12. A. S. Hafleigh and C. A. Williams, Jr., *Science* **151**, 1530 (1966).
13. Zuckerkandl and Pauling have pointed out the possibility of using proteins as evolutionary clocks (see *14*).
14. E. Zuckerkandl and L. Pauling, in *Horizons in Biochemistry*, M. Kasha and B. Pullman, Eds. (Academic Press, New York, 1962), p. 189; in *Evolving Genes and Proteins*, V. Bryson and H. J. Vogel, Eds. (Academic Press, New York, 1965), p. 97; R. F. Doolittle and B. Blömback, *Nature* **202**, 147 1964); E. Margoliash and A. Schejter, *Advan. Protein Chem.* **21**, 113 (1966); J. Buettner-Janusch and R. L. Hill, *Science* **147**, 836 (1965); L. F. Smith, *Amer. J. Med.* **40**, 662 (1966); J. Derancourt, A. S. Lebor, E. Zuckerkandl, *Bull. Soc. Chim. Biol.* **49**, 577 (1967).
15. A. C. Wilson, N. O. Kaplan, L. Levine, A. Pesce, M. Reichlin, W. S. Allison, *Fed. Proc.* **23**, 1257 (1964).
16. M. Reichlin, M. Hay, L. Levine, *Immunochemistry* **1**, 21 (1964); M. Reichlin *et al.*, *J. Mol. Biol.* **17**, 18 (1966).
17. M. Reichlin, personal communication; E. Margoliash, M. Reichlin. A. Nisonoff, in *Conformation of Biopolymers*, G. N. Ramachandran. Ed. (Academic Press, New York, 1967), vol. 1, p. 253.
18. C. A. Leone, Ed., *Taxonomic Biochemistry and Serology* (Ronald Press, New York, 1964); C. A. Williams, Jr., in *Evolutionary and Genetic Biology of Primates*, J. Buettner-Janusch, Ed. (Academic Press, New York, 1964), vol. 2, p. 25.
19. S. N. Salthe and N. O. Kaplan, *Evolution* **20**, 617 (1966).
20. V. M. Sarich, in *Human Evolution 1968*, S. L. Washburn, Ed. (Holt, Rinehart, Winston, New York, in press).
21. This date will probably appear relatively recent to many. There is, however, certainly no fossil material to contradict it. The first fossil Old World monkeys (*Mesopithecus, Prohylobates*) are found in the middle-to-early Miocene of Africa [E. L. Simons, in *Evolutionary and Genetic Biology of Primates*, J. Buettner-Janusch, Ed. (Academic Press, New York, 1963), vol. 1, pp. 65–129; *Nature* **205**, 135 (1965)]. On the other hand, it can fairly reliably be assumed that all living primates derive from a common ancestral form living no earlier than the late Cretaceous. At least one of the living prosimians, *Tarsius*, represents a lineage already distinct in the early Eocene (see Simons). As there is no reason to believe that any other prosimian group shows closer relationship to the higher primates, these suborders (Anthropoidea and Prosimii) were already distinct no less than 50 and no more than approximately 70 million years ago. Although no upper limit on the time of origin of a separate Old World monkey line is available, there would probably be general agreement with the suggestion, which is entirely consistent with the immunological evidence, that appreciable periods of common ancestry characterize both the Catarrhini (apes, man, Old World monkeys) after their separation from the ancestors of the New World monkeys and the living Anthropoidea after their separation from the Prosimii. To contain these periods within the 50-to-70 million year limits set above would seem to require that a reasonable upper limit on the time of divergence of apes and Old World monkeys be set at 30 to 45 million years. We feel that the lower end of this time scale is more probable. If this were not so, the relationship between ID and time of divergence for primates as a whole would be extremely complex. We feel that on immunological grounds this is unlikely (*20*).
22. B. M. Funnell, *Quart. J. Geol. Soc. London* **120s**, 179 (1964).
23. J. R. Napier and R. P. Davis, *Fossil Mammals of Africa, No. 16* (British Museum Natural History, London, 1959); W. E. LeGros Clark, *Fossil Evidence for Human Evolution* (Univ. of Chicago Press, Chicago, ed. 2, 1964); F. Ankel, *Folia Primatol.* **3**, 263 (1965); S. L. Washburn, *Classification and Human Evolution* (Aldine, Chicago, 1963), pp. 190–203.
24. The availability of antiserums to *Pan* and *Hylobates* albumins allows a partial answer to a caveat that might be made against these conclusions. It could be argued that the similarities among, for example, hominoid albumins are a result of parallel evolution and not recent common ancestry. That is, immunologically similar albumin structures have evolved in genetically separated lineages. It can be seen that this would, in the case of apes and man. require a most improbable set of coincidences, for the ID's of other apes and man obtained with antiserums to gibbon albumin are very nearly equal (obviously except for the siamang), indicating these albumins have changed to much the same degree since their separation from the gibbon; yet the antiserums to human and chimpanzee albumins show that human, chimpanzee, gorilla, and orang albumins are quite different from one another. To support the idea of parallelism, then, one would have to postulate that gibbon albumin evolved in parallel to those of the other apes until the radiation of these lineages began, and then diverged from all of these (clearly gibbon albumin cannot have evolved in parallel to all four nonhylobatid lineages at the same time). We see that to reconcile the immunological data with many of the current views concerning hominoid evolution (*3*) we are forced to postulate a remarkable series of coincidences. In the same way those who would attribute the morphological similarities in the trunk and upper limbs, among all the living apes and man, to parallelism must make a similar appeal to coincidence.
25. Supported by a PHS predoctoral fellowship to V.M.S. (1-F1-GM-30,454-01) and an NSF grant to A.C.W. (GB-6420). Portions of this work have been reported at meetings of the American Association of Physical Anthropologists (8 April 1966 and 24 April 1967) and to the AAAS (30 December 1965). We thank N. Arnheim, J. Gerhart, T. Jukes, G. Sensabaugh, and S. L. Washburn for helpful discussion.

