their sucker. If the circulation is then restarted, the lampreys recover completely within two or three hours.

My tanks are of heavily galvanized iron and rest on a stone floor in a basement room which is kept cool by a through draught of air. Last winter the outside windows were closed in error one week-end. This caused the temperature of the water to rise from 13° C. to 22° C., and despite the fact that the water was still circulating all the lampreys had died.

The high sides of the tank cut out direct light and keep the lampreys from jumping out when they are being netted.

Details of the apparatus and experiments will be published elsewhere.

Note added in proof. The pump mentioned above has since been replaced by a ½ h.p. A.C. centrifugal pump which needs little servicing.

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Feeding-Rates of Sponges, Lamellibranchs and Ascidians

Many water-living animals obtain their food from suspended material, micro-organisms or fine dispersed detritus occurring in the surrounding water. Sponges and ascidians, for example, are typical filtering animals. Most lamellibranchs belong to the same group. But filtering forms may also be found among gastropods, crustaceans, insects, polychætes, vertebrates, etc. Apart from lamellibranchs, little information is available regarding the rate and efficiency with which particles are retained from water passing the filtering organs. Bidder[1] calculated the water propulsion through *Leucandra aspersa* (a sponge), whereas investigations on the rate of feeding of ascidians seems to be lacking. During a stay at the Plymouth Laboratory, I was offered an opportunity of determining the feeding-rates of species of sponges and ascidians. The feeding-rates have been calculated from the rate of disappearance of particles from the surrounding water[2]. Suspensions of colloidal graphite were used, namely, 'Prodag' grade C and 'Aquadag' grade S, manufactured by E. G. Acheson, Ltd., London; the former has an average particle-size of 4–5 μ, whereas the latter has an average particle-size of 2 μ. The concentrations of the graphite suspensions were determined photometrically by means of a portable Eel photometer.

The results of the experiments are given in the accompanying table. The feeding-rates are expressed as per mgm. of amino-nitrogen of the experimental animals instead of the normally used weight and length or similar measurement, which provide an unsatisfactory basis for comparisons between animals. In the table are also included results obtained on *Mytilus edulis*, which will be published in greater detail elsewhere[3]. It is seen that—when expressed in relation to the amino-nitrogen content, which may be regarded as a measure of the amount of protoplasm in the animals—the feeding-rates of the species investigated are all of the same order of magnitude, *Halichondria* and *Mytilus* showing somewhat lower values. However, the experiments on *Halichondria* were carried out on a fragment weighing about 50 gm., whereas the calcareous sponges used were all smaller intact specimens.

Species	Number of animals in each experiment	Length (cm.)	Average content of amino-nitrogen (mgm.)	Feeding-rate (mgm. per mgm. amino-nitrogen)
Ascidians				
Molgula sp.	20	1–2	1·8	138, 124, 118, 142
Ciona intestinalis (L.)	2	6–7	6	125, 92
,, ,, ,,	3	6–7	5·3	125
Sponges				
Grantia compressa (Fabricius)	1	7	8·5	135
,, ,,	9	3–7	3·3	180
Sycon coronatum (Ellis and Solander)	9	4–6	2	200, 145, 170
Sycon + *Grantia*				
Halichondria panicea (Pallas)				65
Lamellibranchs				
Mytilus edulis (L.)		*c.* 3	25	36
,, ,, ,,		*c.* 1·5	2	80

The feeding-rates of sponges and ascidians were found to be independent of the particle-size, which varied from about 2 μ and 4·5 μ respectively and upwards. The upper limit of the particle-size in the suspensions was not known, as the graphite did not form fully dispersed suspensions in sea water. A certain amount of aggregated particles was always observed when the suspensions were examined under the microscope.

As regards the ascidians, the independence of feeding-rate and particle-size is in good agreement with the view held by MacGinitie[4] concerning the feeding mechanism of ascidians. He observed that ascidians when feeding are covering the gill basket with a continuous layer of mucus which retains all particulate matter in the water transported through the gills.

My thanks are due to the director, Wing-Comdr. F. S. Russell, for placing the working facilities of the Plymouth Laboratory at my disposal, and for his great interest in my work. The investigations were made possible through a grant from the Rask-Ørsted Foundation.

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[1] *Quart. J. Mic. Sci.*, **67**, 293 (1923).
[2] Jørgensen, Barker, *Acta physiol. Scand.*, **5**, 297 (1943).
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Varagu Poisoning

Varagu (Tamil), *kodo*, *kodaka* (Hindustani), *Paspalum scrobiculatum* (Linn.), is a millet largely used by the working and poorer classes of people in all parts of India as a staple article of food. More than 300,000 tons of this millet is produced in the Madras Presidency alone. In 1946, when rice was rationed and millet eaters were compulsorily asked to take a portion of their requirements in rice and millets in the form of *varagu*, complaints were received that consumers in several parts of the Presidency developed symptoms of food poisoning as a result of eating *varagu* in the cooked as well as the raw state.

