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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 Also, I avoided any mention of unpublished materials and oral statements.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

second messenger (*13*). Furthermore, studies with insoluble NGF derivatives and ^{125}I-labeled NGF indicate that, like insulin (*14*), NGF exerts its action on responsive neurons by first combining with a surface membrane receptor (*15*) and that the properties of this interaction are quite similar to those of insulin (*16*).

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Odor-Following and Anemotaxis

In the study of Farkas and Shorey (*1*), male pink bollworm moths, *Pectinophora gossypiella*, flew through a "response" plane near the source of an odor plume of female sex pheromone. In both still air and moving air large majorities of those moths which crossed the response plane flew within the odor plume toward its source. Farkas and Shorey concluded that (i) these moths can stay within an odor plume in the absence of an air current, (ii) these moths can sense the direction of the source of an odor plume in the absence of an air current, and (iii) for these moths anemotaxis (orientation to an air current) is not necessary for locating the source of an airborne odor. The results of Farkas and Shorey support the first but not the last two conclusions.

In the still-air trials, replicates were "abandoned" in which moths did not cross the response plane within 20 seconds. Thus, moths which remained stationary in the odor plume short of the response plane would not be counted. Also not counted would be moths flying in the plume but turning away from its source. In short, data which might have supported the need for anemotaxis were discounted. Furthermore, since moths were released at the end of the odor plume away from the source, they were not given an equal opportunity to fly in the "wrong" direction. In a critical test, one would release moths at the longitudinal midpoint of the odor plume in both still air and moving air and compare the percentages (of all moths released) moving toward or away from the source of the plume. A nonsignificant difference would support the last two conclusions.

The results of Farkas and Shorey thus show that moths can follow an airborne odor trail in still air. However, their results cannot be interpreted as a rejection of the generally held hypothesis that animals must orient to an air current (anemotaxis) to find the source of an airborne odor.

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In our report (*1*) we asserted that the mechanism by which pink bollworm male moths steer toward a source of female sex pheromone does not require a sensing of wind direction. Grubb has raised two questions with regard to our tabulation of data and our experimental procedure that could cast doubt on the validity of this assertion.

First, he pointed out that we abandoned those replicates in the still-air trials in which the moths did not cross the response plane (the cross-sectional area of the tunnel located 30 cm "downwind" from the pheromone source) within the arbitrary 20-second interval during which we considered the plume still remained intact. Thus, moths which remained stationary in the odor plume short of the plane and moths turning away from the odor source would not be counted. We regret that we omitted the following important information. In each of 27 replicates conducted with a plume suspended in still air, a single male moth left the release cage and entered the plume. Seven of these moths either left the plume or remained in the plume but did not pass the response plane in the 20-second interval. The remaining 20 moths proceeded about 1.5 m from the release cage and passed through the response plane. Sixteen of these remained within the central portion of the flight tunnel occupied by the pheromone plume and four flew outside this area. Thus, approximately 60 percent of the moths that initially entered the plume exhibited odor trail-following over a short (1.5 m) distance.

We agree with Grubb's second point. Our experiment did not conclusively demonstrate that the odor trail possessed an inherent polarization, indicating to the moths the direction along the axis of the trail toward the odor source. Although we could not detect an anemotactic reaction, we did not perform the critical experiment that would allow us to say without doubt that such a reaction is not needed to provide the directional cues to the trail.

We did not intend to disclaim the existence of anemotaxis as one of the mechanisms that may be used by some species of insects during their in-flight orientation to a distant odor source. However, we continue to question the universal application of this phenomenon, which is generally accepted as a truism although it has received almost no experimental validation, to all cases of olfactory orientation. Our demonstration that pink bollworm moths follow an airborne odor trail in still air, even if the trial is not polarized, provides an additional mechanism for aerial approach to an odor source.

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