Welfare Implications of Avian Osteoporosis

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ABSTRACT Cage layer fatigue was first noticed after laying hens began to be housed in cages in the mid-20th century. Hens producing eggs at a high rate were most susceptible to the disease. Early research revealed that cage layer fatigue was associated with osteoporosis and bone brittleness. Severe osteoporosis leads to spontaneous bone fractures commonly in the costochondral junctions of the ribs, the keel, and the thoracic vertebrae. Vertebral fracture may damage the spinal cord and cause paralysis. Osteoporosis appears to be inevitable in highly productive caged laying hens. The condition can be made worse by metabolic deficiency of calcium, phosphorus, or vitamin D. Hens in housing systems that promote physical activity tend to have less osteoporosis and rarely manifest cage layer fatigue. Genetic selection may produce laying hens that are less prone to bone weakness. The welfare implications of osteoporosis stem from pain, debility, and mortality associated with bone fracture. The chicken has well-developed neural and psychological systems specialized to respond to pain associated with trauma and inflammation. Although studies on the chicken have not focused on pain due to bone fracture, physiological and behavioral similarities to other species allow inference that a hen experiences both acute and chronic pain from bone fracture. There is little information on osteoporosis in commercial caged layer flocks, however, evidence suggests that it may be widespread and severe. If true, most caged laying hens suffer osteoporosis-related bone fracture during the first laying cycle. Osteoporosis also makes bone breakage a serious problem during catching and transport of hens prior to slaughter. Estimates of mortality due to osteoporosis in commercial caged layer flocks are few, but range up to a third of total mortality. Many of these deaths would be lingering and attended by emaciation and possibly pain. Osteoporosis-related bone breakage during processing has reduced the marketability of spent caged laying hens, contributing to the need to develop humane on-farm killing methods to support alternative means of spent hen disposition. Overall, the evidence indicates that cage layer osteoporosis is a serious animal welfare problem. A determined effort must be made to make the laying hen no longer susceptible to the harmful effects of excessive bone loss.

(*Key words*: avian osteoporosis, bone fracture, pain, mortality, animal welfare)

INTRODUCTION

Shortly after laying hens were housed in cages in the mid-20th century, a pathological condition was noticed that came to be called cage layer fatigue. As described by Couch (1955) and Grumbles (1959), cage layer fatigue was manifested as the hen being unable to stand but still being willing to eat and drink. Affected hens could become emaciated and die if unassisted, but most would recover in several days if placed on the floor. Young hens producing eggs at a high rate were most susceptible. Bone brittleness was characteristic of cage layer fatigue and many hens diagnosed with the condition had broken bones. Urist and Deutsch (1960) demonstrated that normal laying hens or chickens treated with exogenous estrogen developed osteoporosis, i.e., increased porosity and reduced thickness of bone cortex which caused their bones to become fragile. Osteoporosis developed while medullary bone grew in volume. Bell and Siller (1962) also noted that normal laying hens had thinned cortical bone and reduced trabecular bone, and that these conditions were accentuated in hens suffering from cage layer fatigue. Both bone types contribute to bone strength. These authors separated cases of cage layer fatigue into 2 types: peracute, in which hens died suddenly without prior symptoms, and acute, in which hens collapsed due to sudden paralysis of the legs. Hens suffering the acute form of cage layer fatigue were in danger of death, but if they survived a few days they could recover completely after a month or 2 of assistance with feeding and drinking. Bell and Siller (1962) also noted that some genetic lines of hens appeared to be more susceptible to cage layer fatigue than others.

Abbreviation Key: UEP = United Egg Producers.

