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.

John T. Bickmore
Xerox Corporation (retired), 1849 Blossom Road, Rochester, NY 14625, USA. E-mail: jtbickmore@aol.com

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

hooves and hair. Hancock *et al.*'s reference 2 states that "numbers of coliforms, and *Escherichia coli* on stored trimmings increased *little* and *not at all*, respectively. Increased total counts, and numbers of both coliforms and *E. coli* in displayed product indicated *loss of control* of the temperature of the displayed packaged product" (italics ours).

Their reference 3 indicates that "*all of the available evidence* suggests that organisms with inducible mechanisms of acid resistance will be better equipped to survive these acid challenges and cause disease" (italics ours).

Their reference 5 states that the inducible sigma gene "may play an important role in determining the infective dose of SLTEC" [shiga-like toxin-producing *E. coli* (for example, O157:H7)].

Their reference 4 indicates that "the importance of gastric juice in controlling the outcome of food-borne infections is well recognized. To cause human illness, an invading organism must survive the acidic environment of the stomach before it reaches the intestine. Thus, the acidity of the gastric juice provides a first line of defense against food borne pathogens." *Escherichia coli* O157:H7 is a deadly bacterium in humans, and controlled "dose inoculation" studies would be dangerous.

Medium-quality timothy hay is not dangerous, and Hancock *et al.* appear to have confused hay feeding with starvation. Their reference 8 reported an increase in *E. coli*, but only "after 48 [hours] of fasting." In their reference 6, most statistical tests "were not significant," and even a Fisher's exact test only showed $P < 0.05$ on one day (day 24). In their reference 7, sheep were switched from alfalfa hay pellets (not grain) to a sagebrush/bunch grass mixture or kochia weeds. These latter rations would not have met National Research Council standards for maintenance, and a statistical significance was not reported. The reductions in *E. coli* noted in reference 8 were smaller than ours, but their diet shift was very modest. Their initial diet had only 59% (not 90%) grain, and they fed only 50% (not 100%) hay after the diet shift. References 9 and 10 do not relate directly to our studies. We indicated that "grain increased the number and acid resistance of *E. coli*" in the gastrointestinal tract, but we did not state that grain feeding would affect the percentage (prevalence) of cattle carrying *E. coli* O157:H7.

Hancock *et al.*'s reference 11 examined the rumen (not the colon), and "abrupt changes of diet" caused only "a *slight* disruption of fermentation" (italics ours). Their reference 12 indicates that cattle fed lucerne hay did *not* retain salmonella, and "viable organisms in the feces were rarely detected." Their reference 13 shows a decrease in volatile fatty acids in *mice*, but this decrease was caused by an antibiotic, not a diet shift. Their reference 14 examined young chickens, not cattle, and noted that "it is unlikely that propionic acid itself is solely responsible for the decrease in salmonella colonization, but [it is] more likely due to other factors such as competition for limiting nutrient(s) that result in propionic acid formation." Their reference 15 examined *swine*, not cattle, and their reference 12 indicates that hay feeding does not promote salmonella in cattle.

Intellectual freedom, criticism, and debate are the foundations of science, but meaningful discourses should be based on logical, fair-minded, and carefully constructed arguments.

James B. Russell
Section of Microbiology, Division of Biology, Cornell University and Agricultural Research Service, U.S. Department of Agriculture, Ithaca, NY 14853–8101, USA. E-mail: jbr8@cornell.edu

Francisco Diez-Gonzalez
Department of Food Science and Nutrition, University of Minnesota, St. Paul, MN 55108, USA. E-mail: fdiez@che1.che.umn.edu