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An HSI Report: Human Health Implications of Intensive Poultry Production and Avian Influenza

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Abstract

The high stocking density, stress, unhygienic conditions, lack of sunlight, and breeding practices typical of industrial poultry and egg production systems may facilitate the emergence and spread of diseases, including highly pathogenic avian influenza viruses with public health implications such as H5N1.

Introduction

In nature, the influenza virus has likely existed for millions of years as a harmless, intestinal, waterborne infection of waterfowl, particularly ducks.[1] All strains are thought to originate as mild, low-grade, low-pathogenicity avian influenza (LPAI) viruses, but H5 and H7 strains have the potential to become virulent, high-grade HPAI—highly pathogenic avian influenza—"fowl plague" viruses.[2]

Influenza viruses are normally benign in waterfowl, but strains that are able to infect land-based birds may become more dangerous to humans, as viral mutations naturally selected to be better adapted to terrestrial species may be better suited for airborne spread. For influenza to mutate into a highly pathogenic strain, the virus also needs to be able to overwhelm host defenses while retaining efficient transmissibility. Unlike the conditions typical of commercial intensive farm animal production facilities, in nature, animals are not overcrowded and confined at unnatural densities, and virulence is presumably constrained since spread is dependent on the host remaining mobile enough to infect others.[3]

In 1989, an avian influenza virus infected horses in China, killed 20% of a herd, and then lost its virulence.[4] Overcrowded mink fur farms have also suffered influenza outbreaks, but the viruses caused only localized outbreaks before dying out.[4] Such epidemics tend to be self-limited,[5] presumably since the population is restricted in size and not rapidly replenished with new hosts. Under overcrowded unhygienic conditions with frequent restocking, though, natural biological checks and balances on virulence may no longer apply.[3]

Low to High Pathogenicity

Avian influenza viruses only tend to "heat up," in the words of Dutch virologist Albert Osterhaus, "when they pass from wild birds to poultry."[6] The World Organisation for Animal Health (OIE) and the Food and Agriculture Organization of the United Nations (FAO) agree that it has been "prove[n]"[7] that once certain LPAI viruses gain access to poultry facilities, they can "progressively gain pathogenicity in domestic birds through a series of infection cycles until they become HPAI."[8] According to researchers with the U.S. Department of Agriculture (USDA), "high density confinement rearing methods" typical of industrial poultry production systems give avian influenza "a unique chance to adapt to the new species."[9] Industry trade journal *World Poultry* listed some factors that make intensive poultry facilities such "ideal"[10] "breeding grounds for disease"[11]: "inadequate ventilation, high stocking density, poor litter conditions, poor hygiene, high ammonia level, concurrent diseases and secondary infections."[12] Indeed, an avian virology textbook states: "Viral infections can move fastest through groups of birds maintained in closed, crowded, unsanitary conditions."[10] There has never been a recorded transformation of a mild strain to a highly pathogenic flu virus in any backyard or free-ranging chicken flock.[13]

Stocking Density

One factor allowing for an increase in the virulence of avian influenza is the high stocking density of intensive poultry and egg production facilities. According to anthropologist Wendy Orent, "H5N1 has evolved great virulence among chickens only because of the conditions under which the animals are kept—crammed together in cages, packed into giant warehouses. H5N1 was originally a mild virus found in migrating ducks; if it killed its host immediately, it too would die. But when its next host's beak is just an inch away, the virus can evolve to kill quickly and still survive."[14] In a typical commercial poultry production facility with tens, if not hundreds, of thousands of intensively confined and overcrowded susceptible hosts, large viral loads can rapidly cycle from one bird to the next, enabling the virus to accumulate adaptive mutations.

In industrial broiler chicken systems, 20,000 to 30,000 day-old chicks[15] are placed on the floor atop coarse wood shavings or other litter material in an otherwise barren shed. As they grow, the crowding intensifies. According to the standard reference manual for intensive chicken production, "Under standard commercial conditions chickens weighing 4.5 to 6 lbs have little more than a half a square foot of living space per bird in the last two weeks of their 42-47 days of life."[16] As one researcher reported, "it looks as though there is white carpet in the sheds—when the birds are fully grown you couldn't put your hand between the birds, if a bird fell down it would be lucky to stand up again because of the crush of the others."[17] "Obviously," Louisiana State University veterinary scientists write, under these conditions "the potential for a disastrous epidemic is very high."[15]

The majority of egg-laying hens in the world are confined in battery cages,[18] barren, wire enclosures, and stocked at such densities that each hen is typically allotted less floor space than a standard letter-sized piece of paper.[19] Research has found that a hen needs an average of approximately 1,880 cm^2 (291 in^2) of space to flap her wings, 1,270 cm^2 (197 in^2) to turn around, and 475 cm^2 (74 in^2) to stand freely.[20] Currently, U.S. commercial battery-cage facilities allow each bird an average of approximately 430 cm^2 (67 in^2).[19] With up to ten hens per cage and thousands of cages stacked vertically in multiple tiers, industrial egg production facilities can average more than 100,000 chickens per shed.[21]