Riddell et al. (1968) described the relationship between cage layer fatigue and bone pathology. Affected birds had fragile bones. Deformation of the keel and of the ribs at the costochondral junction was common due to fractures that caused the bones to fold. In surviving birds, these would heal to form permanent skeletal abnormalities. Most birds manifesting cage layer fatigue had fractured thoracic vertebrae. These fractures compressed the spinal cord, sometimes with bone fragments penetrating cord tissue, and resulted in spinal cord degeneration. Riddell et al. (1968) postulated that this spinal cord damage would produce the paralysis typical of cage layer fatigue. A number of birds found dead had extensive internal hemorrhages overlying compression fractures of the thoracic vertebrae. Hens with cage layer fatigue died of starvation if left in their cages. Of the 12 paralyzed birds set aside with readily available food and water, 3 died or were euthanized after failing to recover within 3 wk. The remaining 9 hens were able to stand in an average of 11 d (range: 2 to 21 d), although none showed a normal gait 14 d after first standing. Upon necropsy, these hens were found to have had breaks in the spinal column similar to the other birds examined in the study. The breaks had healed, but often with distortion of the vertebrae and pressure on the spinal cord. By contrast, even though fed a diet that led to a high rate of cage layer fatigue in caged hens, hens kept on litter did not develop paralysis typical of cage layer fatigue and had only minor rib and sternal deformation (Riddell et al., 1968).

Thus, it was evident very early that osteoporosis was associated with the underlying cause of cage layer fatigue. A great amount of subsequent research on bone strength and osteoporosis has confirmed many of the early observations and expanded our knowledge of factors affecting bone strength in laying hens. Osteoporosis has a number of implications for the welfare of commercial laying hens. These implications are addressed in the remainder of this discussion.

FACTORS AFFECTING BONE STRENGTH

It is beyond the scope of this paper to attempt to review bone biology and the etiology of osteoporosis in the chicken. Nonetheless, to properly understand the welfare implications of osteoporosis in the laying hen, it is necessary to review briefly how flock husbandry can influence bone condition in hens.

The beginning of sexual maturation coincides with medullary bone development and cessation of remodeling of structural bone, i.e., cortical and cancellous (or trabecular) bone, in the pullet (Whitehead and Fleming, 2000). A hen must absorb large amounts of dietary calcium to calcify eggshells when producing eggs at a high rate. Significant quantities of this calcium are stored each day in medullary bone, from which it is later released for calcification of eggshell at times when calcium is not available in the digestive tract (Etches, 1987). Medullary bone is formed at the expense of structural bone (Taylor and Moore, 1954; Simkiss, 1967). Structural bone resorp-

tion to supply calcium to remodel medullary bone without concomitant ability to remodel structural bone causes a hen to be predisposed to osteoporosis. Rennie et al. (1997) concluded that the modern hybrid laying hen is highly susceptible to osteoporosis, and that osteoporosis cannot be prevented during lay in this type of bird. The close association between high egg production and reduced bone condition was shown by observations that femur bone mineral content and tibial bone strength decline during the first few weeks of egg production (Cox and Balloun, 1971; Harms and Arafa, 1986).

Calcium

Inadequate dietary calcium leads to reduced bone mineral content and increased bone fragility in laying birds (Mehring and Titus, 1964; Douglas et al., 1972; Cheng and Coon, 1990a,b). These changes can develop quickly if the dietary calcium deficiency occurs when the hen has a high metabolic need for the element (Roland and Rao, 1992; Elaroussi et al., 1994). Low feed intake and high early egg production by modern commercial layers may raise the requirement for calcium inclusion in the ration beyond expectation in the first weeks of egg production, resulting in suboptimal performance and reduced bone strength (Roland et al., 1996). On a positive note, loss of bone mineral and bone strength early in production can be mitigated by including particulate limestone in the feed (Fleming et al., 1998).

Excessive calcium-related bone mineral depletion can also occur later in lay, as shown by Rowland and Harms (1970) who found that raising dietary calcium level for late-cycle egg-type hens increased tibial ash, with a numerical but not statistically significant increase of tibial breaking strength. In addition to reducing risk of structural bone resorption, the extra dietary calcium may have improved mineralization of tibial medullary bone, which can provide a modest contribution to bone strength (Fleming et al., 1996).

Feed withdrawal from laying hens, such as may be done to induce molt or to reduce feed costs at flock termination, imposes complete calcium deprivation and causes a very rapid decline in bone strength while hens remain in production (Savage, 1991; Newman and Leeson, 1999). Heat stress also can reduce bone integrity by affecting levels of circulating ionic calcium in the blood (Koelkebeck et al., 1993).

