
Welfare Implications of Injurious Pecking in Laying Hens

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Modern layers descend from the red jungle fowl, a species with a highly diverse diet including fruits, seeds, herbage, insects and even small vertebrates (Savory et al. 1978) (Figure 4.1). Despite over 8,000 years of domestication (West & Zhou 1988), there is no evidence that their exploratory and feeding habits have substantially changed. Similarly to the jungle fowl, which spend about 60% of the active day ground pecking and 34% scratching (Dawkins 1989), modern laying hens, when given the opportunity, spend most of their daylight hours foraging (Nicol 2019). They scratch the ground with their claws and peck it vigorously to explore and manipulate possible edible items, making about 14,000-15,000 pecks at food items and other objects in the course of a day - nearly 1,000 pecks per active hour (Webster 2002).



Figure 4.1 Plate “Red junglefowl”, painted by G.E. Lodge (courtesy of the Smithsonian Libraries, Washington, D.C)

Under some commercial housing conditions, however, the strong motivation to forage and ground peck is not fulfilled, giving rise to the emergence of so-called abnormal behaviours, or behaviors that serve no function and are not as prevalent in their wild counterparts. One such behavior is injurious pecking, an umbrella term used for pecking behaviors that are re-directed towards flock mates, in which the target are their feathers (commonly referred to as ‘feather pecking’) and tissues (in this case, the terms ‘tissue pecking’ and ‘cannibalistic pecking’ are used, or ‘vent pecking’ when the targeted area is the vent) (Figure 4.2). Injurious pecking occurs to varying degrees in all housing systems and compromises the welfare of affected birds in multiple ways, as we discuss in this chapter.



Figure 4.2. Laying hens with bald and wounded skin patches as a result of injurious pecking. Illustration: Olga Kurkina.

There are different forms of feather and tissue pecking, each with different consequences for the affected bird's welfare (Rodenburg et al. 2013). 'Gentle feather pecks' are highly prevalent during both the rearing and laying periods (Nicol et al. 2013) and consist of soft pecks to the tips of the feathers of conspecifics which do not result in much damage and are often ignored by the recipient. 'Severe feather pecking', in turn, involves forceful pecks and pulling of feathers from victims, especially from their back, vent and tail (Rodenburg et al. 2013). Severe feather pecking is painful and results in various negative outcomes for affected birds (Figure 4.3). Although severe feather pecking does not show the perseverance typical of a stereotypy (Kjaer et al. 2015), when repeatedly performed it can lead to bald patches where the skin of the victim is exposed (Figure 4.2). Tissue pecking is referred to as such when pecking continues on the bald skin, which can lead to wounds and secondary infections. Bleeding of skin and underlying tissues, and the exposition of mucosa, leads to the exacerbation of pecking, as birds are attracted to pecking at the color red, with some birds ultimately being pecked to death¹.

¹ Although cannibalism is also observed in wild birds (Caro et al. 2014; Neves et al. 2015), it is often associated with food shortages, serving a nutritional function.

Vent pecking is another common form of tissue pecking, particularly at the onset of laying. During oviposition, the cloacal mucosa becomes visible while the egg is expelled. If the mucosa is damaged in some way (e.g. by the environment or its tearing during the expulsion of the egg), it can swell and remain outside the cloaca, becoming an attractive target for peckers, who target the vent of the victim and in some cases pull out inner organs. Despite appearances, both severe feather pecking and tissue pecking do not stem from aggression, but rather from the re-directed expression of foraging and feeding habits.