The Royal Geographical Society notes: "Massive demand for chicken has led to factory (battery) farming which provides ideal conditions for viruses to spread orally and via excreta which inevitably contaminates food in the cramped conditions that most birds are kept in."[22]

Europe is moving away from this level of intensification, for both chickens raised for meat and egg-laying hens. In 2005, the European Commission proposed legislation to impose a maximum stocking density for broiler chickens throughout Europe.[23] In sharp contrast to the U.S. standard commercial "half a square foot of living space per bird," certain organic standards in the United Kingdom already reportedly require a minimum of 16 m^2 (170 ft^2) per bird.[24] For the health and welfare of egg-laying hens, the European Parliament voted to ban conventional battery-cage systems entirely by 2012.[25]

In a joint consultation, the World Health Organization (WHO), the FAO, and the OIE noted that the sheer number of intense contacts between birds with increasing flock density serves to spread and amplify disease agents like avian influenza.[26] This is supported by research showing that increasing stocking densities of chickens result in an increased burden of infectious disease agents,[27] a relationship also found in other species.

In the influenza pandemic of 1918, during which an estimated 50 million people died, a U.S. Army regiment whose barracks allowed only approximately 4.2 m^2 (45 ft^2) per soldier reportedly had a flu incidence more than ten times that of a regiment afforded about 7.25 m^2 (78 ft^2) per person.[28] In pigs, respiratory diseases[29] such as chronic pleuritis and pneumonia have reportedly been strongly correlated to increased crowding of pigs per pen[30] and per building,[31] corresponding to increased levels of bacteria cultured in the air.[30] Similar studies on influenza in commercial pig operations have come to the same conclusion: An increased density of pigs per pen, pigs per operation, and pigs per municipality all have been shown to be associated with increased risk of swine flu infection.[29] Researchers blame the increased risk in part on diminished air volume per animal, increasing the

concentration of infectious particles and thereby facilitating aerosol spread.[29] Dorothy H. Crawford, a professor of clinical microbiology at the University of Edinburgh concluded that "overcrowded farms are a hotbed of genetic mixing for flu viruses."[32]

Richard Webby's research team at St. Jude's Hospital reportedly considers increased poultry density a "big factor" in the rise of highly pathogenic viruses. The "more hosts in close confinements," the more easily the virus can mutate into a form capable of infecting humans and eventually spreading throughout the human population.[33]

Virus Survival and Spread

After passing through an industrial, confined animal production facility, virus may continue to survive. Depending on the ambient conditions, influenza may endure in wet manure for weeks.[34] During this time, the virus may spread on footwear, clothing, tires, trucks, cages, crates, insects, rodents, or even via the wind, expelled outwards by ventilation fans inside poultry sheds.

Spatial analyses of the spread of H5N1 in Asia found that outbreaks corresponded to areas with the greatest numbers of chickens per square mile. Whether within a shed, on a farm, or across a region, "outbreaks of avian influenza correspond to where [poultry] population density is very high,"[35] determined Shigeru Omi, the WHO's regional director for the Western Pacific.

Stressors

Frederick A. Murphy, Dean Emeritus of the School of Veterinary Medicine at the University of California, Davis, has noted how intensification in farm animal production practices "often allow[s] pathogens to enter the food chain at its source and to flourish, largely because of stress-related factors."[36] The physiological stress created by crowded confinement can have a profound impact on immunity,[37] predisposing animals to infection.[29] Diminished immune function reduces protective responses to vaccinations. "As vaccinal immunity is compromised by factors such as…immunosuppressive stress," writes Richard Witter, a leading[38] USDA expert on chicken vaccines, "mutant clones have an increased opportunity to selectively multiply and to be seeded in the environment."[39] Studies exposing birds to stressful housing conditions provide "solid evidence in support of the concept that stress impairs adaptive immunity in chicken."[40]

Chickens placed in overcrowded enclosures develop, over time, "increased adrenal weight," a swelling growth of the glands that produce stress hormones like adrenaline, while, at the same time, experiencing "regression of lymphatic organs," a shriveling of the organs of the immune system.[41] This is thought to demonstrate a metabolic trade-off in which energies invested in host defense are diverted by the stress response, which can result in "extensive immunosuppression."[42]

Leading meat industry consultant Temple Grandin, an animal science professor at Colorado State University, described the stresses of battery-cage life in an address to the National Institute of Animal Agriculture: "When I visited a large egg layer operation and saw old hens that had reached the end of their productive life, I was horrified. Egg layers bred for maximum egg production…were nervous wrecks that had beaten off half their feathers by constant flapping against the cage."[43] Referring to egg industry practices in general, Grandin reportedly noted, "It's a case of bad becoming normal."[44]

