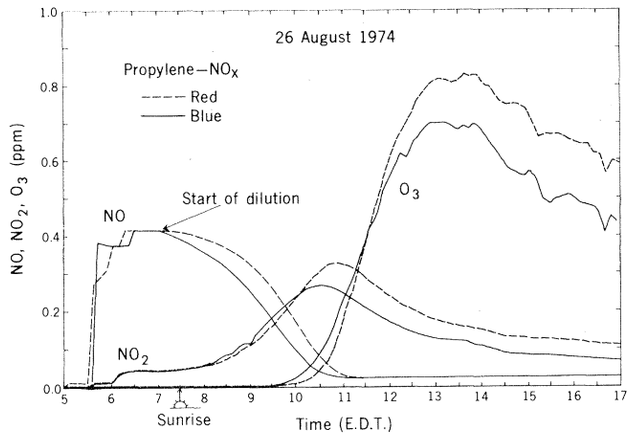

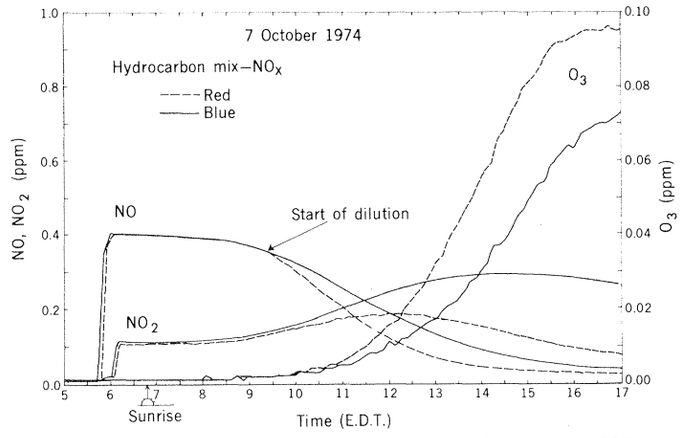

Fig. 1. Static/dilution experiment, with propylene as the hydrocarbon. Initial conditions: red side, 2.8 ppm of carbon NMHC and 0.45 ppm of NO_X; blue side, 2.6 ppm of carbon NMHC and 0.45 ppm of NO_X. Blue side diluted at 5.7 percent per hour. Fig. 2. Static/dilution experiment, with urban mix as the hydrocarbon. Initial conditions: red side, 2.4 ppm of carbon NMHC and 0.50 ppm of NO_X; blue side, 2.4 ppm of carbon NMHC and 0.50 ppm of NO_X. Red side diluted at 9.5 percent per hour.

erated in the blue side at a faster rate than it is being removed by dilution and actually appears almost as fast as NO_2 in the red (undiluted) chamber. This creates a situation in which the ratio of NO_2 to NO is higher in the blue chamber than in the red. If one assumes that pseudophotostationary equilibrium can be used to approximate the O_3 concentration at each point in time (Eq. 1), then the ratio of the NO_2 concentration to the NO concentration governs the O_3 concentration at a given light intensity.

$$\frac{[NO_2]}{[NO]} = \frac{k_3[O_3]}{k_1} \quad (1)$$

where k_1 (Φk_a for NO_2) is the rate constant for the reaction

$$NO_2 + h\nu - NO + O$$

and k_3 is the rate constant for the reaction

$$NO + O_3 \rightarrow NO_2 + O_2$$

In the reactive system (Fig. 1) O_3 is initially generated more quickly in the diluted side. However, NO_x is being removed by dilution and the potential NO_2 peak is therefore greater in the undiluted side. Hence, a higher ratio of NO_2 to NO is ultimately reached in the undiluted side during that period of the day when the solar intensity is greatest (that is, greater value for k_1) and is accompanied by a faster O_3 generation rate and higher O_3 concentrations.

The less reactive smog system can be analyzed in a similar manner. Both sides initially contain an urban hydrocarbon mix consisting of 2.4 ppm of carbon and 0.5 ppm of NO_x (20 percent (NO_2). Dilution begins in the red side at about 09:00 (Fig. 2), and, soon after, this side is generating O_3 faster than the static side. The major difference between this less reactive system and the reactive propylene system is that for the less reactive system a higher ratio of NO_2 to NO is not reached in the undiluted side until the sun is beginning to set. The net effect is lower and lower values of k_1 during the greatest period of potential O_3 generation for the undiluted side.