The symptoms observed within twenty minutes of taking the food were tremors, giddiness, perspiration, inability to speak or swallow. There were no fatalities, and the symptoms disappeared after twenty-four hours, though the persons affected had to be taken to hospital for treatment.

It was difficult to feed monkeys, as they refused to take the food. Two ounces of the millet produced the same symptoms on dogs; tremors and paralysis were characteristic effects, and the dogs died in twenty-four hours. The Stas Otto test was negative, showing the absence of alkaloids. The fat was extracted with petroleum ether (60–95° C.), or chloroform, or ether, and it was found that 1·5 gm. of fat corresponding to 50 gm. of the millet were fatal to dogs. The fat had the following figures (average):

Melting point	42° C.
Refractive index (60° C.)	1·4650
Iodine value	93·6
Saponification value	170·7

The defatted residue was found to be non-toxic. The poison in the fat seems to be decomposed by dilute acid and alkali, as after acid and alkali treatment the treated fat is no longer poisonous. The liquid, decanted after shaking the millet with petroleum ether, develops a characteristic red colour when shaken with concentrated sulphuric acid. But as the fat in the millet after treatment with dilute acid—when it is no longer poisonous—still gives the colour test, it is inferred that the colour observed is not due to the poison in the fat but to a decomposition product of it. The poison seems to be neither an alkaloid nor a glucoside, as it is not extracted with acid, water or 90 per cent alcohol. It seems to be adsorbed chromatographically in silica column. Further work is in progress.

The fat obtained from non-poisonous varieties of *ragi* is quite harmless, and does not give the sulphuric acid test described above.

The most surprising observation is that the fat derived from the poisonous variety develops symptoms of poisoning in dogs and monkeys when injected intramuscularly, 1 gm. of the fat being fatal. Crows seem to be extremely susceptible to the poison in the fat, either when ingested orally or injected intramuscularly. Within ten minutes of oral ingestion, the crow puts down its beak and vomits; even after all the contents have been vomited the crow puts down its beak in an effort to vomit. It loses the mobility of its eyes and soon its power of using its legs, while the wings are spread out and can no longer be brought together. The crow dies in twenty-four hours. The effects are slow when the fat is given by injection; but the collapse is more pronounced and the symptom of vomiting, even when there is nothing to vomit, is a characteristic of the poisoning in the crow.

These observations do not appear to have been recorded so far in the literature. They give a means of distinguishing the poisonous from the non-poisonous variety, and it should lead to the reclamation of an important article of food which, of late, has come into disrepute owing to the ill-effects attending its consumption.

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Rh Phenotypes and Fisher's *CDE* Notation

Since the introduction of Fisher's theory and notation[1] of the *Rh* blood-group system, the tendency has been to express phenotypes in terms of the probable genotype. This method is most convenient for European populations, where every common phenotype consists very largely of a single genotype; but its extension to non-European populations usually involves serious ambiguities. In this and other connexions, there is an undoubted need for an unambiguous and generally understood phenotype notation expressed in Fisher's symbols, which would be available to supplement current practices. Several attempts have been made to express phenotypes with the use of these symbols, probably the earliest being that of Bushby[2]. The present proposals closely resemble those of Bushby; but allowance is here made for the fact that the six typing sera (anti-*C*, *D*, *E*, *c*, *d*, *e*) are not usually all available as Bushby tacitly assumes. No originality is claimed for these proposals; it is probable that other workers have thought of and used them independently; and, in fact, when recently I was discussing an *Rh* problem with Dr. M. Bessis of Paris, we found that we had both for some time been using this system in working out *Rh* problems.

The phenotype formula is derived as follows: considering first the *C* locus of Fisher, if a specimen of blood has been tested with anti-*C* alone and the result is positive, a single *C* is written. If the result is negative, *cc* is written, whether or not the blood has been tested with anti-*c*, since a negative result with anti-*C* indicates the presence of two *c* genes. If the blood has been tested with anti-*C* and anti-*c* and both give positive results, *Cc* is written. If the result with anti-*c* is negative, *CC* is written, and this can be done even in the absence of a test with anti-*C*. The *D* and *E* systems are treated similarly. But if both the antisera required for one particular system (for example, *Ee*) are unavailable, the statement of the results (for example, *Ccdd*) makes this quite clear. To take a concrete example, let it be supposed that blood of the genotype *CDe/cde* is being tested with the commonest four antisera, namely, anti-*C*, anti-*c*, anti-*D* and anti-*E*, the results will, in that order, be + + + − and the phenotype will be written *CcDee*. The only available information which is missing from this expression of the results is as to whether the blood has been tested with anti-*e*; but the negative result with anti-*E*, in fact, makes such knowledge irrelevant. The absence of a second *D* symbol (*D* or *d*) shows that no test has been done with anti-*d*.

The above suggestions only relate to the common antigens *C*, *D*, *E*, *c*, *d*, and *e*, in respect of which all the suggested conventions appear self-evident. In order to deal with the rarer alleles such as C^w, it would be necessary deliberately to adopt certain further conventions by agreement between the workers concerned.

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