In battery cages, laying hens are unable to engage in most of their natural behaviors, including nesting, perching, dustbathing, scratching, foraging, exercising, running, jumping, flying, stretching, wing-flapping, and freely walking, which can lead to frustration and additional stress. Overcrowding may impose a social stress that has been shown for nearly 30 years to weaken resistance to viral infection[45] and, more recently, a multitude of other disease challenges.[41] One industry specialist wrote in *World Poultry* that it is "proven that high stress levels, like the ones modern management practices provoke," lead to a reduced immune response.[46]

Other sources of stress for many birds raised for meat are the mutilations performed on them without anesthesia or analgesia; their combs, spurs, claws, and toes or portions of toes can be cut off to limit the damage of stress-induced aggression or for identification purposes.[27] Egg-laying birds also undergo mutilations without pain relief. Typically, U.S. laying hens, when chicks, are "beak-trimmed"—parts of their beaks are sliced off with a hot blade, an acutely painful[47] procedure shown to impair their ability to grasp and swallow feed.[48] Already banned in some European countries as unnecessary,[49] the procedure is viewed by some poultry scientists as "stop-gap measures masking basic inadequacies in environment or management."[50]

A National Defense University Policy Paper on agricultural bioterrorism specifically cited mutilations, in addition to crowding, as factors that increase stress levels to a point at which the resultant immunosuppression may play a part in making U.S. animal agriculture vulnerable to terrorist attack.[51] Ian Duncan, Emeritus Chair in Animal Welfare at the University of Guelph, has been outspoken about the animal and human health implications of these stressful practices: "All these 'elective surgeries' involve pain, perhaps chronic pain. No anesthetic is ever given to the birds. These mutilations are crude solutions to the problems created by modern methods of raising chickens and turkeys."[52]

According to William E. Donaldson, the former head of the Department of Poultry Science at North Carolina State University:

> [Newborn turkey chicks] are squeezed, thrown down a slide onto a treadmill, someone picks them up and pulls the snood off their heads, clips three toes off each foot, debeaks them, puts them on another conveyer belt that delivers them to another carousel where they get a power injection, usually of an antibiotic, that whacks them in the back of their necks. Essentially, they have been through major surgery. They have been traumatized.[53]

Research performed at the University of Arkansas' Center of Excellence for Poultry Science suggests that the cumulative effect of multiple stressors throughout turkey production results in conditions like "turkey osteomyelitis complex" (TOC), where decreased resistance to infection leads to a bacterial invasion into the bone, causing the formation of abscessed pockets of pus throughout the birds' skeletons. USDA researchers blame TOC on "stress-induced immunosuppression" in turkeys who "respond to the stressors of modern poultry production in a detrimental manner."[54] The stress of catching and transport alone has been shown to induce the disease.[55]

Unhygienic Conditions

The tens to hundreds of thousands of animals reared in a single, intensive confinement production building produce an extraordinary amount of waste. Since avian influenza viruses may survive in wet manure for weeks, these unhygienic conditions pose significant risk.

A 25,000-bird broiler chicken flock produces more than 1 tonne (1,000 kg or 2,205 lbs) of droppings every day.[56] According to the USDA, 1 g (0.035 oz) of manure (approximately the weight of a paper clip) from an infected chicken can contain "enough virus to infect 1 million birds."[57]

Due primarily to genetic selection for fast growth and the strain such unnatural weight gain takes on their bodies, a majority of commercially farmed birds suffer from crippling leg disorders and gait abnormalities.[58,59] The birds are bred for such size that their legs may become so weak that they cannot support the weight of their bodies, leading to more time resting on the floor in the litter, which may increase fecal contamination of the carcass.[60] Grandin writes: "Today's poultry chicken has been bred to grow so rapidly that its legs can collapse under the weight of its ballooning body. It's awful."[61]

By six weeks old, chickens raised for meat have reached market weight. Unnaturally heavy and experiencing such stress on their hips and legs, they spend more than three-quarters of their time lying in their own waste.[62] By the time they are slaughtered, all of their carcasses show evidence of gross fecal contamination.[60] This is one

reason why poultry products are such prime carriers of food-borne illness,[63] especially since, unlike with cows and pigs, the skin can be eaten with the meat.[64]