26 September 1967

Visceral Tissue Vascularization: An Adaptive Response to High Temperature

Abstract. Electrical heat sources implanted in the abdominal cavities of sheep were heated to give initial temperatures of 42° and 45°C at the surfaces of the heaters. During 18 days of constant heating, a vascularized connective-tissue envelope encapsulated the heat sources, and the temperatures at the surfaces of the heaters declined 0.8° and 1.8°C, respectively. The degree of vascularization and the magnitude of the decrease in the surface temperature appeared to be related to the proximity of the tissue's initial temperature to 45°C, a temperature ordinarily considered detrimental to cell structure. The vascularization thus appears to be adaptive.

While studying the effects of additional endogenous heat in animals, we implanted aseptic, electric heat sources covered with medical-grade silicone rubber in the abdominal cavities of sheep. Two of these devices (5.1 cm wide, 15.1 cm long, and 0.64 cm thick) were implanted end to end in the dorsal abdominal cavity and extended from the renal artery caudally to the aortic bifurcation. After recovery of the animals, voltage (direct-current) was applied to each heater through insulated wire leads from an external electronic circuit capable of sensing the heater-coil temperature and maintaining it within $\pm$ 0.05°C. Another ewe with identical implants to which no heat was applied served as a control.

The temperature at the surface of the heater was calculated from the measured temperature of the heater coil and a predetermined conductance factor for the silicone rubber covering each heater.

Power input was continuously recorded, and calculated temperatures at the heater surfaces were tabulated. Data from one of these experiments are plotted in Fig. 1. The two strip heaters were designated anterior and posterior, according to their positions relative to each other in the dorsal abdominal cavity. The initial surface temperature of the anterior heater (T_{sa}) was 45°C; that of the posterior heater (T_{sp}) was 42°C. Both temperatures were in the noxious range (*1*). As heating continued, a decline in both temperatures occurred in spite of the increase in power to each heater required to keep heater-coil temperatures constant.

