and microelectrodes were inserted into single retinular (photoreceptor) cells in accordance with procedures which are described in detail elsewhere (*7*). These receptor cells produce graded depolarizations (reductions of membrane potential) in response to light. Optic nerve discharges (or spike frequencies) are linearly related to the depolarizations induced in the receptor cell (*8*). The receptor cells were kept in a constant state of dark adaptation by regularly applying a constant test stimulus. The response to the test stimulus provided a control against possible changes in the viability of the receptor during the experiment.

The results of a representative experiment are shown in Fig. 2. Each group of graphically superimposed traces represents responses to light flashes of a given intensity but of different durations. The intensities are indicated in relative log units in the figure; we also obtained responses to other intermediate intensities. Four different durations, namely, 10, 40, 160, and 640 msec, are illustrated; we obtained responses to intermediate durations as well. The responses to the different durations can be easily identified since longer durations always gave more prolonged responses. Each trace in the figure begins at the beginning of the light flash and each trace is a plot of the instantaneous response as a function of time after stimulus onset for a given stimulus. Each group of superimposed traces illustrates the totality of the responses as a function of stimulus duration for a given intensity.

The range of intensities employed begins below the level at which a clear-cut neural transient occurs (compare 0.0 log units). These responses to the weakest stimuli are quite irregular; this is characteristic of the response to weak stimulation in dark-adapted visual systems. Despite these irregularities, it is fairly clear that no appreciable transient occurs at the lowest intensity; progressive increases in intensity elicit a progressively clearer transient. Moreover, the longest duration produces a clear-cut plateau except at the very highest intensity where the cell has not yet equilibrated after the transient (compare 3.6 log units). Thus these data cover the entire range of stimuli that might be expected to produce a neural correlate of brightness enhancement.

Certain trends are clearly present in these data. First, longer stimuli always produce responses whose peak height (and hence whose associated maximum spike frequency) is greater than or equal to that produced by shorter stimuli. Second, longer stimuli always produce responses whose total area (and hence whose associated total number of spikes) is greater than that produced by shorter stimuli. Thus, the neural response to lights of varying durations is always monotonically related to stimulus duration. There is no correlate in these data of the psychophysical phenomenon of brightness enhancement even though we have clearly elicited neural transients and even though we have covered the entire range of appropriate stimuli. We have seen no exception to this generalization in any of our experiments. Although we have discussed only two features of the total response, namely, peak height and area, no other feature appears to violate this generalization (*9*). Latency appears to be independent of stimulus duration.

These data do not demonstrate that some other single-cell response might not be isomorphic with brightness enhancement although we are not presently aware of any nonillusory single-cell correlate of the Broca-Sulzer effect. But the mere presence of a neural transient at *any* level of the nervous system cannot be presumed to be related to the Broca-Sulzer effect. The search for the correlate of brightness enhancement cannot benefit from the assumption that stimulus duration is in any simple way related to time in the nervous system.

Such an assumption is likely to have frequently occurred in the analysis of other substantive problems because of certain differences in the methods employed in the two types of research: Psychophysical experiments manipulate duration because there is no reliable way of obtaining a continuous numerical description from a subject of the temporal course of perceptual events. On the other hand, physiologic experiments automatically provide a measure of activity as a function of time. A substantial additional investment has to be made to manipulate stimulus duration as a separate parameter. Other postulated psychophysiologic isomorphisms involving temporal factors might therefore be examined for the possible presence of an illusory correlation by exactly replicating the psychophysical procedure with the physiologic material.

GERALD S. WASSERMAN
KING-LEUNG KONG
Department of Psychology, University of Wisconsin, Madison 53706

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5. Such observations have been repeatedly made since the days of Adrian and Matthews (*2*). Reviews of this material may be found in any authoritative treatment, such as G. S. Brindley, *Physiology of the Retina and Visual Pathway* (Williams & Wilkins, Baltimore, 1970).
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9. These data were collected as part of a study on the reciprocity of stimulus intensity and duration. Previous single-cell studies of reciprocity (*2*) contain data which can be subjected to the same analysis that we have here applied to our data. Such analyses corroborate and lend generality to our findings.
10. This research was supported by funds provided by the University of Wisconsin Research Committee. Earlier versions of this report were kindly read by L. J. Chapman, W. Epstein, and C. T. Snowdon.

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Maternal Lymphocytes: Suppression by Human Chorionic Gonadotropin

Adcock *et al.* (*1*) devised an ingenious theory of fetal protection based on the proposition that human chorionic gonadotrophin (hCG) is a constituent of the trophoblast surface and that it can inhibit maternal lymphocytic responses. They tested the latter part of the proposal by attempting to stimulate lymphocytes with phytohemagglutinin (PHA) in the presence of hCG. Since they observed apparent inhibition, they concluded that their original theory remains tenable.

