

Compensation for Avian Influenza Cleanup

To the Editor: Since 2003, highly pathogenic avian influenza (HPAI) H5N1 has shaken the world. In 10 countries, 258 confirmed cases in humans and 154 deaths have been reported (1). The number of countries with confirmed HPAI in poultry and wild birds jumps to 54 (2). Almost all persons infected with H5N1 have had close contact with sick or dead poultry by having butchered them, plucked them, or played (children) with them (3). Because H5N1 can potentially mutate or reassort into a strain capable of efficient human-to-human transmission, rapid elimination of the H5N1 virus in poultry and other risk-reduction interventions are thought to be essential for preventing further spread of HPAI (4). As a result, thousands of workers around the world have culled millions of domestic poultry (5).

Preemptive culling creates a major concern with regard to compensation. In Nigeria, for example, affected farmers have yet to be compensated >50 million Nigerian Naira (>US\$ 0.4 million) because of the ministry's cash flow problems (6). On the other hand, US poultry farmers who participate in a US Department of Agriculture (USDA) program to prevent the spread of disease would be fully compensated for loss of poultry and equipment if even a low-pathogenic strain of avian influenza was found in the United States (7). This rule not only strengthens US protection against avian influenza but also minimizes any negative effect on the US poultry trade.

As discussed by the World Bank (8), the situations of these 2 countries raise several questions: Who should pay the compensation? For what should compensation be paid? Who should be compensated? With regard

to the first question, each country's government is an exclusive funding source. However, in Nigeria, the amount of compensation overwhelms the government's capacity. Some countries, like Australia, may get additional funding from alternative sources such as private sectors, regional economic groups, or international funds (9). Because national resources are often scarce, most developing countries must rely on international donors for a great deal of the funding for compensation programs.

The response to the second question, extent of compensation, varies. In Nigeria, farmers are partially compensated for loss of poultry; however, in the United States, farmers who are part of the USDA program are fully compensated for loss of poultry and equipment. Setting the amount of compensation is difficult and can affect the outcome of culling efforts. In Thailand, to take advantage of the program in which compensation was perceived as high, some farmers reportedly moved infected poultry into previously uninfected areas. In Vietnam, where compensation was perceived as low, culling compliance was poor (8).

The last question, who should be compensated, seems straightforward for the United States, where only farmers who participate in the USDA program would be fully compensated. However, H5N1 does not affect only farmers who sign up for such a program. And not all poultry are raised in commercial operations, especially in developing countries. In Thailand, for example, >80% of infected poultry are reportedly raised in backyards (10). Reasonable assumptions are that those backyard farmers do not honestly report dying poultry or that they rush sick and dying poultry to market, causing the disease to spread. Additional questions revolve around potential compensation for those who are involved in the poultry industry

but who do not own poultry (e.g., poultry processing plant operators and their staff).

Because each country's needs and circumstances differ, building a coherent plan for tackling HPAI is difficult. However, each stakeholder should consider compensation as part of an overall package of prevention, preparedness, and response strategies toward controlling and preventing the spread of HPAI. Because H5N1 does not respect international boundaries, donors worldwide should step forward to support the most affected and vulnerable developing countries.

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Frog Virus 3 Infection, Cultured American Bullfrogs

To the Editor: Ranaculture, the practice of farm-raising frogs for scientific and culinary purposes, is practiced in many countries, including the United States (1). As with aquaculture, most ranaculture challenges relate to husbandry and disease. In aquaculture, iridovirus infections are reportable and can result in large-scale fish deaths (2,3). The family *Iridoviridae* is composed of *Iridovirus*, *Chloriridovirus*, *Ranavirus*,

and *Lymphocystivirus*. The first 2 infect insects; the latter 2, lower vertebrates (4). Infection with frog virus 3 (FV3), the type species of the genus *Ranavirus*, results in edema, hemorrhage, and necrosis of lymphoid tissue, hematopoietic tissue, liver, spleen, and renal tubules (3,5); mortality rates in free-ranging amphibians are >90% (6).

In May 2006, a commercial American bullfrog (*Rana catesbeiana*) ranaculture facility suffered massive (>50%) deaths of frogs that had recently undergone metamorphosis. The facility, with >25 years of experience, uses an on-site breeding colony and an all-in, all-out system, in which cohorts are moved through the system as 1 unit. Well water is used throughout. The breeding colony and larvae are housed in outdoor tanks to expose them to ambient climatic conditions, thought to facilitate breeding and development. Outdoor tanks are covered with mesh to prevent predation by birds. After metamorphosis, animals are moved indoors.

Full necropsies were performed on 3 of the recent metamorphs. A set of fixed tissue sections from all organs

was routinely processed for light microscopic examination. An identical set of fresh tissue sections was collected for routine bacterial culture and viral analysis. Bacterial isolates were speciated by using an automated system (Sensititer, Trek Diagnostic Systems, Westlake, OH, USA) or conventional biochemical testing. Virus isolation was performed by using a variety of cell lines; random isolates were verified by electron microscopy. A heminested PCR targeting the major capsid protein gene was performed (3), amplicons were sequenced (SeqWright DNA Technology Services, Houston, TX, USA), and a GenBank BLAST search (www.ncbi.nlm.nih.gov/Genbank) was performed.

Pathologic changes in all metamorphs were similar. Gross findings were as follows: irregular gray patches on the skin, cutaneous and enteric erythema, mottled heart and kidneys, pale and friable livers, and enlarged gall bladders. Histologic examination showed lymphoid depletion and necrosis in the thymus and other lymphoid tissues and necrosis in the liver, spleen (Figure), and epidermis.

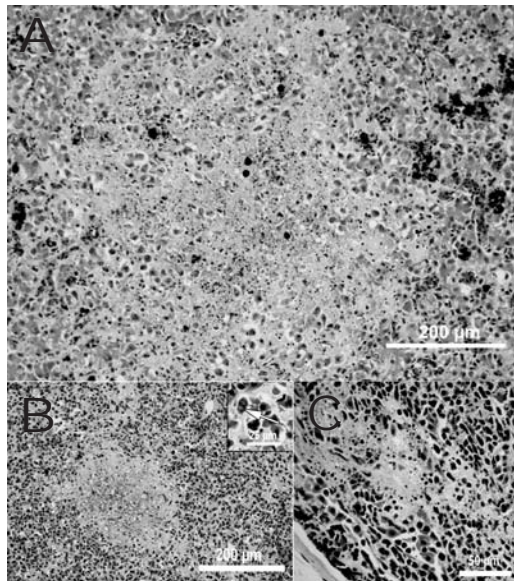


Figure. Light microscopic images of the liver (A), spleen (B), and thymus (C) showing necrosis in an American bullfrog (*Rana catesbeiana*) metamorph infected with frog virus 3. Spleen (B) inset shows intracytoplasmic viral inclusion bodies. Hematoxylin and eosin stain.