Phosphorus

Low dietary phosphorus during lay can lead to elevated incidence of cage layer fatigue, reduced bone ash, increased severity of osteoporosis, and diminished bone strength (Simpson et al., 1964; Riddell et al., 1968; Harms et al., 1990). It is possible, on the other hand, to reduce bone strength by feeding a hen too much phosphorus (Sohail and Roland, 2002). Inadequate dietary phosphorus at a critical stage of development, such as during initial modeling of medullary bone (Rao et al., 1995), can

have a long-lasting deleterious effect on bone integrity. During lay, late-cycle hens may be more sensitive to phosphorus deficiency than younger hens because structural bone volume and strength tend to be at a minimum at this stage (Whitehead and Wilson, 1992; Fleming et al., 1998; Cransberg et al., 2001; Sohail and Roland, 2002).

Vitamin D

Osteoporosis and reduced bone strength can be caused by vitamin D deficiency (Antillon et al., 1977; Frost et al., 1990). Frost et al. (1990) produced evidence suggesting that late cycle commercial layers can metabolize enough 1,25 dihydroxycholecalciferol from vitamin D_3 to maintain shell quality, but not enough of the metabolite to maintain tibial strength.

Housing and Exercise

The fact that cage layer fatigue suddenly appeared when hens were first housed in cages led early researchers to speculate that the underlying condition preexisted the disease, but did not manifest as a problem until hens were placed in cages (Grumbles, 1959). Bell and Siller (1962) followed with the suggestion that the severe osteoporosis seen in bones of caged layers resulted from disuse atrophy. An abundance of research has now shown that the type of housing in which laying hens are kept, e.g., floor pen, perchery, aviary, enriched cage, or unenriched cage, has an influence on bone strength (Rowland et al., 1968; Rowland and Harms, 1972; Meyer and Sunde, 1974; Harms and Arafa, 1986; Arafa and Harms, 1987; Knowles and Broom, 1990; Gregory et al., 1991; Wilson et al., 1992, 1993; Hughes et al., 1993; Fleming et al., 1994; Tauson and Abrahamsson, 1994; Barnett et al., 1997; Moinard et al., 1998; Newman and Leeson, 1998). In accordance with the fact that the condition and strength of a bone is dependent on the load-bearing activity it undergoes (Lanyon, 1992), those housing systems that foster the greatest physical activity produce hens with the strongest bones, and the parts of the skeleton made to do more work in a given housing design receive the greatest stimulation of bone strength.

Factors to Improve Bone Strength

Merkley (1981) found that hens provided Na fluoride in drinking water during the growing period had much stronger humeri and tibiae and greater tibial ash percentage than control birds at 45 wk of age. Fluoride supplementation during lay had no effect on bone strength. Injections of the bisphosphonate, alendronate, which is used clinically to prevent bone loss in humans, appears to prevent structural bone resorption in pullets during initial medullary bone modeling, but not during medullary bone remodeling after egg production has begun (Thorp et al., 1993; Wilson et al., 1998). Bone strength is a heritable trait (Bishop et al., 2000). The best potential to reduce problems associated with osteoporosis in the long run, therefore, might be through genetic selection.

Induced molting of laying hens is a common practice in some countries. An induced molt program causes hens to stop laying eggs and allows time for regression of the reproductive tract so that production performance is improved when the hens are re-stimulated to lay. Remodeling of structural bone can occur when a hen is not producing eggs. Since induced molting keeps hens out of production for several weeks, it is worth asking if the procedure alleviates osteoporosis. The net outcome of bone resorption and remodeling resulting from simple feed withdrawal for several days may not improve bone strength, at least in the short term (Newman and Leeson, 1999). However, molted flocks at the end of the second cycle of production may have stronger bones on average than unmolted flocks at the end of the first production cycle (Arafa and Harms, 1987; Gregory et al., 1991).

