

September 30, 2006

To: Members of the National Organic Standards Board

Re: Access to the outdoors for organically raised poultry

On behalf of the organizations listed below, we wish to convey strong support for continued requirement for access to the outdoors for organically raised poultry.

Our support is based on all four of the principles the National Organic Standards Board's Livestock Committee lists as the original intent of the provision:

- to satisfy the birds' natural behavior patterns
- to provide adequate exercise area
- to provide preventive health care benefits
- to answer consumer expectations of organic livestock management

Four arguments could be put forth against giving poultry outdoor access, but each can be addressed readily, effectively, and responsibly:

1. There is increased danger of predation.

While it is true that birds with outdoor access may face greater chance of predation, that can be reduced to negligible risk via simple means: shutting poultry into barns at night, fencing outdoor areas, providing ample cover, and ensuring that people periodically walk the outdoor perimeter. The latter provision is sufficient to deter daytime predatory birds such as hawks and should be standard practice for inspection of stock irrespective of whether the animals are reared indoors or outdoors.

2. Not all birds in large flocks go outdoors.

This assertion has no relevance in determining whether birds should be denied outdoor access, nor any pertinence for the overwhelming majority of birds who do go outdoors when allowed. Three main factors prevent that minority of birds who may not choose to access the outdoors: 1) unnaturally large flock size; 2) lack of cover typical in many outdoor areas, which is an unnatural setting for chickens who are descendents of Red Jungle fowl, forest animals; and 3) lack of adequate means of egress. We are pleased that the NOSB included a requirement to "illustrate how the producer will maximize and encourage access to the outdoors," as this will maximize the number of birds who can benefit. The producer should provide ample and responsibly sized doorways to allow egress from the house and should also consider providing cover, such as trees, bushes, and incomplete fences in addition to shade cloth.

3. Free-ranging birds face more health challenges, such as cannibalism.

This is not true. For birds who are not beak trimmed, rates of cannibalism are no worse in free-range systems than in other non-cage systems, and, in any case, beak trimming (a practice that, if performed, should at least always be performed with anesthesia or analgesic) is just as effective at preventing cannibalism and feather pecking in birds allowed access to outdoors than those raised in other systems.

4. Outdoor access will increase the risk of the emergence and spread of highly pathogenic strains of avian influenza.

Due to the potential public health implications of some avian influenza viruses, this issue deserves close attention. It is important to recognize that all bird flu viruses seem to start out harmless, arising out of the perpetual, benign, stable reservoir of innocuous waterfowl influenza; they begin as mild, low-grade, low pathogenicity avian influenza (LPAI) viruses. H5 and H7 viruses, however, have the potential to mutate into virulent, high-grade “fowl plague” viruses, now known as HPAI—highly pathogenic avian influenza.

The World Organization for Animal Health (OIE) and the Food and Agriculture Organization of the United Nations (FAO) consider it “prove[n]”(1) that once low pathogenicity avian influenza viruses gain access to poultry facilities, they “progressively gain pathogenicity in domestic birds through a series of infection cycles until they become HPAI.”(2) More specifically, U.S. Department of Agriculture researchers believe that “high density confinement rearing methods” give bird flu “a unique chance to adapt to the new species.”(3) That is, intensive factory farming practices may remove the natural obstacles to transmission that prevent the virus from becoming too dangerous.

According to USDA’s leading bird flu researcher and director of the agency’s chief poultry research laboratory, David Swayne, there has never been a recorded emergence of an HPAI virus in any backyard flock or free-range poultry operation.

This is not surprising. Consider an outdoor setting: A duck flying overhead expels a dropping laden with relatively innocuous virus into a grassy field through which a flock of hens is pecking. The hens may be exposed to the virus, but coming straight from waterfowl, the virus is so finely-tuned to duck physiology that it may not gain a foothold before being successfully attacked by a healthy chicken’s immune system. When researchers create deadly bird flu viruses in the lab by passing a harmless waterfowl virus through enough chickens, they facilitate transmission by injecting infected lung tissue from one bird to another. “The conditions under which we generated highly virulent viruses from an avirulent strain are generally not duplicated in nature,” one research team admitted. “However, viruses with low pathogenicity can cause viremia”—the successful invasion of the bloodstream by the virus, an incursion deemed more likely to occur in compromised hosts—“in physically compromised chickens.”(5) The NOSB correctly notes the stress reduction and strengthened immunity associated with outdoor access may make infection less likely.

