. This copper-catalyst blocking agent inhibits the activity of tyrosinase[1] without affecting cytochrome oxidase[9], which is apparently capable of catalysing the oxidation of a diphenol to melanin[7].

In the light of these observations it seemed possible that the character of tumour cells might be so coupled to copper-enzyme catalysis, that their metabolism might be more sensitive to its blockage by a chemical of specific action than that of normal cells. On this view, experiments were designed and carried out in collaboration with Dr. J. G. Carr and Mr. H. Drayton, of the British Empire Cancer Campaign Unit at the Poultry Research Centre, Edinburgh, to test the effect