WELFARE IMPLICATIONS

It is clear that commercial laying hens are susceptible to structural bone osteoporosis due to their high sustained rates of egg production. It is also clear that osteoporosis can be made worse by insufficient intake of minerals essential for bone or eggshell formation or of nutrients essential for bone metabolism. This places a premium on good flock management because anything that disrupts the supply or intake of essential nutrients, for instance, errors in feed formulation or feed delivery, heat stress, illness, competition for access to feed, etc., can harm the bone condition of significant numbers of hens in a flock. Until primary breeders can produce stocks of layers with improved bone strength, the only feasible defense against bone fragility in commercial layer flocks is awareness of the problem and application of best management practices to minimize osteoporosis as much as possible.

Pain

Osteoporosis increases risk of bone fracture in laying hens, and one might presume that bone fracture is painful. However, to properly consider the welfare implications of osteoporosis, it is important to evaluate the ability of the chicken to experience pain.

Nociceptors, i.e., sensory receptors preferentially sensitive to noxious stimuli, have been identified in various parts of the chicken's body. A noxious stimulus is any event impacting an organism that is sufficiently strong to potentially cause tissue damage. The chicken's beak contains 3 types of nociceptors: mechano-thermal (polymodal) nociceptors associated with unmyelinated C-fibers having mechanical stimulation thresholds of 2 to 50 g and responsiveness to heat above 40°C, thermal nociceptors that respond to heat in a manner similar to the polymodal nociceptors but with no response to mechanical stimuli, and high threshold mechanical nociceptors (Gentle, 1989, 1992a). The high threshold mechanical nociceptors have response thresholds from 5 to 50+ g and are associated with C-fibers or small myelinated A-delta nerve fibers. The ankle joint capsule is well supplied with

C-fiber and A-delta mechanical nociceptors (Gentle, 1992b; Gentle and Thorp, 1994), and the skin of the leg possesses C-fiber mechanothermal nociceptors (Gentle and Tilson, 2000).

Two ankle joint inflammation models that have been used to investigate the sensory properties of nociceptors demonstrate increased nociceptor sensitivity due to inflammation. Injection of Freund's adjuvant into the joint capsule causes inflammation mimicking that caused by bacterial infection. For both C-fibers and A-delta fibers, it induces increased receptor field size, decreased response threshold to mechanical stimulation, and increased proportion of units responsive to joint movement (Gentle and Thorp, 1994). C-fibers, but not A-delta fibers, show increased receptor field size, decreased response thresholds, increased response to joint movement, and a high level of spontaneous activity during ankle joint inflammation induced by Na urate injection, which mimics gout (Gentle, 1997a). Both models, therefore, give evidence of hyperalgesia in response to inflammation.

The chicken manifests both active and passive behavioral responses to noxious stimulation depending on the type of stimulus. Comb pinch elicits wing flapping, vocalization, and escape attempts, whereas a 50°C thermal stimulus applied to the comb produces a motionless crouching posture with eyes closed and neck withdrawn (Woolley and Gentle, 1987). Oral stimulation of mouth lesions with pain-producing substances causes motionless crouching (Gentle and Hill, 1987). The adoption of passive behavior during sustained nociception appears to reflect a transition to a coping state enabling the bird to better deal with noxious stimuli that cannot be avoided. For instance, Gentle and Hunter (1991) found that sequential feather removal initially caused hens to be agitated, but thereafter, they adopted crouching immobility as feather removal continued. The hens' EEGs while in this immobile condition were high-amplitude low-frequency patterns similar to those typical of sleep, suggesting that they had entered a hypoesthetic state that reduced cognitive awareness of the noxious stimuli. Similarly, Na urate-induced ankle joint arthritis in caged hens causes one-legged standing or sitting (pain coping behavior), with sitting being associated with a drooping posture, immobility, and dosing, i.e., hypoethesia (Gentle and Corr, 1995).

Pain-related behavior may be controlled at different levels of the brain. For instance, pain coping behavior elicited by Na urate-induced ankle joint arthritis appears to be organized in the brain stem because it is performed by decerebrate chickens (Gentle, 1997b). Thus, the behavior can be produced without cognitive input. This may not be true of all pain-related behavior. An intact forebrain is necessary for development of pain coping behavior in response to ankle joint arthritis induced by Freund's adjuvant and for beak-guarding behavior after beak trimming (Gentle and Thorp, 1994; Gentle et al., 1997).