In summary, the effect of different dilution rates and time at which dilution begins cannot be quantified at this time. However, on the basis of the two experiments that have been presented here, certain phenomena of dilution/static smog systems may be elucidated. Reactive systems, which would normally achieve maximum oxidant values during the early afternoon, may initially generate oxidant faster under dilution conditions but probably will not reach the oxidant concentrations generated by static systems. By contrast, less reactive systems under certain dilution conditions may actually generate more oxidant. In the experiment considered here, the oxidant concentration of the diluted side actually exceeded the federal air quality standard (0.08 ppm) (*1*) although that of the undiluted side did not. Greater control of the hydrocarbon content as compared to the NO_x content tends to shift the urban atmosphere in the direction of one containing less reactive mixtures. Dilution as opposed to static experimental conditions may prove to be more important if smog chamber data are to be used as guides in developing certain future control strategies.

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Behaviorally Evoked Hippocampal Theta Waves: A Cholinergic Response

Abstract. *Forced running in a treadmill results in the instantaneous appearance of a synchronous electroencephalographic pattern in the dorsal hippocampus of the rat. A similar bioelectric response in the absence of movement is induced by physostigmine. Both responses are blocked with scopolamine. Lesions of the medial septal nucleus abolish hippocampal theta waves induced by forced running or physostigmine.*

There is substantial evidence that the hippocampal theta pattern recorded from the unanesthetized, unrestrained mammal is associated with the initiation of movement (*1–4*). This bioelectric pattern can be elicited instantaneously and maintained indefinitely by placing the animal upon a moving treadmill (*4*). The amplitude of these behaviorally evoked hippocampal theta waves varies directly with the speed of movement (*4*).

Earlier electrophysiological experiments with acute, immobilized preparations have demonstrated that hippocampal theta patterns can be obtained with either high-frequency electrical stimulation of the brainstem reticular formation or with systemic administration of physostigmine, a cholin-

esterase inhibitor. These rhythmic hippocampal slow waves were abolished by lesions of the septum (*5*).

According to Vanderwolf (*6*) there are two distinct rhythmic responses recorded from the hippocampus: a low-frequency response (4 to 7 hertz) associated with behavioral arousal and a synchronous response of higher frequency (7 to 12 hertz) associated with the initiation of movement. Vanderwolf (*6*) finds that the low-frequency "activation" response is sensitive to muscarinic blockade by atropine, while hippocampal synchronous activity associated with movement is not affected by cholinergic blockade.

In our forced-running experiments (*4*) and in earlier work (*2*) we found a synchronous theta response (7 to 10 hertz) associated with movement. This response persisted in the resting animal after a bout of running, suggesting the possible buildup of neurotransmitters as a consequence of forced running.

Since physostigmine causes a buildup of acetylcholine levels throughout the central nervous system, producing a marked theta response in the hippocampus, we felt it would be instructive to compare the response of the hippocampus to forced running and to physostigmine, then challenge both responses with scopolamine (a known cholinergic blocking agent). An evaluation of the effect of septal lesions on both responses was also carried out.

Thirty male Sprague-Dawley rats weighing 230 to 280 g were anesthetized with sodium pentobarbital (50 mg/kg) and electrodes were implanted in the dorsal hippocampus, septum, and posterior cerebral cortex. Bioelectric signals were monitored from recording electrodes in the freely moving subject 1 week after surgery. The signals were amplified differentially by Tektronix FM 122 units with a band-pass setting of 0.2 to 50 hertz and were recorded on a Dynograph paper chart recorder. On-line frequency analysis of the signal in the range of 2.0 to 20 hertz was carried out with an Ortec time histogram analyzer in conjunction with a Krohn-Hite band-pass filter. The rats were initially placed on the treadmill with the motor off, and a baseline (sitting) recording was obtained. Next, the treadmill was disengaged from the drive shaft and the motor turned on. This procedure served as a control for activation produced by the sound of the treadmill motor and as a control for possible electrical artifacts generated by the motor. The rats were then forced to run at a speed of 8 cm/sec for sessions lasting approximately 5 minutes. Drug dosages were 0.5 and 1.0 mg of physostigmine per kilogram and 5 and 10 mg of scopolamine hydrochloride per kilogram. Septal lesions were produced through the implanted electrodes in the unanesthetized animal. Tests of the effectiveness of the lesion were made 30 minutes and 1 week after the lesion was produced by passing 2 ma of direct current for 20 seconds through the bipolar electrodes.

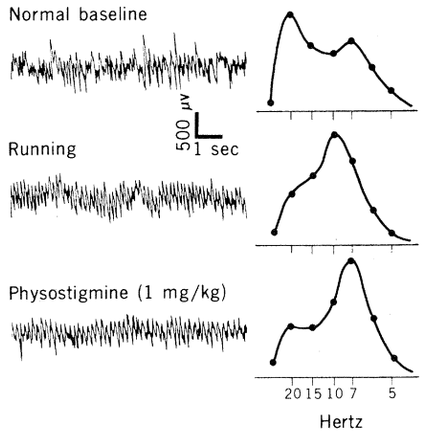

Fig. 1. Typical EEG tracings (rat No. 331) from the dorsal hippocampus of the rat sitting quietly (top), running (middle), and after intraperitoneal injection of physostigmine. Frequency analyses of these signals are shown to the right of the tracings.

