cation would take a full lifetime to write, and the resulting program 1000 years to de-bug. There is no way of knowing in advance; we must find out by experimenting.

Dogmatic assertions of the omnipotence of computers tend to stir up a multiplicity of, often unpleasant, reactions in the reader. They do not have the supposedly compensating advantage of being true.

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Self-Stimulation Experiments

Your publication of papers by Margules and Olds [*Science* **135**, 374 (1962)] and by Hoebel and Teitelbaum [*Science* **135**, 375 (1962)] leads me to propose the following physiological explanation of the association which they describe between mechanisms for self-stimulation and for feeding in the lateral hypothalamus. It is my opinion that in a self-stimulation experiment the negative feedback loops of normal feeding mechanisms are replaced by an artificially constructed loop having a positive sign.

Under natural conditions, an activation of the lateral hypothalamus induces or facilitates feeding behavior. Included in the many possible varieties of such behavior is bar pressing—one of the responses which lateral hypothalamic activity will induce for feeding. Ordinarily such behavior induced by the lateral hypothalamus leads to ingestion of food, and this leads in turn to a number of physiological changes which inhibit further intake of food and suppress the activity of the lateral hypothalamus. But when, as in a self-stimulation experiment, the bar pressing leads not to food ingestion but to electrical stimulation of the lateral hypothalamus, then that part of the brain can only become still more active. Consequently the animal is that much more likely to press the bar again, and every further press enhances the chances of more presses. Induced to press the bar in the first place by a naturally occurring activation of its lateral hypothalamus, the animal receives for its press only a recurrent stimulation into the region which originated the bar pressing.

This distinction between a normal, negative feedback loop and an artificial, positive loop avoids the paradox mentioned by Olds [*Physiol. Rev.* **42**,

554 (1962)] in these words: "In any event it is clear that stimulation of the same lateral area has two usually dissociated effects . . . the effects of the primary drive itself . . . [and] the effect of the primary reward related to that drive. . . . Therefore the possibility that the electric stimulus constitutes a simple internal surrogate for either is unlikely" (pp. 593–94). It seems more probable that self-stimulation of the lateral hypothalamus is a surrogate for natural stimulation of the lateral hypothalamus—nothing else.

If my interpretation for the relationship between feeding and self-stimulation is correct, then one can predict that any region of the brain where self-stimulation is observed must function as a component of a similar physiological system, in which the animal can be taught to use bar pressing as a part of some normal behavioral sequence.

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While I find myself in sympathy with Brobeck's view, I find it difficult to agree fully for two reasons:

1) At the beginning of a self-stimulation experiment, bar pressing has never been previously associated with or been instrumental in feeding behavior. It is a random response like ear twitching or tail movement, and it should be kept in mind that any random response may be used to trigger the electric stimulus. The chosen response will quickly rise in frequency, gradually excluding other responses from the immediate repertory, until the chosen response predominates and occurs at a maximum possible rate. It is difficult to understand why this response should be chosen for repetition just because of its temporarily contiguous relationship to the subsequent increment in lateral hypothalamic activity. The increment should make all food-related or other possible responses more likely, but I do not find in Brobeck's explanation any reason why the response emitted just prior to artificial stimulation of the hunger drive should be marked for immediate repetition. We think of a hungry animal trying the habitual responses in an effort to get food, and if these fail, trying others. If some item of the new repertory were followed by a sudden rise in hunger or in any internal activity generator, would that response be repeated? If so, why?

2) If the size of the supra-threshold

electric field in a self-stimulation test is approximately a 1-mm sphere, it seems unlikely that the sphere in such a complex structure as the hypothalamus in a small animal like the rat is homogeneous in regard to function. One millimeter is the cross-sectional diameter of the whole medial forebrain bundle which might so far as we know mediate the whole gamut of emotional control. Thus, on anatomical considerations alone it seems that an electric stimulus here must be having more than one effect.

Two possible explanations occur to me; different from Brobeck's, but equally plausible.

1) The electric stimulus might simply activate two different mechanisms, one yielding eating behavior, the other yielding behavioral reinforcement. The mechanisms might be grouped in anatomical proximity in the lateral hypothalamus so that both could be brought under control of a common deficit-sensor (such as the hypothetical glucose receptor). In such a case, a deficit in nutrients would have two consequences, (i) generating activity directly in the eating behavior system, and (ii) lowering thresholds in a "taste" system so that stimulation by food would "taste better"; that is, it would have more power as a positive reinforcement over antecedent operant behavior patterns. If such an anatomical proximity existed, electric stimulation, acting as something of a bludgeon, would have two effects, evoking directly the eating behavior system and the positive reinforcement mechanism of the "taste" system.

2) The electric stimulus has the same effect as food in the mouth which causes eating and repetition of antecedent behavior. These views are related both to one another and to Brobeck's view, but they emphasize the distinction between drive-caused behavior and reward-caused selection of a particular behavior for repetition. In drive-caused behavior an antecedent condition heightens the activity level of the organism facilitating *all* behaviors, thereby causing an increase in the diversity of behavior. In reward-caused behavior a stimulus subsequent to a random response causes that *particular* response to be repeated at the expense of all other random responses, thereby diminishing the diversity of behavior.

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