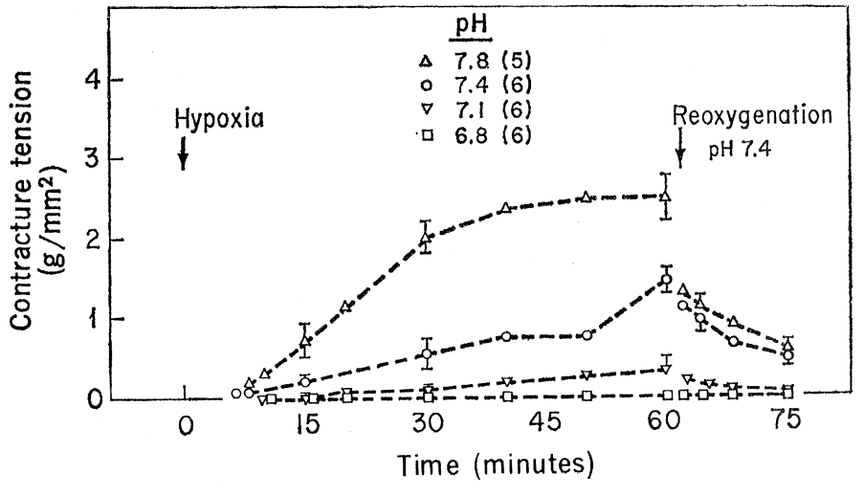

Fig. 2. Contracture tension, developed by rat heart muscle preparations during hypoxia and reoxygenation in the presence of 5 m*M* glucose. The numbers of muscle preparations are in parentheses; brackets show ± 1 standard error of mean. During hypoxia, the most severe contracture is seen at an alkaline *p*H. No contracture occurs during hypoxia at *p*H 6.8.

95 percent O_2 and 5 percent CO_2 took place at *p*H 7.4. The in vitro length of each muscle was measured at the apex of its length tension curve. Following each experiment, the muscle was blotted and weighed, and cross-sectional area was calculated by assuming cylindrical uniformity and a specific gravity of 1.000. Developed and contracture tension were normalized for muscle cross-sectional area. Changes are expressed in absolute terms or as a percentage of prehypoxia control values.

The mechanical activity of isolated rat heart muscle during hypoxia at *p*H 7.4 has been described (*3*). At an acid *p*H, developed tension declined rapidly early during hypoxia (Fig. 1). At *p*H 7.8, on the other hand, higher levels of tension were present at this time. After 15 minutes of hypoxia, developed tensions at *p*H 6.8, 7.1, 7.4, and 7.8 were 13 ± 2.1, 18 ± 2.6, 26 ± 1.6, and 39 ± 3.4 percent of prehypoxia control values, respectively. Developed tension at *p*H 7.4 at this time was significantly different from that at *p*H 6.8 ($P < .001$) and *p*H 7.8 ($P < .01$). Thus, during early hypoxia, an alkaline *p*H enhanced the performance of hypoxic heart muscle while an acid *p*H depressed tension development. These observations are in agreement with those of others (*4*) and document the additional depressive effect of acid *p*H on the mechanical performance of hypoxic heart muscle. Despite the rapid decline in mechanical activity early during hypoxia at acid *p*H, developed tension stabilized, and after 60 minutes approximately 10 percent of prehypoxia tension was developed in all *p*H groups.

After 15 minutes of reoxygenation at *p*H 7.4, preparations previously hypoxic at *p*H 7.4 and 7.8 redeveloped approximately 50 percent of prehypoxia tension; in preparations previously hypoxic at *p*H 6.8 and 7.1, developed tension returned to almost 100 percent of prehypoxia values.

Contracture during hypoxia appeared earliest and to the greatest degree at *p*H 7.8 (Fig. 2). After 60 minutes, contracture tension was 44 percent of prehypoxia developed tension. Lesser degrees of contracture were seen at *p*H 7.4 and 7.1. At *p*H 6.8, no contracture was observed at any time during the 60-minute period of hypoxia. Upon reoxygenation, contracture gradually diminished as recovery took place. Most rapid and complete recovery was seen in those preparations previously functioning at acid *p*H.