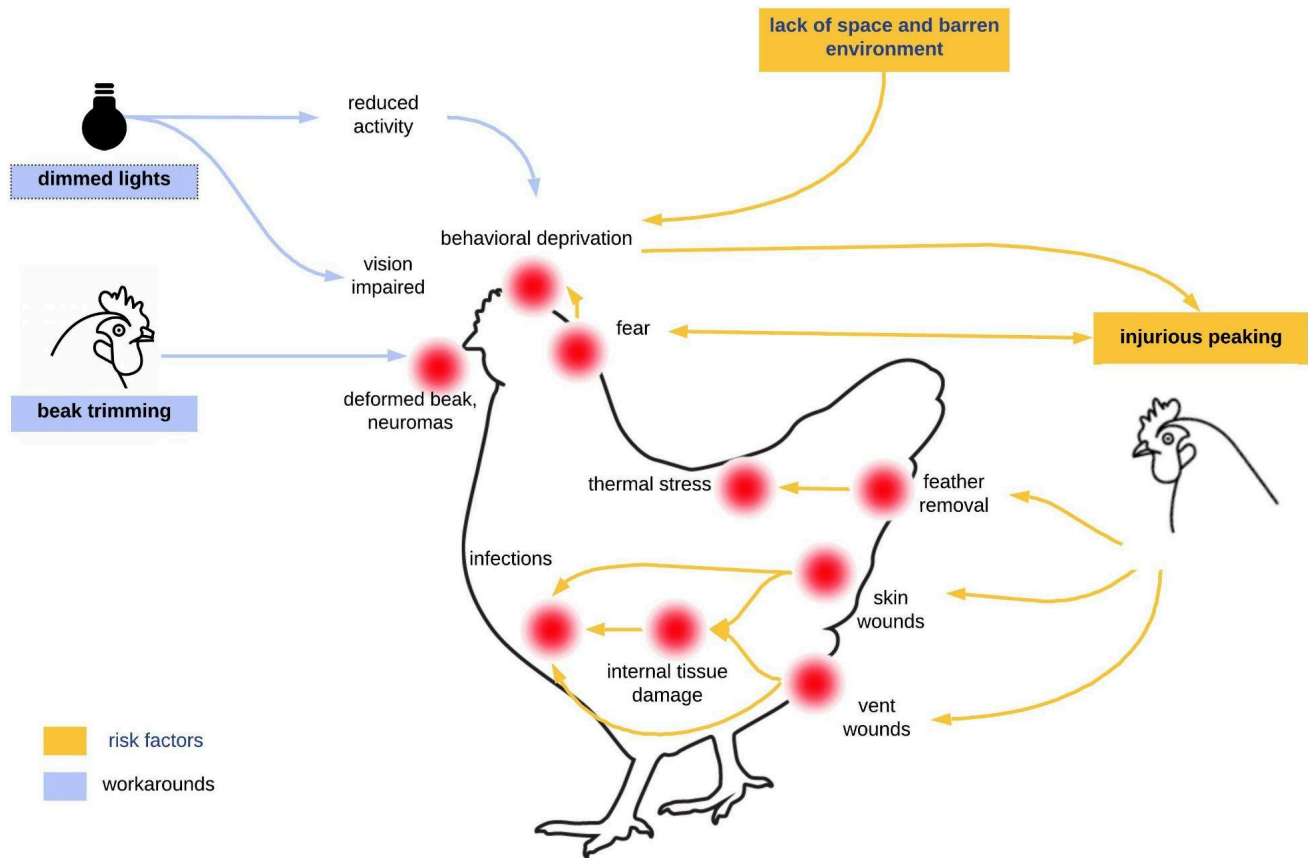


Figure 4.3. Summary of possible direct and indirect effects of injurious pecking on the welfare of laying hens.

Injurious pecking has multiple consequences for the welfare of impacted birds (Figure 4.3). In addition to the physical pain associated with feather removal (Gentle & Hunter 1991), with the resulting wounds and with fatal cannibalistic attacks, feather pecking also leads to plumage loss. In hens with poorer feather coverage, the ability to thermoregulate is impaired and energy requirements are increased, ultimately increasing the likelihood of cold stress. Moreover, extreme cases of cold stress can be fatal, particularly during transport.

Besides its direct effects, injurious pecking has also been associated with greater stress responses (Kjaer & Jørgensen 2011; Rodenburg et al. 2009) and greater risk of diseases such as egg peritonitis

and infectious bronchitis (Pötzsch et al. 2001). However, it is not yet clear whether injurious pecking is caused or exacerbated by these factors. Similarly, there is a positive association between injurious pecking and fearfulness (Campo et al. 2008), though the relation between feather pecking and fearfulness is complex and not completely elucidated to date. For example, victims of feather pecking may become more fearful, as illustrated in the following passage: “[victimized birds] *are poorly-feathered, underweight or emaciated birds which can be found hiding in corners or under nest-boxes. They appear to be highly fearful of other birds to the extent that they spend most of their time in partial isolation, usually crouching in a timid manner with the head lowered or retracted and the back facing the other birds. Generally they avoid contact with flockmates, but when they move amongst other birds to reach a feeder or drinker they often scurry with the head down, and this seems to incite pecking and mobbing. The fear that these birds develop can be severe enough to result in emaciation, dehydration and death*” (Gregory 2004). This is consistent with evidence suggesting that victimized birds are less likely to access feeders and drinkers (Nicol et al. 2013), and generally (though not always) score worse for behavioral and physiological indicators of fear and stress (Campo et al. 2008; Hughes & Duncan 1972). On the other hand, there is also strong evidence to suggest that fearful birds are themselves more likely to initiate feather pecking (Rodenburg et al. 2004; Rodenburg et al. 2010).