Twenty-three of 30 rats exhibited a well-defined hippocampal theta response when forced to run in the treadmill. Presented in Fig. 1 are electroencephalographic (EEG) tracings obtained from one such animal. The top record shows the hippocampal EEG while the rat is sitting quietly. Frequency analysis shown to the right indicates a wide spectrum with two peaks, one at 20 hertz and the less prominent peak at 7 hertz. With forced running, we obtain a prominent shift in the hippocampal bioelectric pattern (Fig. 1, middle tracing). This synchronous pattern appeared instantaneously with the onset of running and persisted for minutes after a 5-minute session of sustained running. Frequency analysis of this epoch shows a prominent peak at 10 hertz.

Histological analysis of electrode placements indicates that the successful cases (N = 23) were those having electrode tips in close proximity to the pyramidal cell layer of the dorsal hippocampus. If the electrode tips were at the ventral surface of the hippocampus, no change in bioelectric activity was obtained with running. Furthermore, those rats showing no theta response to forced running showed no EEG response to physostigmine (0.5 and 1.0 mg/kg). Of 23 rats showing a behaviorally evoked hippocampal theta response, 19 rats showed a prominent theta response when given 1 mg of physostigmine per kilogram by intraperitoneal injection. Drug-induced theta was seen 3 minutes after the injection, and lasted for about 10 minutes.

The lower dosage of 0.5 mg of physostigmine per kilogram had little or no effect upon the hippocampal signal. A comparison of the EEG tracing and corresponding frequency spectra produced with running and physostigmine (1.0 mg/kg) is shown in Fig. 1. Although there was a tendency for the physostigmine response to be of a slightly lower dominant frequency, the response to both treatments was remarkably similar in every animal. All the rats

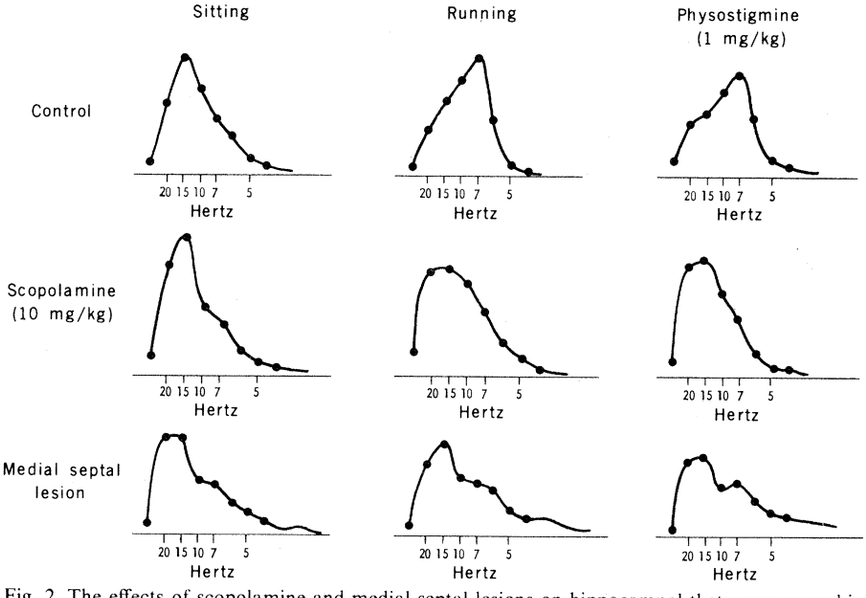

Fig. 2. The effects of scopolamine and medial septal lesions on hippocampal theta responses: hippocampal frequency spectra of rat No. 347.

showing physostigmine-induced theta responses were relatively immobile; they exhibited piloerection, defecation, shivering, and contractions of the facial musculature, best characterized as "cheek puffing." The four rats that showed running-induced theta, but no response to physostigmine, did not show much of an overt autonomic response to the cholinergic drug; they exhibited piloerection, but were relatively free of tremors.

Because of its toxicity, it was not advisable to administer physostigmine in doses higher than 1.0 mg/kg. However, two rats that had exhibited excellent theta responses with running but no response to 1.0 mg of physostigmine per kilogram did exhibit a prominent theta response to a dose of 1.5 mg of physostigmine per kilogram. There was never a case where a rat exhibited a physostigmine-induced theta response and failed to show theta with running.