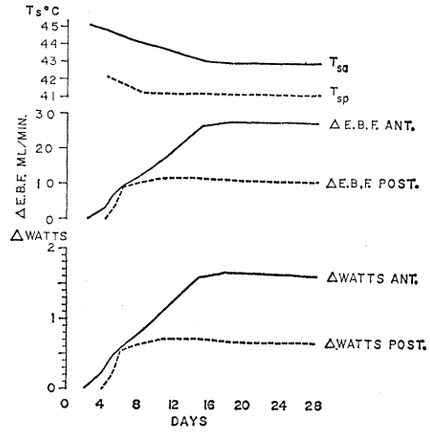

Fig. 1. Differential heating of anterior and posterior dorsal abdominal heat-exchangers in a sheep. T_{sa}, Surface temperature of anterior heater; T_{sp}, surface temperature of posterior heater; $\Delta watts$, increase in power necessary to maintain the heater coil at a constant temperature; $\Delta E.B.F.$, increase in effective blood flow equivalent to increase in power (0.85 assumed to the specific heat of blood).

After 18 days of heating, T_{sa} had declined almost 2°C, and power input had steadily climbed to approximately 35 percent above its original level. On the other hand, T_{sp} had declined only 0.8°C, and power input had increased only 18 percent over the original level. Thus an initial surface temperature of 45°C appeared to promote a much greater response than the stimulus of 42°C did.

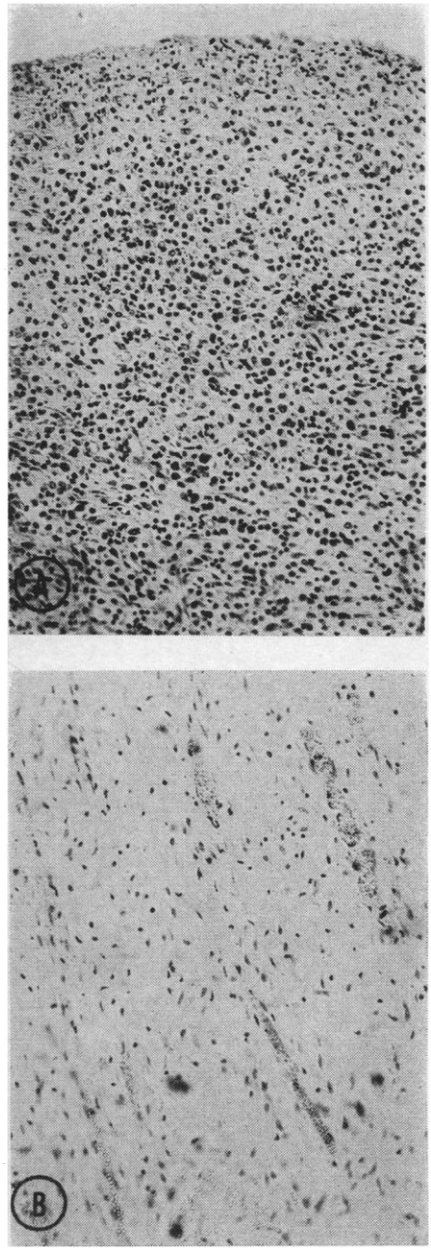

Fig. 2. Histological differences between encapsulating tissue formed at body temperature (A) and that formed in the presence of additional heat (B). (A) Densely packed nuclei in relatively avascular connective tissue. (B) Less-dense fibrous sheath penetrated by numerous vascular channels containing erythrocytes (× 126; hematoxylin and eosin).

The phenomenon observed could result from increased local convective heat transfer by the blood, caused by increased cardiac output, or from increased local effective blood flow, or both. Since it is unlikely that the former underwent such a significant increase, the thermal data suggest a significant change in local vascularization.