During hypoxia, mammalian cardiac muscle must rely on limited stores of anaerobic substrate to maintain activity and preserve integrity. Recovery of function after hypoxia may depend on the extent to which energy stores are depleted during hypoxia. It is well recognized that *p*H influences the activity of several glycolytic enzymes. An acid *p*H, by inhibiting glycolysis, may conserve carbohydrate reserves and facilitate recovery after a period of hypoxia. An equally important mechanism by which a low *p*H may preserve energy stores of ischemic or hypoxic myocardium is by depression of contractile activity, a major energy-consuming reaction in the heart (*5*). It is also possible that *p*H changes during hypoxia may influence the transport of substrate into the cell.

If these observations on isolated heart muscle during hypoxia can be extended to myocardial ischemia in the intact animal, the present results may have clinical relevance. Acidosis is generally implicated in the development of irreversible deterioration and cell death following coronary occlusion. The present experiments have demonstrated a protective effect of acidosis during hypoxia on heart muscle function following hypoxia. It would seem possible, at least during early hypoxia, that acidosis may merely accompany and perhaps even retard other intracellular events that are responsible for cell deterioration.

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6. Supported in part by the Medical Foundation, Massachusetts Heart Association grant 940, NIH grant HE-08613, and Boston City Hospital general research support grant 5S01RR-5569. We thank Ms. K. E. Hansen for her assistance.

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Phytoplankton Algae: Nutrient Concentrations and Growth

I believe that O'Brien misinterpreted the studies of Monod and others in his report "Limiting factors in phytoplankton algae: their meaning and measurement" (*1*). It is not true that ". . . changes in the concentration . . . of most factors that have been identified as limiting . . . cause changes in the growth rate, but not necessarily in the final yield."

If O'Brien had extended the curves in the figure showing what he terms "type II growth," he would have had to show a difference in final yield, as he did in the figure for "type I growth." All he demonstrates in the figure for type I is that (i) nutrients are not rate limiting (that is, they are saturating) and (ii) the final yield is directly proportional to the concentration of the limiting nutrient. The nutrient concentration in the figure for type II is both rate limiting and yield determining.

Therefore, it can only be said that growth yield will depend on the concentration of a limiting nutrient and not necessarily on how fast growth can occur. These are principles that are well known in bacteriology.

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Reference

1. W. J. O'Brien, *Science* **178**, 616 (1972).

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O'Brien (*1*) has made a distinction between two phytoplankton growth responses: increase in final biomass (type I) and increase in growth rate (type II). We believe that the quantitative dynamics of growth, including changes in both growth rate and final biomass, must be considered.

If we express growth by the Monod relationship (Michaelis-Menten kinetics), as O'Brien (*1*) and several other workers (*2, 3*) have, and use the nutrient content of the biomass (N_b) to express biomass or population, we may write

$$\frac{dN_b}{dt} = \mu N_b \left(\frac{N_s}{K_s + N_s}\right) - RN_b \quad (1)$$

where μ is the maximum growth rate, N_s is the nutrient in solution, K_s is the value of N_s at which $dN_b/dt = \frac{1}{2}\mu$, and R is the respiration rate expressed as nutrient liberated from the biomass per unit biomass per unit time. If N_t is the total nutrient material in the biomass and in solution, $N_t = N_s + N_b$, and by substitution

$$\frac{dN_b}{dt} = \mu N_b \left(\frac{N_t - N_b}{K_s + N_t - N_b}\right) - RN_b \quad (2)$$

A change in total nutrient must produce a change of nutrient uptake, which must be related to growth rate. If N_s is very large with respect to K_s, then a change in N_s will produce a slight or undetectable change in growth rate. This is the situation described by Goldman *et al.* (*4*) and cited by O'Brien (*1*). If, however, the initial N_s used in an experiment is similar to that normally encountered in nature, it will be of the same order of magnitude or smaller than K_s, and a change in nutrient concentration will produce a change in growth rate. This is shown by the K_s values measured by Eppley *et al.* (*3*) and other workers (*2*). Type I growth is therefore an experimental artifact except in cases of very extreme enrichment.