Another test of the hypothesis that behaviorally induced hippocampal theta is a cholinergic response is to compare the effect of scopolamine, a known muscarinic blocking agent, on both physostigmine-induced and running-induced hippocampal theta responses. The results of this experiment are shown in Fig. 2. While scopolamine (10 mg/kg) had no discernible effect upon the hippocampal frequency spectrum when the rats were sitting quietly (Fig. 2, middle row, left), the synchronous theta responses to running and physostigmine (1 mg/kg) were completely blocked when challenged with scopolamine (compare top row with middle row in Fig. 2). This effect, which began 3 minutes after scopolamine administration (10 mg/kg, intraperitoneally) lasted for 40 minutes. It was seen in every animal (N = 10). Despite this dramatic block of behaviorally induced theta, scopolamine had no effect whatever on running in the treadmill. The behavioral topology of the scopolamine-treated rats was identical to the behavior of normal rats.

As expected, scopolamine (10 mg/kg) when mixed with physostigmine (1 mg/kg) completely blocked physostigmine-induced hippocampal theta responses (Fig. 2, middle row). This blockade had little or no effect upon the overt effects of physostigmine such as shivering, piloerection, and "cheek puffing." The lower dose (5 mg/kg) of scopolamine was not effective in blocking hippocampal theta responses.

Since Petsche *et al.* (*5*) have shown that destruction of the medial septal nucleus abolished physostigmine-induced hippocampal theta responses, the effect of septal lesions on drug-induced and behaviorally induced hippocampal theta patterns was compared. It was possible (N = 6) to completely abolish the theta response to both forced running and physostigmine with lesions of the medial septal nucleus. Lesions of comparable size that missed the target had no effect on behaviorally induced hippocampal theta responses (N = 3).

In one such case, the cingulum (a prominent pathway to the hippocampus) was destroyed. The pathway from the medial septal nucleus to the hippocampus was spared, and so was the response to running and to physostigmine.

It is possible that scopolamine could act directly on hippocampal neurons to block the behaviorally induced theta response. Feldberg and Vogt (*7*) and MacLean (*8*) have demonstrated that the hippocampus is rich in cholinergic neurons. Recently, Kuhar *et al.* (*9*) have demonstrated that medial septal lesions cause a profound reduction of acetylcholine levels in the dorsal hippocampus. There is evidence that the cells of the medial septal nucleus are cholinergic and act as pacemakers for the theta rhythm of the hippocampus (*5*). In addition, Friedman and Wikler (*10*) have shown that cholinergic agents acting directly upon cells of the posterior hypothalamus have a profound effect upon the hippocampal theta rhythm produced by means of electrical stimulation of the brainstem and hypothalamus. For this reason, it is not yet possible to specify where the scopolamine is acting to block the running-induced hippocampal theta response. The present findings are of considerable significance in that they provide a demonstration of a highly localized brain bioelectrical alteration, brought about by behavioral manipulation, that can be instantaneously induced, maintained for a considerable period of time, and can be related to the activity of a specific neurotransmitter.

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Floating Glacial Ice Caps in the Arctic Ocean

Abstract. *Two arguments are presented, one in favor of the existence of thicker ice in the Arctic Ocean during glacial time, and the other in favor of a full-fledged Arctic ice cap. The first is based on the Greenland air temperature record obtained from isotopic studies of the Camp Century ice core. The second is based on the oxygen isotope record of benthic foraminifera from a deep Pacific Ocean core.*

Although the idea of the areal expansion of floating ice in the Arctic Ocean and adjacent seas during glacial times receives frequent mention in the literature, only a few references to the possibility that much thicker ice existed can be found. Mercer (*1*) suggested that ice cover of the west Antarctic type may have developed in the Arctic Ocean during prolonged times of full continental glaciation. He also pointed out that in 1888 Sir William Thomson proposed to the Geological Society of Glasgow that a lapse in circulation between the Arctic Ocean and neighboring seas would cause it to be filled with solid ice. I would like to present two arguments in support of this old but not particularly fashionable idea. The first, which is quite firm, calls for thicker ice than at present, and the second, which is rather tenuous, calls for a very thick and largely buoyant "cap."

Under the climatic conditions which existed during glacial time, thicker Arctic ice is to be expected. To understand this we need to consider the factors controlling the present-day ice conditions in the Arctic and then project what might have been the case during times of major continental glaciation. In today's Arctic the ice stabilizes at a thickness of a few meters. The net loss of ice resulting from surface ablation in the summer is compensated by basal growth of sea ice during the winter. The rate of freezing beneath the ice depends on the rate of conduction of heat through the ice to the atmosphere. Hence the amount of winter growth is strongly dependent on ice thickness (other factors being equal, the growth