After a broiler chicken shed has been depopulated, the building may not be cleaned before a new flock is introduced, in which case hatchling chicks are placed directly on the tons of feces that have already been layered down. Veterinary experts have been critical of this practice. As specified in the journal of the OIE, fecal waste should be removed from the shed before adding a new flock.[65] The FAO agrees.[66] Placing day-old chicks in sheds contaminated with "built-up" litter is said to expose the birds to "a wide range of poultry pathogens."[67] Indeed, millions of Americans are sickened by *Campylobacter* infection each year,[63] and the Advisory Committee on the Microbiological Safety of Food reported that the most significant source of *Campylobacter* infection in chickens is "the environment of the industrial broiler house."[68] The poultry industry suspects that "general farm hygiene could reduce the numbers [of *Campylobacter* bacteria on carcasses] by around 40%." A "zero tolerance" policy is impractical, the industry emphasizes, "because it is impossible to achieve at reasonable cost…."[69]

In a specially commissioned feature on preventing disease to celebrate *Poultry International*'s 40-year publishing history, the trade magazine noted: "Replacing used litter between flocks is a standard practice worldwide, but it will not gain acceptance in the United States." The investment would evidently not be worth the return. "[U]nless federal regulations force drastic changes," the article concluded, "nothing spectacular should be expected."[70]

Contaminated Air

Feces decomposition generates several irritating chemicals, including hydrogen sulfide, methane, and ammonia,[71] which "in a poultry house is nauseating to the caretaker, irritates the eyes, and affects the chickens," states one poultry science textbook.[16] Given the extreme stocking density of intensive production facilities, the litter can get so saturated with excrement that birds may develop sores or ammonia burns on their skin, known as breast blisters, hock burns, and footpad dermatitis, all of which have become significantly more common and serious over the last 30 years.[27]

Studies have shown that high levels of ammonia also increase the severity of respiratory disorders, such as pneumonia,[72] in part by directly damaging the respiratory tract, predisposing birds to infection.[73] A large-scale study of millions of birds from nearly 100 commercial farms across multiple countries found that ammonia levels increased the excretion of the stress hormone corticosteroid,[74] a potent immune depressant.

Ammonia may also directly suppress the immune system. The gas gets absorbed into the birds' bloodstreams, where it may interfere with the action of individual white blood immune cells.[75] Although airborne aerosol spread of H5N1 avian influenza virus remains relatively inefficient, even among birds,[76] the ammonia damage associated with intensive poultry production may facilitate the virus acquiring so-called pneumotropic, or "lung-seeking," behavior.[77]

In sum, high concentrations of birds in a typical shed lead to high concentrations of aerial pollutants, which subsequently result in increased respiratory disease challenge to the birds' immune systems.[27] In addition to fecal material, the airborne dust in such facilities has been found to contain bacteria, bacterial toxins, viruses, molds, nasal discharge, feather and skin debris, feed particles, and insect parts.[71] Poultry confinement buildings can average 7 million bacteria floating in every cubic meter (1.3 yd^3) of air.[71] These dust particles clog the birds' lungs, overwhelming the lungs' clearance mechanisms. Researchers demonstrated decades ago that exposing a chick to a normally harmless strain of *E. coli* in an environment clouded with dust or ammonia can cause disease.[78] The very air birds breathe in intensive confinement may predispose them to infection with influenza.

Lack of Sunlight

Approximately 0.9 million tonnes (0.9 billion kg or 2 billion lbs) of poultry litter, including feces, are fed to U.S. cattle each year,[79] and much of the rest is spread upon cropland as fertilizer. The open air, combined with the sanitizing rays of the sun, rapidly dries the manure and kills the fecal micro-organisms.[71] In contrast, human pathogens like *Salmonella*[80] and *Campylobacter*[81] and viruses like H5N1 can thrive in the moist litter found inside dimly lit poultry sheds.

Transmission experiments with chickens reveal that the spread of H5N1 is predominantly via the fecal-oral route rather than in respiratory droplets. H5N1 can survive in wet feces for weeks but is inactivated as soon as the feces dry out in ambient temperatures.[82] As such, the spread of avian influenza viruses like H5N1 are expected to be relatively inefficient in outdoor, free-range settings.

In countries like Thailand, the combination of tropical heat and crowded confinement necessitates "evaporative cooling" in poultry sheds, which uses large fans and a water mist to cool down the birds during the hot season.[83] Although this practice reduces heat stress, the high level of humidity ensures that the litter is moist, which may facilitate the spread of pathogens like avian influenza. Though so-called "evap houses" increase flock survival, they may also increase virus survival.