At autopsy on the 29th day, a gross histological survey revealed enlarged venous channels radiating from the sites of implantation. The heaters were encased in highly vascularized connective-tissue envelopes that appeared deep red. The tissue around the anterior heater was darker red and appeared more vascular than that around the cooler, posterior heater.

At 60 days, the unheated heaters in the control were found encapsulated in tough, fibrous, relatively avascular tissue like that usually associated with implanted prosthetic devices. This tissue, unlike that around the heated implants, was pale yellow. This finding supports the suggestion that the vascularization of the encapsulating tissue was a result of the heat stimulus. Histological examination of the tissues revealed a marked vascularization of capsular tissue around the heated implant compared to the relative avascularity of that around the unheated one (Fig. 2).

The findings suggest that, in visceral tissue, temperatures high enough to be ordinarily considered damaging to cell function can be used to stimulate vascularization, and at the same time to increase effective local blood flow. Such a response occurs in heated skin of humans (*2*).

The response of visceral tissue in our experiment points up the curious phenomenon that tissue within the body cavity, which is never subjected to noxious temperatures under natural circumstances, exhibits adaptive, compensatory capabilities against such high temperatures.

ROBERT O. RAWSON
JAMES D. HARDY
John B. Pierce Foundation of Connecticut, New Haven
KENT A. VASKO
Division of Animal Care, Yale University, New Haven, Connecticut

References and Notes

1. J. D. Hardy and J. A. J. Stolwijk, in *Touch, Heat and Pain*, A. V. S. de Reuck and J. Knight, Eds. (Churchill, London, 1966), pp. 27–50.
2. M. Lipkin and J. D. Hardy, *J. Appl. Physiol.* **7**, 212 (1954).
3. Research performed pursuant to PHS contract PH 43-66-978.

5 September 1967

New High-Temperature Chlorella

Abstract. *Growth characteristics of* Chlorella *1-9-30, a new strain of green, high-temperature algae, are compared with those of the widely used* Chlorella *7-11-05. Under comparable conditions,* Chlorella *1-9-30 has the same temperature range for growth as has* Chlorella *7-11-05, but it generally has a higher growth rate. It also differs from* Chlorella *7-11-05 morphologically. Because of its high capacity for organic synthesis,* Chlorella *1-9-30 may be useful in biochemical, biophysical, and physiological research.*

Strains of the green, high-temperature algae were first isolated in 1951 and introduced in 1953. One of those strains, *Chlorella* 7-11-05 (*1*), is now extensively used in physiological and biochemical research. In studies of the adaptation of unicellular algae to mass production, this strain has also been used as a source of organic material for food and for industrial purposes or as a photosynthetic gas-exchanger in closed ecological systems. The growth characteristics of *Chlorella* 7-11-05 have been reported (*1–4*).

I now report on the growth characteristics of another strain of green, high-temperature alga, *Chlorella* 1-9-30, which seems to be superior in its capacity for organic synthesis. *Chlorella* 7-11-05 was isolated from a sample collected in 1951 from Waller Creek on the campus of the University of Texas in Austin, and *Chlorella* 1-9-30 was isolated from another sample collected from the same stream in 1954. The techniques for obtaining the growth characteristics of these two strains have been described (*2*). The capacity for growth is depicted for *Chlorella* 1-9-30 in Fig. 1.

Temperature range (limits) for continuous growth is practically the same for the two strains. Optimum temperature for growth is 38° to 39°C, maximum temperature is about 42°C, and minimum temperature (depending on light intensity) is somewhere between 15° and 20°C. Minimum temperature suitable for growth extends generally to lower temperatures at lower light intensities; that is, the capacity to grow at lower temperatures is limited in the high-temperature strains by increase in light intensity (*5*).

Under comparable conditions, growth rate for *Chlorella* 1-9-30 is higher than for *Chlorella* 7-11-05. Thus, at 38°C