data he determines the critical points to occur most frequently in the neighborhood of 4.5°, 9°, 15°, 20°, 25°, 27° and 30° C. As he points out, the agreement of these figures with those of Setchell can hardly be accidental. It may be assumed, therefore, that the temperatures for spawning of the lamellibranchs here listed fall where they do by reason of similar fundamental processes which control vital phenomena in the plants and animals considered by Setchell and by Crozier.

II

Although spawning has been the most extensively studied in relation to temperature of all vital processes in bivalve molluscs, observations of ciliary activity made thus far on lamellibranchs reveal a similar distribution of critical temperatures. Gray[12] showed in *Mytilus edulis* a progressive increase in speed of the cilia, with normal amplitude of beat, from 0° to 32.5° C. One may take 0° C. therefore as the critical temperature for the initiation of ciliary activity in this form.

In *Ostrea virginica* I have shown (Nelson, T. C., a, c, d.) that the critical temperature for ciliary activity and for shell opening of animals taken during cold weather is close to 5.6° C. Below this temperature ciliary activity is practically in abeyance. Galtsoff[13] finds that the cilia of the oyster come to a standstill at 5° C. This critical temperature of 5° for ciliary activity is accompanied by a spawning temperature of 20° C. Roughley,[14] working with the Australian oyster, *O. cucullata,* has shown that this form fails to open in water of a temperature lower than 10° C., whereas above this point the molluscs are active. This observer also notes that pulsations of the heart become slow and weak at 10° C. while above this temperature the beats are vigorous and more rapid. The spawning temperature for this species is not known, but from the restricted distribution of *O. cucullata* and from the observation of Roughley that in some seasons it does not spawn at all in the northern part of its range, it is probable that its spawning temperature will be found to be either 20° or 25° C.

Of interest in this connection is the observation of Takatsuki[15] that the pulsation of the heart of *Ostraea circumpicta* is abolished at 0° C. but begins at 5°–7° C. It is hqped that future work at the Asamushi Station will establish the critical temperatures for spawning and for ciliary activity in this little known species of oyster.

[12] *Proc. Roy. Soc.* 95, 6, 1923.

[13] SCIENCE, LXIII, 233, 1926.

[14] *Proc. Linn. Soc. N. S. W.,* LI, 446, 1926.

[15] *Scientific Report,* Tokhoku University, 4th Ser., II, 3, 1927.

The bearing of critical temperatures upon the distribution of these lamellibranchs is of prime importance, but can not be discussed here further than to mention the following facts. *Ostrea virginica,* the most valuable mollusc in the world, is barred from most of the otherwise favorable coast lines of the earth since the waters there rarely attain a temperature of 20° C. for a sufficient period to permit spawning. The inferior species *O. lurida* and *O. edulis* may thrive there since much of the coastline of the northern hemisphere rises to 15° C., or slightly above, for the time necessary to permit these species to spawn. *Teredo navalis* with the same spawning temperature has been carried into most of the ports of the world. *Mytilus edulis* with its still lower spawning temperature is the most widely distributed marine lamellibranch in the northern hemisphere. *Venus mercenaria,* the hard clam, on the contrary, is found only in a relatively few sheltered areas where subtropical spawning temperatures of 25° C. are attained at some time during the summer.

Many more observations of other species of pelecypods in widely different environments are needed to determine whether the critical temperatures shown above are characteristic for this group of animals as a whole. This preliminary paper is presented with the suggestion that study of the spawning temperatures of groups of aquatic species in the light of our newer knowledge of critical temperatures will prove a valuable method of attack upon problems concerning the distribution and behavior of such organisms.

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STARVATION KETOSIS OF THE PRIMATES

IN the course of experimental work on monkeys afflicted with "cage paralyses" the writer found that they excrete relatively large quantities of acetone bodies when starved, and, by analyzing the data in accordance with the ketogenic-antiketogenic conceptions of Shaffer,[1] the excretion (with the exception of the lemur) could be considered comparable in every way to that of man.

The following animals were used:

Black ape, *Cynopithecus niger* (Desmarest) (Celebes), male and female;

Bonnet Macaque, *Pithecus sinicus* (Linn.) (India), male and female;

Brown capuchin, *Cebus capucinus* (Linn.) (South America), male.