From an avian virology textbook:

> Birds that are housed indoors year-round should be considered more susceptible to infectious diseases," an avian virus textbook reads, "because of decreased air quality, the accumulation of pathogens in a restricted environment, and the lack of exposure to sunlight. These factors function collectively to decrease a bird's natural resistance to disease.[10]

The absence of adequate ventilation and direct sunlight common in intensive confinement settings is a combination that may facilitate the spread of influenza virus. During the pandemic of 1918, Boston hospitals filled beyond capacity. A tent hospital was set up in nearby Brookline. Though exposing ailing patients to the chilly New England autumn was reportedly condemned by Bostonians as "barbarous and cruel," the fresh breeze and sunshine seemed to afford the overflow patients far better odds of survival than those inside the overcrowded, poorly ventilated hospitals.[84] Perhaps the best-studied illustration of the danger of crowded, enclosed spaces in human medicine was a commercial airline flight in 1977 that was grounded on the tarmac for more than four hours due to a mechanical failure while a young woman lay prostrate in the back of the cabin feverishly coughing with the flu. Within three days, nearly three out of four passengers fell ill with her virus.[85]

A study of the 1957-58 pandemic also demonstrated the potentially therapeutic role of sunlight. Ultraviolet (UV) rays, which damage genetic material,[86] have been used in tuberculosis (TB) wards to kill off some of the TB germs coughed into the air. To see if influenza could be killed in the same way, researchers compared influenza rates in patients in TB-infected buildings with UV lights to patients in TB-infected buildings without UV lights during the mid-1950s pandemic. In the rooms without UV lights, 19% of patients got the flu, while only 2% of those in rooms with UV lights became infected, a statistically significant difference.[87] This suggests that sunlight may help sanitize influenza virus from the air and highlights the increased risk of crowding poultry indoors. For flocks raised outdoors, according to the FAO, the natural UV rays of the sun may "destroy any residual virus."[88]

Despite the evidence that sunlight has an effective disinfectant quality and that adequate ventilation contributes to reduced risk of viral infection, the commercial poultry industry has not yet incorporated these two important, health-related components into common practice. Because increased light encourages greater activity by the chickens, as one poultry industry journal describes, "birds burn energy on activity rather than on growth and development." Natural lighting has a negative impact on "feed conversion,"[89] meaning the animals expend energy on moving rather than gaining weight to more quickly reach market size. According to trade publication *Broiler Industry,* "It is obvious that the light supplied by sunshine during the day and normal darkness at night is the most inferior of any lighting program."[90]

Genetic Selection for Production Traits

Breeding for such traits as greater breast muscle in birds raised for meat or increased rates of lay in egg-laying hens has contributed to diminished immune competence among modern poultry, which in turn has led to greater susceptibility to illness, infection, and mortality. Given the intensive genetic selection for productivity over immune functionality, almost all modern commercial chickens may be compromised in a way that would facilitate wild waterfowl viruses taking hold. "[D]omestic poultry have been bred to be plump and succulent rather than disease-resistant," Bryan Eaton, a senior virologist at the Australian Animal Health Laboratory, reportedly points out. "[T]hey're sitting ducks, so to speak, for their wild cousins' viruses."[91] Researchers corroborate, finding that broiler chickens selected for accelerated growth suffer from weakened immunity, which increases mortality by making them more susceptible to a variety of infectious diseases.[92]

Today's commercially reared chickens are significantly different than their predecessors. Red Junglefowl, ancestors to the modern-day chicken, laid only about 25 eggs a year,[93] while today's laying hens produce more than ten times that number,[94] leading to increasing problems with uterine prolapse[95] and poor skeletal bone mass that can contribute to broken bones due to critical weakening, as skeletal calcium is mobilized to form shells for the eggs.[96] It took approximately four and one-half months for chicken ancestors to reach about 1 kg (2.2 lbs);[97] in the 1950s, poultry industry manipulation resulted in chickens exceeding 2 kg (4.4 lbs) in less than three months. Today, due mostly to selective breeding (in addition to growth-promoting drugs), chickens may reach 2.4 kg (5.4 lb)[98] in an average of 45 days.[52] According to the National Chicken Council, an industry trade group, every year, producers are able to reach target weight at least one-half day earlier.[98]

Such intense genetic selection for productivity has jeopardized the health and welfare of poultry. Mortality rates of broiler chickens, for example, are up to seven times that of chickens not bred for fast growth. Ongoing efforts to increase breast-meat yield, for example, have created a higher propensity for musculoskeletal problems, metabolic disease, immunodeficiency, and male infertility, in part, perhaps, because the extra protein going to breast muscle production comes at the expense of internal organ development.[99]

Researchers conclude, "It appears that broilers with faster growth rate are under physiological and immunological stress that makes them more sensitive to infectious diseases…."[100] This has been shown for both viral[101] and bacterial[102] pathogens. In one study, broiler chickens were intentionally infected with *E. coli*, and around 40% of the fast-growing, heavier birds died, compared to 8-20% mortality for slower-growing breeds. The scientists commented, "These results indicate that rapid growth rate substantially reduces broiler viability…."[103]