The chicken can exercise cognitive control over the expression of pain-related behavior. Different levels of pain coping behavior are produced in response to Na urate-induced ankle joint arthritis depending on the environment in which the chicken is placed (Gentle and Corr, 1995). The greatest pain coping behavior is shown if the bird is tested in a cage. Less pain coping behavior occurs if the bird is tested in a large pen, and marked hypoalgesia or complete analgesia is evident in hens tested in novel pens. Complete analgesia was also observed during pre-laying behavior. The remarkable ability of the chicken to suppress pain-related behavior in some circumstances, which is probably associated with suppression of pain perception, may be mediated by endogenous opioids (Wylie and Gentle, 1998). Gentle (2001) suggests that the cognitive control of pain and pain-related behavior involves shifting of attention away from noxious stimuli to other stimuli that evoke stronger motivations, e.g., novel, nesting, feeding, or social stimuli. The chicken is even able to exert cognitive influence on the development of peripheral inflammation, suggesting that there is a neurogenic component of inflammatory response that can be suppressed by attentional mechanisms (Gentle and Tilson, 1999).

Clearly, the chicken has complex neural and psychological mechanisms to facilitate, manage, and respond to nociception. In his review of pain-related behavior in the chicken, Gentle (2001) concluded that because of anatomical, physiological, and behavioral similarities between mammals and birds any consideration afforded to mammals in regard to pain should also be afforded to birds. Because animal welfare is a function of cognitive ability and self-awareness, species differences in these characteristics may cause animals to have different degrees of suffering in the same set of circumstances. Although interspecific comparisons of cognition relevant to the chicken are few, it appears that chickens do not rank high in cognitive ability among avian species (Pepperberg, 2001). Nonetheless, Gentle (2001) is justified in asserting that chickens are sensitive to pain and that this fact ought to bear weight in our consideration of their well-being.

An important welfare implication of osteoporosis in laying hens stems from the relationship of bone fracture to pain in the chicken. Unfortunately, the pain models studied in chickens have focused on pain-related issues other than broken bones, and I could not find literature addressing bone fracture pain in the chicken. Until such research is done, it would be wise to take Gentle's suggestion to see what can be inferred from studies of other species.

There is no doubt that osteoporosis-related bone fractures are painful in humans, with both acute and chronic manifestations of pain (Kessenich, 2000). Metabolically active sites in bones, such as bone marrow and osteochondral junctions of growth plates, are innervated in animals, but cancelleous bone is normally supplied with few nerves (Hukkanen et al., 1993; Nixon, 1994). Nociception from periosteum has been demonstrated in the cat, and it is from this tissue that bone-related nociception is thought to primarily originate (Mandsager and Raffe, 1991). In the rat, calcitonin gene-related peptide-immuno-reactive nerve fibers proliferate in the periosteum at the

site of a healing tibial fracture and in the fracture callus itself (Hukkanen et al., 1993). These nerve fibers appear to have a number of roles including support of the generation of pain. According to the authors, the acute sharp pain immediately associated with a bone fracture follows activation of afferent pain pathways due to trauma in tissues such as the periosteum. Subsequent experience of acute pain, tenderness, and chronic pain appears to arise from nociception associated with calcitonin gene-related peptide-immunoreactive nerve activity and hyperalgesia due to inflammation. This pain evidently motivates the animal to protect the fracture against excessive movement.

Until there is evidence to the contrary, it is reasonable to expect that the chicken experiences acute pain when a bone breaks, followed shortly by chronic pain associated with hyperalgesia of nociceptors in the periosteum and surrounding tissues due to inflammation. Further tenderness and chronic pain from nociception in the fracture callus and overlying periosteum probably would arise during healing. The degree of pain might depend on the size of bone fractured and the severity of the fracture, but this cannot be defined at present. Behaviorally, one might expect that a hen would produce an active response at the time of the fracture, such as vocalization or sudden bodily movement, followed thereafter by crouching and immobility when chronic pain develops. The first reaction would be unnoticed if the fracture occurred spontaneously in a cage, and probably would go unrecognized during the struggle associated with catching. The second stage of response involves behavior that is not unique to pain and could be difficult to identify in a cage or after catching.