Type II growth, in which N_t does not affect final biomass, cannot occur if Monod kinetics pertain. Consider the final, or equilibrium biomass: substituting 0 for dN_b/dt and solving for N_b gives

$$N_b = N_t - \frac{R}{\mu - R} K_s \quad (3)$$

If R is very small in respect to μ, then $N_b \cong N_t$. The final biomass depends on the nutrient added.

Similar conclusions may be drawn if we assume Verhulst or logistic growth kinetics. If we define some maximum growth rate (r) analogous to the intrinsic rate of growth and assume that the carrying capacity is defined by N_t, then

$$\frac{dN_b}{dt} = rN_b \left(\frac{N_t - N_b}{Nt}\right) - RN_b \quad (4)$$

If N_t is much larger than N_b, then as with Monod kinetics, a change in N_s might be difficult to detect. It seems unlikely, however, that in most natural situations or well-designed experiments more than an order of magnitude increase of N_t would occur, and again the change in growth rate would be detectable. The equilibrium solution is

$$N_b = \left(\frac{r - R}{r}\right) N_t \quad (5)$$

and the final biomass depends on the nutrient added. Type II growth can not occur.

From this discussion it can be seen that if Monod kinetics pertain nutrient addition will increase both growth rate and final biomass. Although other factors—such as grazing, light availability, and temperature—affect growth rate in the natural environment, there is good evidence that Monod kinetics pertain (*2, 3*), and even if they do not it seems likely that the growth mechanism is similar to either the Monod or the logistic model. Eutrophic lakes support larger spring and summer phytoplankton biomass and productivity, as suggested by Eq. 3 (*5*).

We submit that an increase in equilibrium biomass due to increase of a limiting nutrient should be and usually is accompanied by an increase in growth rate, both in the laboratory and in the field. A particular experiment or set of observations may detect one or both of these symptoms of eutrophication. The experimenter and the field worker should consider phytoplankton growth kinetics in terms of both standing crop and productivity, nutrient uptake or growth rate; a large increase in either indicates release from limitation and possibly incipient eutrophication.

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7 December 1972

I agree with both Holmes and Kelly and Hornberger that type II growth would have to show a difference in final yield given sufficient time or, graphically, that the lines describing type II growth can be extended to show a change in yield. In ideal field or experimental situations a change in both growth rate and final yield should be observed with the addition of the limiting nutrient. However, natural populations do not have unlimited time, but often have severe rates of mortality, which can be offset only by rapid changes in growth rate; therefore, experiments to detect limiting factors must demonstrate changes in growth rate.

A major point I wished to make is that one cannot rely simply on a change in yield as a definitive proof of nutrient limitation in many experimental settings. Experiments in which populations are enclosed within test flasks for extended periods of time, and in which greater yields are observed in flasks to which nutrients have been added, demonstrate little about the dynamics of natural populations. In such experiments

the populations are completely cut off from nutrient recycling within the water body and isolated from most, if not all, mortality factors; therefore, some nutrient within the test flask must ultimately be used up and become "limiting." That nutrient is not necessarily or would not necessarily become limiting to the populations within a lake or other natural situation.

I disagree that type I growth is simply a result of experimental error. As I reported, most so-called batch bioassay experiments yield data of this type. Holmes and Kelly and Hornberger are correct when they claim that by the theory of Monod and Michaelis and Menten type I growth would occur only when the nutrient is added in high concentrations relative to the K_s value. In fact, because of lag times and other factors which I mentioned (*1*) experiments must be designed this way and the outcome is as I predicted.

I share the enthusiasm of Kelly and Hornberger for the importance of Michaelis-Menten kinetics as a valuable means of visualizing the dynamics of phytoplankton growth. However, phytoplankton growth in both natural and experimental settings is likely more complicated than described by Monod or Michaelis-Menten uptake kinetics or in the logistic growth equation, and it seems premature to use these theories to challenge a large body of experimental evidence or to imply that the process of eutrophication will fit easily into this particular theoretical framework.