Studies with turkeys reveal the same findings. Lighter and slower-growing turkey breeds have better immune performance than those used for conventional, commercial production[104] and are thereby more resistant to stress[105] and disease.[106] Researchers have observed that in natural outbreaks of disease like fowl cholera,[107] turkeys bred for increased egg production and those selected for increased body weight had significantly higher mortality rates.[108] Slower-growing, lighter breeds of turkeys also have greater adaptability to the stresses associated with production, such as overcrowding.[109] USDA researchers at the University of Arkansas went so far as to suggest in a 2005 paper in *Poultry Science* that "fast growth in modern turkey lines" may result in stress responses "incompatible with the severe stressors that sometimes occur during commercial poultry production."[105]

Selection for productivity has been so intense that commercial turkeys, like broiler chickens, can barely support their own weight. A staff editor of the leading U.S. livestock feed industry publication writes that "turkeys have been bred to grow faster and heavier but their skeletons haven't kept pace, which causes 'cowboy legs.' Commonly, the turkeys have problems standing…and fall and are trampled on or seek refuge under feeders, leading to bruises and downgradings as well as culled or killed birds."[110] One group of researchers concluded, "We consider that birds might have been bred to grow so fast that they are on the verge of structural collapse."[111]

Many do collapse and spend much of their time lying in their own waste. Similar to broiler chickens, most turkeys in commercial production are overcrowded in warehouse-like sheds, and the majority[112] suffer from ulcerative contact dermatitis, from breast blisters to bed sore-like hock burns.[113] These painful lesions add to the stress that may impair overall immune performance. USDA researchers conclude: "Selection of poultry for fast growth rate is often accompanied by a reduction in specific immune responses or increased disease susceptibility."[106]

Breeders have tried selecting for antibody response directly, but poultry scientists have found that those with the best antibody responses consistently had significantly lower weights at all ages.[114] Research dating back 30 years shows that chickens bred to be disease-resistant have lower body weight and produce smaller eggs.[115] Indeed, studies suggest that immune defects may actually enhance poultry performance.[105]

Resource Allocation Theory

The relationship between reduced immunity with maximized productivity may best be explained by the "resource allocation theory." There is only a certain amount of energy, protein, and other nutrients entering an animal's system at any one time. Those resources can go to build muscle or produce eggs, for example, or to host defense. Cows in the dairy industry have been bred to "redirect resources from the maintenance of an adequate immune system to milk production in order to maintain advantages in milk yield," reads one dairy science textbook,[116] indicating a trade-off between production traits and immunocompetence.[117]

Studies show that slower-growing chicken breeds have larger[118] and better developed[119] antibody-producing immune organs. Instead of being bred to transfer the bulk of resources to build breast meat while neglecting other needs, these slower-growing breeds presumably had sufficient resources to foster a more functional antibody response system.[118] Antibodies are critically important for vaccine effectiveness, particularly in animals like commercial broiler chickens who are killed around six weeks of age and do not have time to acquire a set of their own immune memories. "Those animals which are intensively reared and slaughtered young," notes one agricultural microbiologist, "will have the greatest potential for carrying pathogens."[120]

The maintenance of an effective immune system is metabolically very costly. The macrophage immune cells burn through almost as much energy as maximally functioning heart muscle.[121] Antibodies are made out of protein. When the body produces thousands of antibodies per second, there is less protein available for growth. Studies show that chickens capable of mounting a decent antibody response have lower weight and lower weight gain than chickens with suboptimal antibody production.[122]

Germ-free chicks raised in germ-free environments grow faster than chickens in unsanitary environments.[123] Even minute exposures to the normal microbial flora of the gut are enough of an immune stimulus to significantly reduce growth rates.[121] Though there's no tissue damage and no evidence of disease, simply the normal day-to-day functioning of the immune system diverts energy from maximal growth,[123] which explains why germ-free chickens in a sanitary laboratory environment can be dosed with antibiotics without any change in growth rates, whereas commercially confined chickens fed antibiotics demonstrate an apparent spurt in growth.[124] Even relatively insignificant challenges to the immune system can significantly affect growth. Simple vaccinations can result in a 21% decline in daily weight gain for farm animals and increase protein demands as much as 30%,[125] demonstrating the inverse balance between growth and immunity.

The poultry industry could breed for improved immunity over productivity even though it has "been shown to result in decreased BW [body weight],"[106] but admits that "disease resistance will not be selected for if the cost in a loss of genetic improvements in other traits is too great."[126] According to Gerard Albers, former Director of R&D of Nutreco's Breeding Research Center, "decisions in the poultry industry are largely and increasingly driven by economic considerations but the psychological impact of flock morbidity and mortality on the farmer cannot be ignored. Mortality rates above a certain psychological threshold are unacceptable." Nevertheless, Albers is not optimistic that breeding for "increased livability" will take precedence over selection for "more

profitable" traits.[127] The same attitude has also been expressed in the egg industry. In an article titled "Industrial Perspective on Problems and Issues Associated with Poultry Breeding," laying hen breeding corporations assert that "[e]gg production per hen housed will continue to be the single most important trait under selection."[128]