There might be another explanation for their findings. That is, hCG, a protein that contains 31.3 percent carbohydrate, has little or no effect on the cells, but acts by combining with

and neutralizing the effect of the PHA. There is considerable information to support this alternative. First, PHA is known to form complexes with glycoproteins (*2*) and, second, the degree of stimulation by PHA is known to be dependent on the amount of serum present in the medium. This may well be due to the glycoprotein content (*3*). In my experience, I have found that, in the same culture system used by Adcock *et al.*, lymphocyte stimulation by 1 : 200 dilutions of PHA is much more sensitive to differences in plasmas than lymphocyte stimulation by dilutions of 1 : 40. Using concanavalin A as a mitogen, a molecule having biological properties very like those of PHA, Tomford and I (*4*) found that 0.25 μg of concanavalin A induce maximal incorporation of ^{3}H-labeled thymidine in 4 million lymph node cells in medium containing 1 percent crystalline bovine serum albumin as the protein source, but 10 to 15 μg are required in the presence of 15 percent homologous serum. Nevertheless, the total amount of thymidine incorporated at maximal response was the same for the two systems. This is evidence that the effects of the inhibitor can be overcome through saturation quantities of the lectin. If the effects of an inhibitory system were on the cells, inhibition could not be overcome by such means. In the concanavalin A system, inhibition similar to that observed by Adcock *et al.* was achieved with methyl-α-D-mannoside, a sugar with a high binding affinity for concanavalin A but which has no discernible effects on lymphocytes at isosmotic concentrations (*5*).

This alternative could be tested by measuring the inhibition of PHA stimulation by hCG, as a function of PHA concentration, or by stoichiometric characterization of any hCG-PHA interactions.

ARNOLD E. POWELL

Department of Surgery,
Case Western Reserve University,
Cleveland, Ohio 44106

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We agree with Powell (*1*) that other possible theories could be proposed which would be equally tenable to explain the effect of human chorionic gonadotropin (hCG) on the stimulation of lymphocytes with phytohemagglutinin (PHA). It is certainly true that the evaluation of the hCG effect will be aided by characterizing the stoichiometric relation between hCG and PHA interactions. While such work is needed, we are inclined to think that Powell's explanation involving a direct effect of hCG on PHA is unlikely in the light of recent data, published from our own laboratory (*2*) as well as from other centers, that hCG also inhibits mixed lymphocyte cultures. The latter involves stimulation of lymphocytes by foreign lymphocytes rather than by PHA. We believe the effect of hCG is likely to be a direct one upon the lymphocytes, although the data we presented in *Science* would certainly not permit us to make that distinction.

EUGENE W. ADCOCK III

Department of Pediatrics,
University of Texas Medical School,
Houston 77004

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Pollution in Coastal Waters

Some important considerations regarding the dispersion of pollutants in the nearshore waters are outlined by Inman and Brush (*1*). However, their use of McClure and Barrett's data (*2*) on the distribution of DDT [1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane] and DDE [1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethylene] in zooplankton to demonstrate the "concentration gradient" argument is inappropriate, because the selection of units (10^{-8} g of DDT and DDE per cubic meter of surface water) does not account for spatial variations in zooplankton biomass. Such biomass gradients in California coastal waters have been documented in the past (*3*). For the period 1955 through 1959 we estimate that the ratio of inshore to offshore zooplankton biomass (B_I/B_O) fluctuated roughly between 4.0 and 0.1; these variations occurred both yearly and seasonally. B_I and B_O correspond to the average zooplankton biomass (grams per 1000 m^3), all taxa combined, for the inshore and offshore regions, respectively. We used the 100-km offshore line as a hypothetical separation zone between inshore and offshore waters from Monterey Bay to Point Dume. If we assume a similar variation of the ratio throughout the period 1959 to 1969, converting the data to units of (grams of DDT and DDE per gram of zooplankton, wet weight), and use 4.0 as an upper bound value of B_I/B_O, then the reported "hot spots," corresponding to McClure and Barrett's isopleths of 5.0×10^{-8} g of DDT and DDE per cubic meter, are damped out, resulting in a nearly uniform inshore-offshore distribution. However, if a lower bound B_I/B_O value of 0.1 is used, an even larger inshore to offshore gradient is established. Therefore, any conclusive statements based on these data should contain values that have been normalized to the zooplankton biomass ratio measured in 1969.

Since the major concern in assessing biological consequences of various toxic pollutants in the marine environment is an evaluation of their impact on the standing crop and the growth dynamics of the biological systems in question, concentration data should always be normalized to biomass-related parameters, so that meaningful interpretations can be made.

Inman and Brush also state that "the effective rate of mixing [of pollutants in nearshore waters] depends both upon the mechanics of the [mixing] phenomena . . . and upon the concentration gradient of the pollutant that is being mixed," the latter being "dependent upon the nature of the substance and its past history of dispersion in the area." If physical dispersion or ambient concentration is implied, the above statement is redundant, since the concentration gradient is a function of the mixing process and cannot be considered independently. If "dispersion" connotes biological accumulation, then "biological accumulation gradient" might be a more appropriate wording.

S. P. PAVLOU
J. R. CLAYTON, JR.

Department of Oceanography,
University of Washington,
Seattle 98195