Bone Fracture

There is little available information about how much osteoporosis actually occurs in commercial flocks of laying hens. This is unfortunate because ignorance may foster inadequate management of the problem, leading to reduced bird welfare and suboptimal production performance. Some evidence suggests that osteoporosis could be widespread in commercial flocks. Rennie et al. (1997) judged osteoporosis to be severe on average among layers of a commercial hybrid stock at the end of the first cycle of production, using a scale suggested by Whitehead and Wilson (1992) based on trabecular bone volume percentage. In all, 89% of the birds in the flock were determined to be osteoporotic. Cancellous bone volumes in late-cycle hens of another commercial stock were found to be in a range that indicated severe osteoporosis (Fleming et al., 1998). Cransberg et al. (2001) scored osteoporosis in laying hens based on nodulation and deformation at the costochondral junction of the rib cage and found that 80% of the birds examined were affected by osteoporosis by 42 wk of age. Thirty percent of the hens were judged to have severe osteoporosis. The skeletal deformity associated with osteoporosis results from bone fracture (Riddell et al., 1968).

From the review of pain-related behavior in chickens, hens suffering from osteoporosis-related bone fracture, e.g., rib fracture at the costochondral junction, keel fracture, or nonparalyzing vertebral fracture, probably have depressed activity in response to chronic pain. The time required for a bone fracture to heal is a function of the severity of fracture, the bone fractured, and the postfracture stability of the injured bone. In the domestic pigeon, the minimum time to form the first cancellous bridge in fractures of the radius or ulna is about 2 wk if the bones are fixed internally and about 3 wk with external fixation (Newton, 1977). Unstable fractures can take several weeks longer to achieve bone union. It is worth noting that in their second experiment Cransberg et al. (2001) recorded a decline in body weight and a drop in egg production that took 3 wk to recover among hens that were judged to have severe osteoporosis (at least 15% of the hens studied). This happened about the time hens in the flock would have reached minimum skeletal calcium reserves. Could suppressed activity due to fracture-related pain have led to reduced feed intake, causing the observed weight loss and depressed egg production?

Bone fragility also makes bone breakage a serious problem when caged layers are caught and handled prior to slaughter. Gregory and Wilkins (1989) reported from field studies that an average of 24% of hens housed in battery cages had at least 1 freshly broken bone after having been caught and removed from commercial houses by commercial catchers. The actual percentage varied widely among farms (range: 13 to 41%), suggesting that bone strength differences among flocks may have affected the incidence of bone fracture. The greatest prevalence of bone fracture occurred in the keel and ischium, indicating that hens tended to suffer impacts to breast and pelvic areas while being removed by the legs from cages. Many other bones were also fractured but at a lesser rate. Sequential events of handling add incremental increases to the total number of bone fractures. Gregory and Wilkins (1989) reported that the percentage of spent commercial layers with broken bones rose from 24% after catching at the house to 31% upon removal from transport crates at the processing plant, and then to 45% after hanging on the shackle line.

It stands to reason that minimization of impact forces on individual bones should reduce bone breakage during handling. Along this line, Gregory and Wilkins (1989) demonstrated that deliberate gentleness during catching of caged hens decreased bone breakage, but bone fragility still led to high incidence of broken bones, i.e., 14% (range: 7 to 29%). Grasping hens by both legs and removing them from cages one at a time reduced bone breakage by half or more (Gregory et al. 1993). The adoption of better procedures for commercial catching of caged layers appears to have lowered rates of bone breakage in the United Kingdom, but the bone fragility of end-of-cycle caged hens still led to bone breakage rates that were undesirably high, i.e., 14% of live birds at the processing plant (Gregory et al., 1994).