In Europe, breeding programs are under evaluation. The European Commission's Scientific Committee on Animal Health and Animal Welfare's broiler chicken report stated that its "most important recommendation" was that "[b]reeders should give a considerably higher priority to health variables in the breeding index, if necessary at the expense of the selection pressure for growth and feed conversion."[27] Conversely, in the United States, growth rates continue to be pushed faster every year.[129] In *World Poultry*, Soledad Urrutia, editor of *Avicultura Profesional*, wrote "Mathematically, it is evident that the present rate of improvement in growth cannot be continued for more than a couple of decades, or the industry will be faced with a bird that virtually explodes upon hatching."[46]

Lack of Genetic Diversity

As of 2000, more than 95% of birds raised globally were provided by four turkey breeding companies, five egg-laying chicken breeders, and five broiler breeder companies.[126] A single pedigreed cockerel can potentially give rise to 2 million broiler chickens.[126]

Mass consolidation has positive and negative aspects. Selection decisions can be propagated across the entire world in a matter of years so, for example, if the industry elected to prioritize selection for stronger immunity, virtually the entire global flock could be replaced with the improved disease-resistant variety in three or four years. However, even greater emphasis on production traits with detrimental effects on immunity would be distributed at the same speed.[126] Another significant downside to diminishing the breeding pool is the increasing genetic uniformity of poultry, which alone may increase the susceptibility of the global flock to disease.[51]

According to the FAO, 740 farm animal breeds have gone extinct,[130] and breeds continue to disappear at a rate of one or two each week. More than 1,000 breeds—one out of four of all farm animal varieties—are presently facing extinction.[131] The greatest threat to farm animal diversity, according to the FAO, is the export of high-producing breeding stock from industrialized to developing countries that dilutes, or completely displaces, local native breeds.[132]

This erosion of biodiversity may have serious human public health consequences. The American Association of Swine Veterinarians has explained why the genetic bottlenecking created by narrowly focused breeding schemes may be a main reason for the mounting concern over human zoonotic diseases. "As genetic improvement falls into the hands of fewer companies and the trend towards intense multiplication of a limited range of genotypes (monoculture, cloning) develops, there is mounting concern that large populations may have increasingly uniform vulnerability to particular pathogens."[133]

This risk is faced by any type of agricultural mono-cropping and was clearly illustrated by the challenges faced by the U.S. corn industry in the early 1970s. At that time, the industry had developed "Tcms" corn, a highly profitable strain adapted for large-scale farming. After 85% of the nation's seed corn acreage was covered with the new variety, the industry realized that the Tcms strain was particularly susceptible to a rare form of leaf blight fungus that then wiped out areas of the U.S. Corn Belt.[134]

Biodiversity is biosecurity. Even the most virulent of diseases typically do not kill all infected individuals, in part due to natural, inborn genetic variability. In the wild, natural selection takes advantage of this variation to pass disease-resistant qualities to the next generation.[135] The diversity in nature tends to ensure that some individuals will survive future diseases and challenges. Artificial selection for production qualities undermines this important ability by inbreeding unnaturally elevated egg production and fleshiness over fitness, as well as by reducing the genetic diversity that can act as resistance insurance against present and unforeseen threats of disease.[136]

Acquired Immunodeficiency Syndromes

The overcrowded, stressful, unsanitary conditions inherent to intensive poultry production may not only directly increase the risk and spread of avian influenza infection, but may predispose the birds to infections with immunosuppressive viruses that could further compromise their already dysfunctional immune systems. The relationship between immune-weakening poultry viruses and avian influenza was first proposed by University of Hong Kong zoologist Frederick Leung and later expanded upon by anthropologist and agroecologist Ronald Nigh.[137] Leung noted a speculative correlation between Hong Kong chicken farms that had suffered outbreaks of an immunodeficiency virus known as infectious bursal disease virus in 1996 and the subsequent initial outburst of H5N1 approximately six months later in 1997.[138]

The bursa is a specialized avian organ responsible primarily for the development of a bird's immune system.[139] Human antibody-producing "B-cells" were named after this organ.[140] Just as HIV in humans replicates in white blood cells called T-helper cells, leading to their destruction and the body's subsequent immunodeficiency, the infectious bursal disease (IBD) virus in birds infects B-cells, crippling the immune system and leaving survivors immunosuppressed for life.[141] With a "severely impaired"[142] ability to produce antibodies, surviving birds respond poorly to vaccinations[27] and are susceptible to a wide variety of viral, bacterial, and parasitic diseases.[143]