I think that both comments disregard my main point, which I believe is valid, that Liebig thought of nutrient limitation in terms of enhanced yield, and that many experiments in aquatic situations, which demonstrate only an enhancement of phytoplankton yield without showing a change in the growth rate of the population, are not always valid in determining a real limiting factor in the natural system.

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Reference

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14 April 1973

Project Sanguine?

With regard to the article on the Navy's Project Sanguine by Wait (*1*), some corrections and redirections of emphasis could be suggested. To begin at the beginning, Project Sanguine was first announced to the public, not in May of 1971 by Wisconsin's Senator Gaylord Nelson, but in the fall of 1968 by former Congressman O'Konski. Before this announcement Senator Nelson was unaware of Sanguine although it had been under consideration by the Navy since the late 1950's.

Wait's reference 12 from the *Congressional Record* (*2*) includes a carefully reviewed report on technical feasibility which was released to the public on 3 May 1971 by the Wisconsin Committee for Environmental Information (WCEI), a branch of Scientists' Institute for Public Information, and contains the first public estimate of the time a Sanguine system would require to transmit a single "bit" of information. This estimate was an almost incredible 100 seconds per bit, which led to the conclusion that (*3*) "on the grounds that it either requires an unrealistic amount of power or is an extremely slow system of communication, and that these features lead to its susceptibility to jamming, the Sanguine system must be regarded as technically infeasible." Whether this conclusion is "strongly worded," as Wait suggests, is, I suppose, a matter of taste. Since that time scientists at Lincoln Laboratory (*4*) have confirmed the WCEI bit time estimate, but claim it can be reduced by a factor of 100 by "clipping" the atmospheric noise peaks (associated with nearby lightning strokes) before detection. The idea is to reduce bit time by making the receiver a factor of 100 more sensitive. However, this also makes the receiver a factor of 100 more sensitive to jamming noise (which would not be reduced by clipping) and has no effect on the ratio of the cost of a Sanguine system to the cost of jamming. And this ratio, I submit, is the central issue in the discussion of technical feasibility.

Wait takes Sanguine critics to task for using an antenna efficiency formula which assumes radiation into an infinite half-space. He points out that assuming radiation into a sharply bounded ionospheric wave guide leads to a factor of 100 increase in calculated radiated power. It is generally agreed, however, that the ionosphere is not sharply bounded (*5*); and the zonal harmonic calculations by Johler and Lewis (*5, 6*), which take the true graduated nature of the ionospheric boundary into account, indicate a reduction in radiated power by a factor of 100. Further research may show that an infinite half-space approximation is more correct. It is to be hoped that the Navy will soon find funds to support a continued study by Johler and Lewis.

How then, one might ask, does the Navy manage to obtain even rudimentary agreement between calculated and observed field strengths? The answer may be that these propagation tests have been carried out only with aboveground transmitting antennas, even though the central component of Sanguine would be a buried transmitting antenna covering several thousand square miles. But, as C. W. Harrison has pointed out (*7*), the theory of the relative efficiencies of aboveground and buried antennas is not at all clear. At Harrison's urging the Navy has agreed to conduct some simple tests on this vital question during fiscal year 1973 (*8*).

But the main technical issue is still the cost of a Sanguine signaling system relative to the cost of jamming. In estimating the eventual cost of Sanguine the public must begin with the Navy's current estimate of about $750 million (*9*). This does not include the probable cost overruns associated with many technical uncertainties, including those mentioned above (*10*). Representatives of the Navy state that a jamming system "would require an investment cost several times larger than the investment cost of Sanguine" (*8*). But since the purpose of Sanguine is to send a "last strike" signal to the nuclear submarine fleet after a preemptory nuclear attack by another nation, it must be assumed that the other nation would know when to jam. Thus, the jammer could be primarily a conventional power generation and distribution system with modifications to permit auxiliary jamming duty for a few hours in the event a preemptive strike were to be attempted (*9*).

It appears the Navy's assertion is based on the assumption that a jamming system could have no other economic value.

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