Carbonic Anhydrase in Seawater: Carbonato Complexes

With regard to the equilibration of atmospheric carbon dioxide with seawater and possible enzymatic control, as discussed by Berger and Libby (*1*), there is experimental evidence for the occurrence of carbonic anhydrase in marine and freshwater algae (*2*). The enzyme itself is a white crystalline solid, stable and active on storage at room temperature. I have used this material in our studies on the fractionation of carbon-13 between gaseous CO_2 and dissolved inorganic carbon species for obtaining rapid chemical and isotopic equilibrium (*3*).

The hydration and dehydration of CO_2 is a fundamental natural process, and it has been estimated that over 200 billion tons of CO_2 are annually exchanged across the ocean surface (*4*). I am studying the participation of any metal carbonato complexes aiding this process in view of the fact that the zinc moiety in carbonic anhydrase is reported to be significant in the enzymic reaction as a whole. The Zn-free enzyme is inactive, and the activity can be restored by the addition of Zn^{2+} ions. It is not clear under open-ocean conditions whether ion-pair complexes like $ZnCO_3^0$, $CuCO_3^0$, $MgCO_3^0$, $CaCO_3^0$, and the corresponding bicarbonate ion-pair complexes and the mixed amine carbonato complexes have something to do with the overall hydration and dehydration process (*5*).

I shall indeed be grateful for exchange of ideas and information in the field of metal carbonato complexes and their possible participation in the hydration and dehydration of CO_2.

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Plasticity of Hypothalamic Motivational Systems

Valenstein, Cox, and Kakolewski (*1*) have criticized an explanation that I offered (*2*) to account for an interesting phenomenon they had reported (*3*). This note is in reply to their criticism.

In their original study (*3*), Valenstein *et al.* examined the effects of electrical stimulation in the lateral hypothalamic area of the sated rat placed in a box with three "goal" objects—food, water, and a piece of wood. First, in preliminary tests, they found the level of current that reliably produced either eating of the food, drinking of the water, or gnawing of the wood at a particular electrode site. Then, using the same current level, they gave additional "stimulation experience" in the absence of the goal object to which each rat had responded in preliminary testing (for example, food), but in the presence of the two remaining goal objects (water and wood). They found that the additional stimulation experience made rats respond appropriately to a new goal object—that is, drinking or gnawing emerged as a result of further stimulation experience at the site that had originally produced only eating. Valenstein *et al.* explain this finding by suggesting (i) that the second stimulation-bound response was mediated by the same lateral hypothalamic drive system that originally mediated the first response, and (ii) that this drive system became capable of mediating the second response as a consequence of its modification by stimulation experience with the first goal object removed. Essentially, then, they postulated a single, plastic lateral hypothalamic drive system, whose behavioral influence is not genetically fixed, but can be altered by experience.

My alternative explanation was based on the assumptions (i) that the electrical stimulation simultaneously excited the fibers of two or more genetically fixed drive-specific systems, and (ii) that the emergence of a new response at a given current intensity reflected a change in the stimulation threshold for that response. The first of these assumptions was supported by my finding that if the stimulation intensity was raised after one stimulation-bound response was observed, a second response could always be elicited during the same testing session. The second assumption was supported by the finding that the threshold for the second behavior did in fact decline with repeated testing, until it was possible to elicit it at the same current level that had produced the first response. Thus my position was that the changes in the effects of the electrical stimulation which were observed in their experiment, as well as in mine, resulted from a change in the efficiency of transmission within genetically fixed, drive-specific systems, while the position of Valenstein *et al.* implied that it resulted from a modification of the efferent connections of a general plastic drive system.

In criticizing my explanation, Valenstein *et al.* (*1*) argue that the added variable in my experiment (manipulation of current level) produced no effect that they were not able to produce without it. They suggest that, in exploring for the current level at which I first elicited the second response, I gave my animals sufficient stimulation experience with the second goal object to produce the second behavior, just as they had done without raising the current. It is true that my animals received some stimulation experience with the second goal object before the current level was raised sufficiently to elicit the second response. However, it is clear from my data (*2*, Fig. 1) that this was not sufficient experience to produce the second behavior without current manipulation. The high thresholds that I found for the second response on the 1st day of testing represent, by definition, the lowest current level that would reliably elicit the response in question. Much more stimulation experience was required for the second response to emerge at the level of stimulation that was initially used to elicit the first response. Thus the threshold changes that I reported occur *during* the stimulation experience necessary to produce the "switching" observed in the experiment of Valenstein *et al.*, and *not after* that experience. It is because stimulation experience produces both the changes in stimulation-bound responding reported by Valenstein *et al.* and also the threshold changes that I reported, that I suggest a single explanation for both findings. The hypothesis that stimulation experience changes the efficiency (or sensitivity) of organized drive systems is necessary to explain my finding of threshold changes and is sufficient to explain the finding of Valenstein *et al.* The hypothesis that stimulation experience changes the drive specificity of a plastic drive system, while it does explain their finding, does not account for mine.

It should be emphasized that the is-