effect (high protein). The largest response occurred with the basal diet. It appears that the maximum levels of brain temperatures achieved may not be related to the specific dynamic action of food. Indeed, the rise in brain temperature may be unrelated to the process of food intake regulation because the time at which the animals ceased vigorous feeding behavior bore no particular relationship to the peak brain temperatures achieved.

Data pertaining to the important question of which, if any, of the variables in the diets is the determining factor as far as regulating food intake is concerned are presented in Table 1. Regulation of food intake on the basis of any one of the intrinsic food factors listed would be evidenced by a constancy in that factor independent of diet composition. It is apparent that none of the observed factors is constant for all diets tested. These data plus the lack of any relationship between intracranial temperature changes and the composition of consumed food or general feeding behavior fail to offer support to the thermostatic theory.

Consideration of multiple factors would seem to be the most reasonable approach to the solution of this intriguing problem. As yet, however, no satisfactory analysis of the manner in which the multiple factors might be integrated has been achieved. Indeed, such an attempt will have to await the results of renewed efforts to identify the individual factors involved.

ALFRED J. RAMPONE
MYRON E. SHIRASU
Department of Physiology, University of Oregon Medical School, Portland

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Fluctuating Brightness of Quasi-Stellar Radio Sources

Some of the quasi-stellar radio sources have been found to vary in brightness with a period much shorter than the time needed for light to traverse the object. Thus the brightness of 3*C* 273 fluctuates with a period of 13 years, while 3*C* 48 has exhibited a 30-percent fluctuation in a single year. This has caused puzzlement. For example, J. L. Greenstein has said [*Sci. Am.* **209**, No. 6, 60 (1963)]:

> Such variations could be explained if 3*C* 48 and 3*C* 273 were merely stars, but if they are several thousand light-years in size, it is difficult to see how a general brightening could take place in a tiny fraction of the time needed for light to travel from one side of the object to the other. In other words, it seems impossible to explain a systematic variation in brightness without a pulse-transmitting signal, and this could not travel faster than light.

It is the purpose of this note to suggest a possible way out of this quandary. Clearly, if the theory of relativity is valid, no triggering influence can bring about such rapid variations of brightness over large surface regions of such an object if we require that the source of the influence lie in or near the surface. But suppose the source were in a compact central region, that it sent out roughly spherical influences, and that these influences, on reaching the outer, charged layers of the object, caused them to fluctuate in brightness. Then, if the object had approximate spherical symmetry, the induced fluctuations in brightness in its outer layers would occur roughly simultaneously over most of the surface, thus causing the object to exhibit rapid overall fluctuations in surface brightness despite its large size.

BANESH HOFFMANN
Queens College of the City University of New York

Tetrodotoxin: Mechanism of Action

From all available evidence, tetrodotoxin, the puffer fish poison, and the chemically and biologically identical tarichatoxin from a California newt (*1*) exert their effects by way of a very potent axonal blocking action. These agents can abolish propagated action potentials in desheathed frog sciatic nerve in a concentration as low as $0.003\mu M$ (about 1 μg/liter) and in this concentration range produce profound physiological derangement in the whole animal (*2*). Such a mechanism of action of these toxins was recently confirmed by Lowenstein, Terzuolo, and Washizu (*3*), who found that tetrodotoxin in concentrations of 1 to 5 μg per milliliter abolished the directly and antidromically elicited action potentials in the stretch receptor neurons of the crayfish. Their interesting observation on separating the generator potential from the spike, however, appears to have led them to some unjustifiable conclusions concerning the sites of genesis of these bioelectric phenomena. In their experiments, the spikes in both the crayfish stretch receptor and the cat Pacinian corpuscles were clearly recorded in axonal extensions of the sensory receptors. Under such conditions, the spikes could be expected to be abolished by tetrodotoxin as well as by other axonal blocking agents such as some commoner local-anesthetic agents. In the presence of such an axonal block, it would be difficult to know whether more proximal spike-producing mechanisms are also affected. Thus, it cannot be concluded unequivocally that different membrane patches in the sensory receptors are responsible for generator potentials and spikes. Definitive evidence to settle this question is still wanting in spite of their interesting observation.

C. Y. KAO
Department of Pharmacology, State University of New York, Downstate Medical Center, Brooklyn

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C. Y. Kao

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