A mandrill, Papio sphinx (Linn.) (West Africa), male, on starving was found to excrete only traces of

[1] Shaffer, P. A., The Harvey Lectures (Series XVIII), Lippincott, 1924.

acetone bodies. The nitrogen excretion during starvation, however, was extraordinarily high. The animal weighed 5 Kg. and normally excreted 1.3–2 g. of nitrogen per day. After the withdrawal of food the nitrogen excretion rose to 4.5–5.3 grams per 24 hours on the three days of starvation.

A lemur *Lemur macaco* (Linn.) during two starvation periods of four days each excreted only faint traces of acetone bodies and the nitrogen excretion increased only slightly.

The animals were placed in metabolism cages and urine collections were made (without catheterization) at frequent intervals. The animals were very quiet, and would move away only when touched or frightened; however, they readily ate food when offered. They appeared to be in good condition—not noticeably emaciated. The urine contained much acetoacetic and β-hydroxybutyric acids after 24 hours starvation, and the excretion of these acids reached its maximum after 48 hours. The ketosis was promptly abolished on feeding glucose or carbohydrate-rich foods.

It will be noted that the starvation ketosis was equally severe in both the new and old world monkeys, and since it was found in monkeys of differing genus and living in widely separated regions, this behavior is believed to hold true for the anthropoidea in general. It is particularly interesting that a marked starvation ketosis has been observed only in the case of man and the anthropoidea. Thus a survey of the literature shows that the following do not develop a marked ketosis: dairy cow, steer, goat, pig, cat, dog, rabbit, guinea pig, rat.

There appears to be a real difference in the fat metabolism of man and the monkey on the one hand and the other animals enumerated above. It can not be explained by dietary habits, since it has been found to be slight in both carnivora and herbivora; nor can one accept the theory advanced by some that the absence of a starvation ketosis is due to adaptation. According to the popular theory fat metabolism proceeds in steps by β-oxidation, resulting finally in one of the acetone bodies, probably acetoacetic acid. The difficulty lies in the disposal of the latter. It is more logical to assume that organisms in general possess the means of *completely* metabolizing fats which are natural and necessary components of the cell. Putrefactive bacteria for example readily metabolize acetoacetic acid (Neuberg). The cat[2] and the dog[3] have a high tolerance and readily dispose of intravenously administered acetoacetic acid. The high tolerance and absence of a marked starvation ketosis is perhaps due to the presence of a "ketolytic" enzyme or catalyst which enables the cell to dispose of the acetoacetic acid formed from fat, etc. Entire loss or disfunction of this cell catalyst results in a ketosis. In the course of evolutionary development of the primates the "ketolytic" cell catalyst appears to have been lost. The acetone bodies do not normally appear in man because of a peculiar adaptation in which it appears that "the fats burn in the fire of the carbohydrates." Lacking metabolizable carbohydrate (as in starvation, phlorhizin diabetes, diabetes mellitus) the primates can not burn acetone bodies because of the absence of the ketolytic ferment. Because of the ease with which the lower animals dispose of acetone bodies they do not need to burn acetone bodies "in the fire of the carbohydrates," and metabolizing carbohydrate therefore probably is not antiketogenic, as in the case of the primates, because the necessary enzymes are not present. The slight starvation ketosis observed in the case of the lower animals, therefore, is not to be considered due to a lack of metabolizable carbohydrate, but is due rather to an altered condition of the cell which results from the low carbohydrate content. Restoration of the carbohydrate content of the cell to normal allows the cell to more efficiently burn the fats and hence leads to disappearance of the slight ketosis.

It is interesting in this connection to note several instances of severe ketoses, without apparent loss of carbohydrate tolerance, observed in cows. Sjollema and Van der Zande[4] report an "acetonaemia," etiology unknown, in milch cows following parturition. Although the animals received food, glucose was absent from the urine and the blood sugar was normal. A similar condition seems to occur in cattle poisoned by white snake root *Eupatorium urticaefolium*. From the meager data at hand, it appears that the carbohydrate metabolism in this condition also is not markedly disturbed. If a further study should confirm these observations, one may hazard the guess that the ketosis is due to blocking or a disfunction of the ketolytic ferment or hormone. A ketosis, in spite of a normal carbohydrate metabolism would confirm the view advanced above that glucose in these animals is not antiketogenic.

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[3] Wilder, R. M., *J. Biol. Chem.*, *31*, 59 (1917); Friedemann, T. E., Somogyi, M., and Webb, P. K., *J. Biol. Chem.* (Proc.), *67*, 44 (1926).

[4] Sjollema, B., and van der Zande, J. E., *J. Metab. Res.*, *4*, 523 (1923).