Other indirect effects of injurious pecking stem from the very interventions used to control it (‘humans workarounds’ in Figure 4.3). Beak trimming is the main practice adopted in the majority of commercial systems to reduce the damage caused by injurious pecking (Riber & Hinrichsen 2017). However, it is a painful procedure with important negative effects on hen welfare (Gentle 2011; Marchant-Forde et al. 2008; Gentle 1986). Another widely used intervention to control outbreaks of feather pecking is the reduction of light intensity to levels below five lux (Coton et al. 2019; Green et al. 2000), or the use of specific wavelengths (e.g. red). Low lighting affects hen welfare in several ways. In darker environments overall activity levels are reduced, along with the expression of visually-mediated behaviors such as foraging (Prescott et al. 2003). Low activity is also associated with osteoporosis and a high risk of bone breakage, particularly during depopulation and transport (Chapter 7 (Schuck-Paim & Alonso 2021b)).

Although the underlying causes and risk factors for injurious pecking have been extensively studied (Rodenburg et al. 2013) (Box 4.1), knowledge on the physical pain and distress suffered by its victims is still limited, as will become clear in the next sections. We address this gap by proposing a number of initial hypotheses on the temporal evolution of the pain associated with the direct physical consequences of injurious pecking (indirect effects, such as fear, are discussed in other chapters), as follows: (1) the physical pain of feather removal; (2) the pain associated with the damaged skin (skin and vent wounds, infected or not) and (3) the pain and time course of fatal cannibalistic attacks.

Here, we highlight the request of collaboration made to the reader in the preface of the book. As it will become clear, the data needed to estimate the temporal evolution of the pain resulting from pecking injuries is extremely scarce. While we tried to bring separate pieces of information together to make the necessary inferences, these estimates would greatly benefit from more empirical data and further expert input. We start by describing the process and time course of skin wound healing in birds, and the specificities of the behavioral responses of victims of severe feather pecking to substantiate the hypotheses proposed in each of the Pain-Tracks presented.

Box 4.1. Common Risk Factors for Injurious Pecking

Injurious pecking has a multifactorial origin. Below is a non-exhaustive list of some risk factors.

- ☐ Environments that restrict or limit behavior, especially foraging opportunities.
- ☐ Insufficient access to resources, including feed, water, perches or nesting space.
- ☐ Laying onset before 20 weeks of age.
- ☐ Overs-selection for (the single trait) high egg production.
- ☐ Stress in parent lines.
- ☐ Genetic differences in feather pecking propensity.
- ☐ Bright lights.
- ☐ Frequent dietary changes.
- ☐ Dietary deficiencies.
- ☐ Heterogeneity in flock color or size.
- ☐ Excessive heat.
- ☐ Abrupt changes in environment or management practices.
- ☐ Allowing sick, injured or dead birds to remain in the flock.

The avian skin and wound healing

Nociceptors sensitive to both thermal and mechanical stimulation have been identified in various avian species, and in multiple sites in the chicken (Gentle et al. 2001). In general, the physiological responses of nociceptors in birds parallel those in mammals, with similar discharge patterns, response thresholds and sensitization following inflammation (Gentle 1997).

The pain from wounds inflicted by tissue pecking can be caused by skin damage, nerve damage, blood vessel injury, infection and ischaemia (a restriction of blood supply to tissues) (Frescos 2011). To better understand the likely intensity of the pain endured following a cut or laceration, such as that caused by tissue pecking, we explore the process and estimate the time course of wound healing. Often, it progresses in predictable stages, as follows (Mickelson et al. 2016):

(1) Hemostasis (or coagulation) phase: lasting from 5 to 15 minutes (Mickelson et al. 2016), this phase is characterized by vasoconstriction, the activation of the clotting cascade, and the release of proinflammatory cytokines, which are signaling proteins produced predominantly by activated macrophages to recruit healing and repair cells. Their release promotes the onset of the inflammatory stage.

(2) Inflammatory and debridement phase: the acute inflammatory response usually lasts for 24 to 48 hours, but may persist for up to two weeks in a poor wound environment (Mickelson et al. 2016). Often, the highest levels of proinflammatory cytokines arise from 12 to 24 h after wounding (Chen et

al. 2016). This phase is characterized by increased vascular permeability and infiltration of neutrophils, macrophages, and lymphocytes into the wound. Here, the classic signs of inflammation, such as heat, redness and swelling, are observed. Swollen tissues push against sensitive nerve endings causing pain. Proinflammatory cytokines can also act directly on the nociceptive sensory neurons, changing their excitability and inducing their persistent sensitization (hyperalgesia, i.e. enhanced sensitivity to pain).

(3) Proliferative (repair) phase: if no further damage is provoked, the wound is rebuilt with new tissue, a process that may last up to three weeks. In this phase, the wound becomes progressively smaller as new tissues are built until the wound edges meet.

