not improve conditions or standards. Rather, they will increase the costs and regulatory burden of researchers and their institutions by introducing redundant or inconsistent regulations, all the way from the supplier to the end user—from the cost of animals, to training of lab personnel, to procedures of animal care committees. Representatives of animal supply companies (Taconic and Harlan) concurred that the added regulatory costs would be borne by the research community.

We fully support the humane care and treatment of laboratory animals. We strongly oppose efforts to achieve this by means of duplicative and expensive new regulations.

A *Science* editorial about animal rights (P. M. Conn and J. Parker, *Science*'s Compass, 20 Nov., p. 1417) urged that "[s]cientists need to respond forcefully to animal rights advocates, whose arguments are confusing the public and thereby threatening advances in health knowledge and care." The biomedical research community can begin by making its voice heard in response to this issue. Comments can be submitted until 28 May to the USDA's Web site at http://comments.aphis.usda.gov.

AAA Public Affairs Committee (Joseph C. Besharse, Bruce M. Carlson, Donald P. Jenkins, David S. Lester, James L. Olds, Peter Satir)
American Association of Anatomists/Federation of American Societies for Experimental Biology, 9650 Rockville Pike, Bethesda, MD, 20814–3998, USA

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Field Primatology and Biomedical Research

The article by Jon Cohen (News of the Week, 5 Feb., p. 772) reporting that human immunodeficiency virus–type 1 (HIV-1) probably originated in central African chimpanzees has prompted calls for collaboration between biomedical scientists and field-workers to investigate the natural epidemiology of retroviruses in nonhuman primates. As field primatologists, we find this development gratifying. Our own studies entail repeated capture, sampling, and release, as well as observation, of African green monkeys (vervets and grivets: *Cercopithecus aethiops*) and baboons (*Papio hamadryas*). Materials drawn from these populations [naturally infected with simian immunodeficiency virus (SIV)] and shared with AIDS researchers have documented the rare occurrence of SIV in Tanzanian yellow baboons in the wild (*1*) and cross-species transmission of SIV between vervets and baboons (*2*) and have confirmed horizontal transmission as the primary mechanism for SIV spread in wild Ethiopian green monkeys (*3, 4*).

Our studies of Ethiopian baboons and

Chimpanzee retroviral ecology may provide a key to the origin of AIDS.

grivet monkeys began in 1973. Typically, in a field season we capture animals, take blood, collect morphometric data, determine the age of the animals from dental casts, and record reproductive features. Thus, we have been able to show that female grivets become SIV seropositive before they are adult, while males become SIV positive only when they are fully grown. Recapture of individually recognized animals has allowed us to document instances of seroconversion.

For chimpanzee retroviral epidemiology to be similarly understood, it will be necessary to sample (preferably without capture) a population that has been subject to the long-term observation that enables individual animals to be recognized and their life histories charted. Such information requires laborious accumulation in often dangerous, and generally underfunded, long-term field programs. Perhaps the research needed to understand the zoonotic origins of HIV could accomplish the remarkable: benefit the primate species, the discipline of primatology, and biomedical research.

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Fluorescent Lamps in Photocopiers

Meher Antia (News of the Week, 29 Jan., p. 617) comments that the startup lag in mercury-containing fluorescent tubes keeps them from being used as brake lights for cars and in fax and photocopy machines. Actually, fluorescent exposure

CREDIT: DANU/JESKE/ANIMALS ANIMALS

CREDIT: BILL BEATTY/VISUALS UNLIMITED

Does feeding cattle hay before slaughter help prevent *E. coli* contamination of beef?

would actually increase contamination with *E. coli* O157:H7. Also, the 1000-fold reductions in total fecal *E. coli* demonstrated by Diez-Gonzales *et al.* are far greater than those recorded in cattle experiencing similar ration changes (*8*). Finally, extensive surveys show that grain-fed feedlot cattle have no higher *E. coli* O157:H7 infection prevalence than similarly aged dairy cattle fed forage (hay) diets (*9, 10*).

Abrupt feed change immediately before slaughter could have unexpected deleterious effects. The proposed diet change has the potential to increase the risk of bovine salmonella infections. The dietary change results in sharply reduced volatile fatty acid concentrations in the large intestine that would be coupled with expected intake reductions of dry matter and gastrointestinal flora disturbances that are known risk factors for such infections (*11–15*).

In advocating the adoption of a major change in cattle husbandry without direct evidence as to its effect on the incidence of cattle or human infections with *E. coli* O157:H7, Diez-Gonzalez *et al.* have drawn a conclusion beyond that supported by their data.

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Response

Hancock *et al.* omit the sentence preceding their quote of our conclusion: "Grain feeding is a practice that promotes the production and efficiency of cattle production, and it is unlikely that American cattle will ever be fed diets consisting only of hay."

Hancock *et al.*'s reference 1 indicates that "when cattle are presented for slaughter they will, at times, carry food poisoning organisms either internally (in the intestinal tract) or externally. Some of these organisms will be transferred to carcass and offal meats." Cattle are held in pens before slaughter. The cattle defecate, and they often have *fresh* manure on their

lamps have been used in photocopy machines for many years. The first xerographic office copier, the Xerox 914, introduced in 1959, used fluorescent lamps. Rapid starting was accomplished by maintaining some power to the lamp filaments during standby operation. This does not, of course, detract from the potential advantages of using xenon instead of mercury, which was the subject of the article.

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Cattle, Hay, and *E. coli*

In their recent report, Francisco Diez-Gonzalez *et al.* (11 Sept. 1998, p. 1666) demonstrate that feeding a high-grain diet to cattle results in an acidic colonic environment that selects for or induces acid resistance among the nonpathogenic enteric *Escherichia coli* population. They conclude, "[o]ur studies indicate that cattle could be given hay for a brief period immediately before slaughter to significantly reduce the risk of foodborne *E. coli* infection." This conclusion is unsupported or contradicted by several lines of evidence.

The *E. coli* that contaminate beef typically originate from the hide, the hooves, or the equipment used in slaughter and processing rather than directly from the colon, and likely replicate in environments unlike the colon (*1, 2*). Therefore, the induced acid resistance of *E. coli* contaminating beef is likely to be unrelated to the pH of its ancestral colonic environment.

The *E. coli* 0157:H7 bacterium uses several mechanisms to survive acid environments, some of which are innate and are not influenced by environment (*3–5*). Although acid resistance is likely a factor in an infective dose, induced acid resistance has not been shown to be a factor in *E. coli* O157:H7 infectivity by experimental (dose-inoculation) or observational (epidemiological) data (*3–5*). Therefore, acid resistance induced by exposure to weak acid may not influence the virulence of this pathogen.

Published data on *E. coli* O157:H7, uncited by Diez-Gonzalez *et al.*, tends to contradict or does not support the effects of the dietary change they propose. Sheep experiencing an abrupt diet change have higher concentrations of fecal *E. coli* O157:H7 for longer periods than sheep fed a consistent high-grain diet (*6, 7*). These results suggest that the proposed dietary change