As would be expected, chickens with stronger bones incur fewer bone fractures during handling (Knowles et al., 1993; Gregory et al., 1994). Bone breakage during catching and processing tends to be less for hens housed in systems allowing greater opportunity for exercise, e.g., floor housing, free range, or perchery vs. cage systems (Meyer and Sunde, 1974; Arafa and Harms, 1987; Gregory et al., 1990; Moinard et al., 1998). Although some of this difference may be due to relative opportunity for impact on hard edges during capture, bone fragility is no doubt an important contributing factor because caged hens have greater bone breakage during processing when impact forces would be the same for all birds (Gregory et al., 1990). The difference in bone breakage during catching and handling between systems is consistent with literature cited earlier regarding relative bone strength in different types of housing. Although cage housing systems compare unfavorably to some other housing systems with regard to osteoporosis-related bone fracture, bone fragility is not the only predisposing factor for bone breakage. Hens in free range and perchery systems have been found to incur higher rates of complete fractures during the production cycle than hens housed in battery cages, presumably because the greater freedom of movement experienced by the free-range and perchery birds led to greater impact forces against hard objects (Gregory et al., 1990). Genetic selection has shown considerable potential to alleviate bone breakage in laying hens. In just 4 generations of divergent selection using an index based on bone radiographic density and bone strength, Bishop et al. (2000) produced lines of laying hens with significantly different incidences of bone fracture after capture and removal from cages.

The data cited above for end-of-flock bone breakage were based on European studies, and it might be expected that different methods of handling used in other parts of the world would produce somewhat different rates of bone breakage. However, there is no reason to believe that propensity for osteoporosis in modern stocks of commercial layers housed in cages would differ regardless of where the birds were located. Given common bone fragility, it is reasonable to expect that bone breakage rates in spent hens would be high during live haul and preslaughter handling in any nation with a commercial egg industry using battery cage systems.

Large numbers of hens are shipped from layer farms to processing plants each year. In countries where induced molting is not practiced, virtually all hens are replaced each year. In nations where molting is carried out, some fraction of the national flock is replaced each year depending on the percentage of flocks molted. In the U. S. in 2002, roughly 111 million spent hens were shipped to processing plants (hen disappearance statistics, J. Self, Cal-Maine Foods, Jackson, MS, personal communication). Even when a truck is run on smooth highways, the vibration produced in a chicken transport module would be fairly uncomfortable for a sitting human (Randall et al., 1993). Healthy uninjured domestic fowl find moderate floor vibration to be aversive (Rutter and Randall, 1993;

Duggan and Randall, 1994). Vibrational forces would exacerbate pain associated with a broken bone, especially if periosteal nociceptors have become hyperalgesic due to inflammation. In the U.S, transportation distances of 80 to 800 km are typical because processing plants are not necessarily close to commercial layer farms. Time in transit can be long. For instance, in a survey of processing plants in Ontario and Quebec, Canada, transportation times averaged 16 h for hens imported from the U. S., 18 h for transportation within the province, and 26 h for interprovincial transport (Newberry et al., 1999). The respective rates of birds dead-on-arrival were 2, 1.7, and 4%, respectively. Hens with broken bones presumably would be less able to withstand the physical forces and environmental stressors of transportation than would intact hens. This appears to be borne out by a European study in which 74% of caged layers that died in transit to the processing plant had broken wings or legs, which was a much higher injury rate than was the case for surviving birds.

Cage Layer Fatigue and Mortality

Another welfare implication of osteoporosis in laying hens is that hens suffering from cage layer fatigue generally die if unassisted, which would invariably be the case in a commercial battery cage environment. There is little information on the amount of mortality due to cage layer fatigue in commercial flocks. Increased mortality rates have been observed among hens on low calcium diets (Douglas et al., 1972; Hurwitz et al., 1975). Roush et al. (1986) reported an increased rate of hen mortality due to cage layer fatigue when dietary calcium was 2.5% of the ration. Rao et al. (1995) found that maturing pullets on reduced phosphorus diets had substantially increased risk of cage layer fatigue and mortality. Thus, cage layer fatigue mortality can result from nutritional deficiencies, but this tells us little about how much this mortality actually occurs.