Consider a second scenario: Tens of thousands of chickens intensively confined inside an unsanitary, football field-sized shed, lying beak-to-beak in their own waste and inhaling air choked with moist fecal dust and ammonia, which irritates the birds’ respiratory passages, further increasing susceptibility in chickens already compromised by the stress of confinement. Since the birds are standing in their own excrement, the virus need not even develop true airborne transmission via nasal or respiratory secretions. Rather, the virus has an opportunity to be excreted in the feces and then inhaled or swallowed by the thousands of other birds confined in the same shed, allowing the virus to circulate rapidly and repeatedly. With so many birds in which to readily mutate, low-virulence strains can sometimes turn into deadly ones. Highly pathogenic bird flu viruses seem predominantly to be products of intensive animal agriculture, often termed factory farming.(8) Indeed, said University of Ottawa virologist Dr. Earl Brown, a specialist in influenza virus evolution, “You have to say that high intensity chicken rearing is a perfect environment for generating virulent avian flu virus.”(9)

The WHO, OIE, and FAO, respectively the world’s leading medical, veterinary, and agricultural authorities, all implicate industrial poultry production as playing a role in the current crisis.(10,11,12) In October 2005, the United Nations issued a press release on bird flu stating: “Governments, local

authorities and international agencies need to take a greatly increased role in combating the role of factory-farming, commerce in live poultry, and wildlife markets which provide ideal conditions for the virus to spread and mutate into a more dangerous form....”(13)

The overcrowding of factory farms conspires with the stress of confinement to cause immune suppression in birds already bred with weakened immunity, offering viruses like bird flu ample opportunities for spread, amplification, and mutation. Placing genetically similar birds into unsanitary conditions typical of factory farms seems the “perfect storm” environment for the evolution of the next superflu strain of pandemic influenza, which raises the question as to why concern about the opposite, free-range flocks, has arisen.

In 2004, while H5N1 traveled across southeast Asia, a highly pathogenic H7N3 outbreak swept through Canada’s Fraser Valley, east of Vancouver,(14) and both backyard chicken farmers(15) and the commercial factory-farming industry laid blame on the other for the outbreak.(16) Publicly, the industry denies culpability, but internally admits to “the growing realization that viruses previously innocuous to natural host species have in all probability become more virulent by passage through large commercial populations.”(17)

University of Ottawa’s Dr. Brown explained to the Canadian Press, “If you get a [H5 or H7] virus into a high-density poultry operation and give it a period of time, generally a year or so, then you turn that virus into a highly virulent virus. That’s what always happens....”(18) Canada’s National Manager of Disease Control within the Food Inspection Agency agreed: “Just passing the virus to 3,000 or 4,000 chickens is enough to change a harmless virus into something more pathogenic.”(19) “It is high-density chicken farming that gives rise to highly-virulent influenza viruses,” Dr. Brown concluded. “That’s pretty clear.”(20)

These conclusions were based on the best available science. **The Canadian outbreak first erupted not in a backyard flock or free-range farm, but on an entirely enclosed, “sophisticated” industrial facility. It then jumped from broiler chicken shed to broiler chicken shed, largely skipping free-range farms.**(21) The spread of the virus was traced mainly to the human lateral transmission of infective feces via equipment or some other fomite moved from farm to farm.(22) This may also explain how the virus was first introduced into the industrial broiler factory farms. Chickens don’t need to come in direct contact with ducks to get infected; they just need contact with the virus, which can be walked into a “biosecure” operation on someone’s clothing.(23) A 2002 outbreak in Virginia led to the deaths of millions of birds and found its way inside 200 poultry operations. In response, a North Carolina State University poultry health management professor understandably wrote in an industry trade journal that “high biosecurity and proper monitoring are still wishful thinking in many areas of intensive poultry production.”(24) That same year, University of Maryland researchers similarly concluded that confined U.S. broiler flocks “are constantly at risk of infection triggered by poor biosecurity practices.”(25)

In the end, epidemiological analyses of the 2004 Canadian outbreak placed **commercial flocks at 5.6 times more likely to be infected than backyard flocks.** Infected backyard flocks were discovered after nearby commercial flocks were infected, suggesting that the virus spread from the industrialized operations to free-range poultry and not vice versa.(26) Birds kept outdoors are more likely to come in contact with wild waterfowl, but also more likely to come in contact with sunlight, space, and fresh air. Lower stress levels may help their bodies better resist the initial infection, and, since they don’t live in their own waste while intensively confined into poorly-ventilated sheds by the tens of thousands as their factory-farmed counterparts do, the virus may not spread effectively enough to mutate into a killer.

It is with our strongest recommendation that the National Organic Program continue to mandate allowance of outdoor access—meaningful outdoor access—for organically raised poultry, not only to

serve its own production principles, but to better protect human and animal health. In addition, USDA must provide improved accreditation oversight of certifiers to allow them to adequately and reasonably enforce the current standards as they apply to outdoor access for livestock.

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