46.7 percent compared to 1/9 or 11.1 percent in lizards held at 40° and 42°C). There were no significant differences between groups in the incidence of skin lesions at the injection site (necrosis, ulcers, or gangrene).

The increased survival of infected animals with elevated body temperature supports the hypothesis that fever following a bacterial infection (*8, 9*) is beneficial to the host. Inasmuch as the in vitro bacterial growth rate was stable between 34° and 40°C, the increased survival of the lizards at 40°C could be attributed to an enhancement of the host's defense mechanisms at the elevated temperature. The specific aspects of the defense mechanisms that might improve with increasing temperature remain unknown. It is possible that several components of the defense mechanisms, including phagocytic index, phagocyte bactericidal activity, leukocyte mobilization, and humoral mediators of inflammation, are temperature-dependent. It is also possible that the toxigenicity of the bacteria decreased as the temperature increased. At 42°C, the decreased bacterial growth rate probably also contributed to the increased survival. Conversely, the reduction in temperature below normal levels (such as to 34°C) following bacterial infection led to increased mortality, possibly due to impaired host defense. Our interpretation of these data is shown in Fig. 2.

At the highest temperature tested, the pattern of deaths was similar for the controls and the infected lizards. Whereas most deaths occurred within 3.5 days in infected lizards maintained at 34° to 40°C, essentially all deaths at 42°C occurred after 3.5 days. Apparently, maintenance at 42°C for a period exceeding 3.5 days is harmful in itself. This suggests that the deaths at 42°C were not due to the bacterial infection but to some undetermined adverse effect of long-term elevation in temperature.

We believe these data may have relevance for mammals. Assuming a common phylogenetic origin of fever in present-day mammals and reptiles, there is reason to expect that the function of fever is similar in these two vertebrate classes. That is, if fever evolved in reptiles as a mechanism to decrease mortality and morbidity following infection, its function should be similar in mammals. If fever in response to infection is beneficial in mammals, then the widespread use of antipyretics to lower the temperature of people with moderate fevers should be reevaluated (*11*).

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11. Clinical observations suggest that an elevation in temperature, following bacterial infection, is beneficial to humans. In certain clinical situations the use of antipyretics is contraindicated. For example, bacterial infections often develop in patients with third-degree burns. As destruction of epithelial layers leads to an increased evaporative heat loss, these patients often encounter thermoregulatory difficulties. The prognosis for survival is considered better in patients developing a fever of 1° to 2°C than in patients who remain at the normal or afebrile body temperature (I. Feller, personal communication).
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HeLa Cells and RT4 Cells

In a note added in proof to their report on cellular contamination in tissue culture, Nelson-Rees *et al.* (*1*) reported that cells of culture line RT4 derived from a human bladder tumor (*2*) had features which suggested that the cells were HeLa cells. We have reexamined the RT4 cell stock maintained in our laboratories. These cells have a modal chromosome number of 47 and glucose-6-phosphate dehydrogenase, type B; they also differ from HeLa cells at three other loci tested, namely, phosphoglucomutase 1, phosphoglucomutase 3, and esterase D. The parent stock of RT4 cells therefore is not HeLa cells (*3*). It should be pointed out that the cells examined by Nelson-Rees *et al.* had been maintained in other laboratories for some years and were not provided directly from our parent stock.

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3. We thank the staff of the MRC Human Biochemical Genetics Unit, Galton Laboratory, University College, London (Dr. S. Povey), and Dr. C. W. Parr, London Hospital Medical School, for the iscenzyme studies.

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The comment by Franks and Rigby emphasizes two points: (i) It is relatively easy to check specificity of cells and (ii) distributing cells without characterizing them whether or not the distributor is the originator is as wrong as finding out that cells are contaminated and not announcing where they came from.

In our report in *Science*, courtesy led us to keep the sources of the cell cultures anonymous, although we had originally cited them when communicating with all parties concerned prior to submitting the manuscript.

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