Beginning in the 1980s, two decades after the IBD virus was identified,[143] dramatically greater numbers of chickens suffering from various respiratory infections were found in the United States. Vaccines were no longer as effective.[139] Investigators discovered that a new hypervirulent strain had arisen in the most concentrated poultry production area in the world,[144] the Delmarva Peninsula,[145] incorporating corners of Delaware, Maryland, and Virginia. Due in part to a "high concentration of poultry in close proximity,"[146] the Delaware variant,[147] as it was called, soon extended far beyond the region. Evidence exists that the IBD virus of domestic chickens has been detected in Emperor penguins in the Antarctic, considered an example of industrial animal agriculture's "pathogen pollution" to the farthest reaches of the globe.[148]

Since the 1960s,[149] there has also been a dramatic increase in the virulence of another viral affliction—Marek's disease (MD), first described a century ago.[150] Besides tumors in the skin, muscles (meat), nerves, and abdominal organs of chickens, the Marek's disease herpes virus also causes immunosuppression.[141]

A major 2005 scientific review described the evolution of virulence:

> Poultry production up to the mid 1900s mainly comprised backyard farming with very low population densities of birds…with low growth rates and low egg production. In this environment, MD was not considered as a major disease even though outbreaks of MD were reported in different parts of the world. However, since the 1960s there have been major changes in poultry production practices. Today poultry production has become a major global industry operating in very high population densities under highly intensive management conditions aimed at higher rates of growth and productivity….Until about 1960, when the poultry production was not on an intensive scale, both the virus and the hosts were able to achieve a state of balanced co-existence. However, the transformation of the poultry industry into the intensive production practices from the early 1960s saw a shift in this balance greatly in favor of the virus. The continuous availability of large populations of genetically susceptible naïve hosts, usually in an overcrowded environment, enabled the virus to spread rapidly, encouraging their rapid evolution towards greater virulence. This was evident when huge MD outbreaks swept through poultry flocks in the 1960s, wiping out large populations all around the world.[151]

The first wave of evolution in the late-1950s shifted the virus from "mMDV" (mild Marek's disease virus) to "vMDV" (virulent Marek's disease virus). Due in part to continued and escalating industrial practices, "vMDV" became "vvMDV," and presently the world is dealing with "vv+MDV."[151]

Other immunosuppressant viruses include chicken infectious anemia virus (CIAV) and a virus that causes hemorrhagic enteritis in turkeys.[143] CIAV was first described in 1979 and has since spread throughout the world

to become ubiquitous in egg- and meat-type chickens worldwide.[152] CIAV destroys immune precursor cells, undermining the immune system before it can even develop.[153] Immunosuppression associated with CIAV is considered to be a factor in "many of the disease problems in flocks raised under the high-density conditions of modern poultry production."[154]

These immunodeficiency viruses can interact with each other to synergistically further predispose the global chicken flock to infection. CIAV infection, for example, can boost the virulence of Marek's virus, and co-infection between IBD virus and CIAV can result in an even more profound vulnerability to additional infectious disease agents.[152] A poultry scientist with Tyson Foods described the U.S. poultry industry as being "in a constant battle with immunosuppressive diseases,"[145] and a 2005 *World Poultry* "Global Disease Update" reported that "[t]he deleterious effects of infections which suppress the immune systems are underrated in many parts of the world."[147]

The unhygienic conditions under which birds are raised in commercial poultry operations conspire to spread these viruses. "The transformation in the poultry farming practices into a highly intensive industry has enormously changed the poultry house environment," read one Marek's disease review. Infection with Marek's disease occurs when a chicken inhales infected dust in a poultry shed saturated with virus flaking directly off the chickens' skin.[151] The emergence of new strains of IBD virus has also been blamed in part on "improper cleaning and disinfection."[139] One reason why the industry may *not* clean and disinfect sheds more frequently is that they want young breeding chickens to get infected with viruses like CIAV early, in hopes that they will clear the infection before egg laying leads to progeny with "poorer performance."[145] Immunodeficiency diseases like Marek's cost the poultry industry more than $1 billion annually,[151] but improving sanitation may be costlier. One animal science textbook explains that "compromise inevitably must be struck because animal agriculture is a business, and providing the best environment possible may be unprofitable."[155]

None of these viruses affect humans directly, but with the threat of avian influenza, anything that leads to immune suppression in chickens may now be an issue of human public health importance. The same factory farming conditions that likely facilitated the emergence of killer viruses like $H5N1^3$ have led and continue to lead to the emergence and spread of immunodeficiency viruses that may in turn facilitate the emergence of future highly pathogenic strains with human pandemic potential.

Conclusion

Genetic selection for productivity and the stressful, overcrowded, and unhygienic confinement of animals in industrial poultry production systems facilitate immune suppression in birds already bred with weakened immunity, offering viruses like avian influenza ample opportunities for spread, amplification, and mutation. Placing genetically un-diverse birds into these kinds of unsanitary environments with inadequate ventilation and sunlight exposure is believed to provide a ripe "breeding ground" for the emergence and spread of such diseases as virulent avian influenza—diseases with human public health implications.

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