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.

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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.

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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.

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23 July 1973

Pavlou and Clayton appear to have missed the point of our discussion of pollution in coastal waters. In our figure 11, we used data from McClure and Barrett (*1*) as an illustration of the fact that "wind- and wave-induced surface currents tend to produce circulation patterns that favor the retention of particulate material near the coast . . . , whereas biological scavenging and absorption by suspended particles (both biogenous and inorganic) concentrate dissolved pollutants in coastal waters" (*2*). In this context the figure we used serves as a valid illustration. We had no intention of entering into a discussion of methods of normalization of biological data, a procedure that is subject to considerable controversy. However, even when the data in figure 11 (*2*) are normalized, as suggested by Pavlou, "the gradients are quite definitely still there! The 'hot spot' still shows apparent concentrations fivefold higher than the average over the whole grid" (*3*).

Pavlou and Clayton comment that a statement in our discussion of mixing is redundant. The concentration gradient can only be described as a "function of the mixing process" when both the mechanics of mixing and the sources or inputs are processes which do not vary independently. For example, a concentration gradient established at some time at a fixed point by mixing processes may change at some later time if subjected to different processes or to variations in their intensities. Further, a concentration gradient previously established by mixing processes at some point may change after advection to some other point where there are different sources and different mixing processes. Also, the source is not always in the form of point sources, as in the case of river runoff or ocean outfall; occasionally, offshore winds can introduce concentrations of pollutants over large water areas. If these contaminants remain in the surface waters, they will tend to be contained against the coast by the prevailing northwesterly winds. Thus, our statement is not redundant.

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19 March 1974

Conditioning or Control?

Harris *et al.* (*1*) described an "instrumental" conditioning procedure in which reinforcement (food-reward and shock-avoidance) was contingent upon specified elevations of the diastolic blood pressure of baboon subjects. The observed significant elevations for each of the four subjects were of large magnitude (30 to 40 mm-Hg) and were sustained over 8 to 10 weeks. The authors stated that the response change was "directly and specifically" a result of the programmed contingencies of reinforcement. In my view the data do not unequivocally support this conclusion.

Demonstrative conditioning of autonomic nervous system activity in general, and cardiovascular responses specifically, requires that the autonomic response of interest be neither an unconditioned response nor mediated by somatic activity (that is, skeletal muscle activity or respiration) (*2*). The possibility of such mediation has been shown for human and infrahuman subjects (*3*). A variety of control tactics has been specified for minimizing such confounding effects, include: bidirectional control, paralysis of skeletal musculature by curariform drugs, yoked-control subjects, and the differential conditioning of a presumed mediating response (*4*). Harris *et al.* report that two additional animals were exposed to identical reinforcement contingencies for *decreases* in diastolic blood pressure, thus completing the bidirectional procedure. If bidirectionality was not observed, then unconditioned mediators cannot be dismissed. The authors reported that this procedure failed to produce decrements after 6 months of training.

Animals reinforced for increments in diastolic blood pressure received a mean of two electric shocks and 25 food pellets per hour. Similar data for the control group were not presented, and it is likely that these animals (not meeting the response requirement) received considerably more shocks and less food. Such an inequality might provide the basis for unconditioned or classically conditioned responses mediating the blood pressure response and entirely account for the differences in blood pressure between the group reinforced for increments and the group reinforced for decrements. Additionally, although short-term peripheral mediation might be ruled out, failure to include concomitant measures of respiration and skeletal muscle activity does not allow specification of possible long-term mechanisms mediating the increments in diastolic blood pressure.

In the absence of such control procedures, it appears judicious to adhere to the recognized distinction proposed by Black (*5*) between *control* and *conditioning* of autonomic responses, the latter reserved for response changes directly attributable to the response-reinforcer contingency. Thus the effects obtained by Harris *et al.* are accurately described as representing control, not conditioning.

This criticism does not deny the utility of the experimental model proposed by Harris *et al.*, but rather urges an important distinction in the study of behavioral-physiological mechanisms that mediate cardiovascular activity.

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