variables are incomplete. However, the constants of apparent generation time and terminal concentration appear to be equivalent to that observed with the virulent V1b strain, although it is noted that most of the animals responding to the 30R challenge were those challenged with virulence-enhanced spores.

Thus it is concluded that the growth rate in the septicemic stage is not affected by specific host resistance, by dose level (or, alternately, time of response), or by enhancement of virulence by treatment of spores with egg yolk, a treatment that in these tests gave a 120-fold increased virulence for guinea pigs. We conclude, also, that immunization significantly lowers the number of cells present in the blood stream at death when normal and immunized guinea pigs are compared, and that the disease caused by a strain of low virulence, once it is established as septicemic anthrax, is or may be equally as severe and dangerous as that caused by the highly virulent strain.

There is no evidence from these data that the host defenses are overpowered or change during the period of septicemic growth—otherwise the response slopes would not be linear. Growth, as indicated by increased cell number during this final stage of the disease, may be compared favorably with the growth rate in the logarithmic growth phase of culture in vitro in media such as tryptose or nutrient broth. Since the terminal concentration of bacilli per milliliter of blood is significantly lower in immunized animals than in nonimmunized hosts, it follows that, if these data are translatable to other animals, treatment will need to be initiated while the number of bacilli per milliliter of blood is much lower in the immunized than in the nonimmunized hosts. The critical level of organisms per milliliter of blood in the guinea pigs was reported by Keppie *et al.* (*2*) to be 1/300th of the terminal concentration, and after this no effective antibiotic cure can be predicted.

Since death by anthrax has been ascribed to a toxemia (*6*), and since toxin has been detected both in vivo and in vitro, our observations raise questions regarding the critical number of organisms per milliliter of blood in relationship to toxin produced in immunized as compared to normal animals. It is possible, since immunization prolongs the time from dose to septicemia, that more organisms and more toxin are produced before septicemia is detected, and therefore the animal dies with a lower level of organisms per milliliter of blood.

FREDERICK KLEIN
BILL G. MAHLANDT
RALPH E. LINCOLN
IRA A. DEARMON, JR.
ALBERT L. FERNELIUS
U.S. Army Chemical Corps, Fort Detrick, Frederick, Maryland

References

1. W. L. Bloom, W. J. McGhee, W. J. Cromartie, D. W. Watson, *J. Infectious Diseases* **80**, 137 (1947).
2. J. Keppie, H. Smith, P. W. Harris-Smith, *Brit. J. Exptl. Pathol.* **36**, 315 (1955).
3. C. B. Thorne and F. C. Belton, *J. Gen. Microbiol.* **17**, 505 (1957).
4. M. Kaga, *Nippon Saikingaku Zasshi* **11**, 470 (1956).
5. N. G. Roth, I. A. DeArmon, Jr., D. H. Lively, *J. Bacteriol.* **72**, 666 (1956).
6. H. Smith, J. Keppie, J. L. Stanley, *Brit. J. Exptl. Pathol.* **36**, 460 (1955).

9 August 1960

Growth of Sporangiophores of Phycomyces Immersed in Water

Abstract. Sporangiophores of *Phycomyces* are capable of sustained growth when immersed in aerated water, and under these conditions they are negatively phototropic.

When immersed in water saturated with air at atmospheric pressure, sporangiophores will grow for many hours at the rate of 1.5 mm/hr. This rate is equal to one-half the growth rate in air (*1*). The reduction of the growth rate is not due to lack of oxygen in the ambient water, because increasing the oxygen tension in the water by (i) saturating the water with pure oxygen or by (ii) saturating the water at 0°C with pure oxygen, warming slowly to 22°C under 4 atm pressure of pure oxygen, and maintaining this increased pressure throughout the test has no effect on the growth rate. In air, if the oxygen tension is raised above 20 percent, there is no change in the growth rate of the plant (*2*). It appears then, that when the rate of oxygen supply is not limiting, 1.5 mm/hr is the growth rate in water, in contrast to 3.0 mm/hr in air. In either case, there is probably no significant oxygen gradient at the cell wall.

I have attempted to measure the oxygen consumption of the growing zone by Warburg manometric techniques, but have succeeded only in establishing an upper limit of about 0.5 mm^3/hr.

Specimens growing in water exhibit *negative* phototropic reactions. This may seem surprising in view of the fact that the refractive index of water (1.33) is slightly below that of the cell contents (1.35), and Buder's theory (*3*) would therefore predict a positive reaction. However, it may be presumed that internal absorption, estimated to be 10 percent by Delbrück and Shropshire (*4*), is sufficient to overcompensate the slight residual lens effect.

The possibility of growing specimens in aerated water opens the way to testing the effects of any water-soluble substrates or inhibitors on growth and reactivity. I have tested a number of enzymatic inhibitors but have not found any that would discriminate between growth and photoreactivity (*5*).

RICHARD BOND STIFLER*
California Institute of Technology, Pasadena

References and Notes

1. H. Gruen, *Plant Physiol.* **34**, 158 (1959).
2. C. Hamilton, personal communication.
3. J. Buder, *Ber. deut. botan. Ges.* **36**, 104 (1918).
4. M. Delbrück and W. Shropshire, Jr., *Plant Physiol.* **35**, 194 (1960).
5. I would like to thank Dr. Max Delbrück for suggesting this work, and for subsequent discussions pertaining to it. The work was sponsored by a National Science Foundation Undergraduate Research Grant.

* Present address: Department of Biology, Massachusetts Institute of Technology, Cambridge.

12 October 1960