Roland and Rao (1992) estimated that 15 to 30% of commercial layer mortality is related to osteopenia, but provided no supporting data. Van Niekerk and Reuvekamp (1994) examined mortality in flocks of commercial layers. They estimated that 13% of the total mortality of caged hens, i.e., about 1.1 of 8.2%, was caused by bone weakness. By comparison, mortality due to bone weakness of birds housed in an aviary was close to zero. McCoy et al. (1996) estimated that 35% of the total mortality in a caged commercial layer flock, i.e., about 3.4 of 9.7%, was due to cage layer osteoporosis. Mortality due to osteoporosis in caged layer flocks is probably variable and follows the severity of the condition in the flock. At times, however, it may constitute a considerable proportion of the total flock mortality. Although some cage layer fatigue deaths seem to be sudden, many deaths would be lingering and attended with emaciation and presumably some degree of pain.

Spent Hen Disposition

The bone fragility associated with cage layer osteoporosis can cause excessive bone breakage during processing (Gregory and Wilkins, 1989; Savage, 1991). Bone fragments can penetrate the meat and create quality and food safety problems. Partly for this reason a diminishing number of poultry processing plants in some countries, such as the United States, have been willing to accept spent commercial laying hens for processing. This loss of market has forced many egg companies to find other ways to dispose of flocks. For instance, in the U.S. in 2002, about 58 million spent hens were sent to renderers or put to other nontraditional uses (hen disappearance statistics, J.Self, personal communication). These alternate uses require that the hens be killed on the farm. Unfortunately, when egg companies began to kill hens on farms, no humane method existed for on-farm killing of spent hens on the scale necessary to depopulate a modern commercial layer house. A feasible method had to be developed to help the egg industry avoid a serious animal welfare problem stemming from the improper killing of hens on farms. To provide such a method, Webster et al. (1996) designed the modified atmosphere killing system to meet essential standards for efficiency and animal welfare. In the United States, the modified atmosphere killing system has been recognized by the United Egg Producers (UEP) animal husbandry guidelines as an appropriate means to kill large numbers of hens on farms (UEP, 2003). The membership of the UEP comprises most U. S. commercial egg companies, and most of these have currently joined the organization's Animal Care Certification program, which is based on the UEP animal husbandry guidelines. However, until such time as all egg companies that kill flocks on farms use a humane method to kill each bird, on-farm killing of spent hens will remain an animal welfare issue at least partially attributable to osteoporosis.

CONCLUSIONS

The data suggest that osteoporosis in commercial layer flocks has serious animal welfare implications. It is imperative that the problem of osteoporosis in caged laying hens be solved as soon as possible. If the percentages and severity of osteoporosis reported by several authors are representative, most caged laying hens suffer some kind of bone fracture during their first laying cycle (e.g., fracture of the ribs, keel, or vertebrae). The chicken has complex neural and psychological mechanisms to sense and respond to painful stimuli, and it is probable that bone fractures are painful to chickens. Bone fractures may take several weeks to heal. Osteoporosis-related bone fractures result in skeletal deformities that are determined by the severity of the fractures. The brittle bone condition typical of spent commercial layers at the time of flock removal can lead to high rates of bone breakage during catching and handling. Hens with fresh bone fractures have reduced ability to cope with the stress of transport and are more likely to die in transit. The amount of mortality that

occurs in flocks of commercial laying hens due to cage layer fatigue is not well understood. The rate probably varies among flocks, but has been estimated to account for up to a third of the total mortality. Many deaths due to cage layer fatigue would be slow and perhaps attended with pain due to fracture of thoracic vertebrae. Bone fragility has contributed to loss of market for end-of-cycle commercial laying hens, creating need to kill flocks of spent hens on farms so that alternative markets can be accessed. The on-farm killing of spent hens will remain an animal welfare implication of osteoporosis until all egg companies that kill flocks on farms adopt a humane killing procedure. At present, little can be done to avoid osteoporosis in laying hens housed commercially in cage systems other than to maintain best husbandry practices so as to not exacerbate the problem. However, genetic selection for bone mass and strength has shown promise in the long run to alleviate osteoporosis in caged laying hens.

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