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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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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Scott has brought up some points in his discussion of my review on Project Sanguine that do not alter any of my views. In giving some of the history of the Sanguine project controversy, I decided to refer only to documentation that is available to a diligent library user. Also, I avoided any mention of unpublished materials and oral statements.

As I had indicated before, there are a number of unresolved technical issues concerning the excitation, radiation, and propagation of the extremely-low-frequency signals from the test transmitter. I urge interested readers to examine the published papers that were presented at the symposium held in Newport, Rhode Island, on 13 September 1972 (*1*). In particular, the papers by the Lincoln Laboratory group answer may of the earlier criticisms that are alluded to by Scott.

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Nerve Growth Factor versus Insulin

The structural similarities between nerve growth factor (NGF) and insulin, as presented by Frazier *et al.* (*1*) are striking, but their comparison of the organs of origin—pancreas and salivary gland—deserves further discussion. Frazier *et al.* state that the phylogenetic appearance of salivary glands "parallels or slightly precedes that of NGF." This is contradicted by reports of the presence of NGF in fishes and amphibians (*2, 3*) although fishes, as stated by Frazier *et al.* lack salivary glands of the type present in higher vertebrates. Teleost fish spinal and sympathetic ganglia can nevertheless respond to mouse NGF by hypertrophy and hyperplasia, in a manner somewhat similar to that of higher vertebrates (*4*). The NGF found in the axial region of fish has been furthermore reported to be immunologically similar to mouse submaxillary gland NGF (*2*).

The emphasis on the submaxillary gland as the site of NGF production in higher vertebrates is likewise not relevant if one considers the time period in which NGF is functional. The embryonic nervous system is responsive to NGF before the development of NGF secretion by the salivary glands. The high levels of NGF in salivary glands occur only after puberty in the mouse, a time when the spinal and sympathetic neurons are no longer responsive to it. Thus this high level is not of developmental significance. However, NGF can be detected in developing vertebrate embryos at a time when the nervous system is responsive to it. At this time it can be detected in the axial region (*2, 5*), the same site in which it is found in fish. Whether or not this is a site of synthesis is unknown; nevertheless the submaxillary gland is clearly not the only site of NGF production. Alternative sites, such as the axial region, deserve further investigation.

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It has been pointed out by Weis and Weis (*1*) that the parallel in the appearance of nerve growth factor (NGF) and the phylogenetic appearance of salivary glands recently suggested (*2*) is contradicted by reports of the presence of NGF in teleosts and amphibians (*3, 4*). The identification of NGF in fish and amphibians, which is immunologically similar to mouse NGF (*4*), is indeed compelling evidence that NGF appeared as a molecular entity prior to the development of mammalian salivary glands. However, this in no way alters the essential validity of the evidence that relates NGF and insulin and suggests that these proteins are a result of parallel evolutionary development along plausible lines from an ancestral protein. The lack of NGF in elasmobranchs (*4*) may well mark the last evolutionary branch point before the appearance of NGF.

With regard to the submaxillary gland as a site of synthesis of NGF, Levi-Montalcini and Angeletti have suggested that NGF may be produced in other tissues (*5*), and many lines of evidence now support this idea (*6, 7*). The fact remains, however, that the mouse submaxillary gland is the only established site of synthesis (*8*) and therefore the only organ of NGF production that can be discussed meaningfully at present. We are quite aware that the relevance of the submaxillary gland to the developmental role of NGF is debatable; however the often overlooked maintenance function of NGF (*9*) in the postpubertal organism should be remembered, especially in view of the demonstration by Hendry (*7*) that submaxillary gland NGF comprises a significant proportion of the serum NGF in adult mice. The fact that synthesis does occur in the submaxillary gland renders germane the comparison of this organ to the pancreas, the organ of insulin synthesis (*2*). The suggestion that NGF may be produced in the "axial region" (*1*) certainly merits consideration in view of our present ignorance concerning the site of NGF synthesis in early development. However, the presence of measurable concentrations of NGF does not necessarily mark a site of synthesis.

The real utility of the hypothesis that NGF and insulin are structurally, functionally, and evolutionarily related proteins (*2*) has proved to be in the many lines of experimentation which this observation has stimulated. Detailed conformational and topographical chemical modification studies have extended the structural comparisons of NGF and insulin to include secondary and tertiary structure (*10*). An investigation of the possible role of cyclic adenosine monophosphate (AMP) in the NGF response (*11*) has revealed that NGF, like insulin (*12*), does not appear to employ cyclic AMP as a