(4) Maturation (remodelling) phase: generally, remodeling can last for weeks to months after injury. In this phase, the newly formed tissue is strengthened and remodeled. Inflammation is substantially reduced, and pain is unlikely.

Anecdotal reports from backyard producers who followed more closely the wound healing process of hens suggest a period of about three to six weeks to (visual) healing completion. However, in addition to differences in wound care² (absent in commercial production), the time course and progression of wound healing will be affected by multiple factors, such as infection, mechanical factors, motion and the presence of debris (e.g. dirt or other foreign materials). Such factors are likely to lead to an intense inflammatory reaction that will prolong the inflammatory phase and pain in commercial settings.

Pain Associated with Feather Removal

In this section we analyse only the pain directly associated with the removal of feathers³. Severe feather pecks usually occur as a single event, or at maximum twice in a row, but will not occur in bouts (Daigle et al. 2015). Therefore, the pain associated with feather removal is determined for a single feather removal event. A tentative hypothesis for the time course and intensity of the pain associated with feather removal is proposed in Pain-Track 4.1, and justified next.

Pain-Track 4.1. Hypothesis on the time course and intensity of the pain associated with feather removal (for the definition of intensity categories, see Box 1.2 in Chapter 1 (Alonso & Schuck-Paim 2021)).

	Pulling	Recovery	Inflammatory reaction
Excruciating			
Disabling	90%		
Hurtful	10%	70%	
Annoying	0%	30%	50%
	1-5 sec	30-105 sec	10-30 min

² Such as antiseptic treatment and the separation of injured hens from other flock mates.

³ Severe feather pecks (SFP) may or may not result in the removal of a feather. Unfortunately, information on how many severe pecks a victim receives on average for each feather that is removed is not available. In Chapter 8 (Schuck-Paim & Alonso 2021c), we tentatively estimate that about 2 to 30 SFPs are needed for every feather removed.

In an experiment where feathers were removed from White leghorn chickens (Gentle & Hunter 1991), initial feather removal resulted in an agitated response, including wing flapping, vocalisation and concomitant changes in heart rate, blood pressure and electroencephalogram arousal, indicating the painful nature of feather removal. The removal of back or tail feathers (those typically removed by feather pecking) was more likely to produce agitation and vocalisation than those removed from other areas (Gentle & Hunter 1991). This is to be expected, as the pulling force required to remove a feather (from 400 g up to 4 Kg) is substantially greater than that needed to activate the mechanothermal and high threshold mechanical nociceptors in the skin (Gentle & Hunter 1991). The behavioral (e.g. vocalization, escape attempts, wing flapping) and autonomic changes that accompany feather removal (e.g. heart rate, blood pressure, electroencephalogram arousal) suggest that the immediate pain associated with it represents more than an annoyance to the bird, one that is very short-lived, lasting only a few seconds, but acute (at the disabling level). In the experiment described, heart rate took approximately 100 seconds to return to baseline following the removal of a feather (hence the total maximum duration of 110 seconds in the Pain-Track). Feather removal also causes an abrupt increase in blood pressure, reaching its peak 5 seconds after the removal (within the ‘pulling phase’), and then returning to normality about 30 seconds later (Gentle & Hunter 1991). We assume that in the 1-2 minutes following the plucking of a feather, pain gradually subsides.

The duration of the pain described in Pain-Track 4.1 is determined by the hemodynamic changes and the observed behavior pattern in the experiments described (Gentle & Hunter 1991). However, the feather follicle in the bird's skin is highly innervated and when the feather is plucked, it becomes inflamed and may even bleed. This raises the possibility that the local inflammatory process is still painful after the recovery phase, when hemodynamic and behavioral changes are no longer observed. As described in Chapter 1 (Alonso & Schuck-Paim 2021), pain at the Annoying level may not visibly affect the performance of normal activities and, in humans, is not always accompanied by changes in heart rate or blood pressure. Therefore, we consider a 50% possibility that the individual still feels pain (of an Annoying nature, given the small area and local nature of the damage) for 10 to 30 minutes, corresponding to the putative duration of the inflammatory reaction to the tissue caused by plucking.

Pain associated with skin wounds

Here we explore the potential time course of the pain associated with small wounds (<2cm), as this type of lesion has been shown to be more common (e.g. 4-8 times (Blatchford et al. 2016)) than larger ones. From this basic Pain-Track, the pain associated with more severe wounds can be derived as needed.

Pain-Track 4.2 Proposed temporal evolution of the pain associated with a skin wound (<=2 cm) that heals spontaneously (for the definition of intensity categories, see Box 1.2 in Chapter 1 (Alonso & Schuck-Paim 2021)).

	Wound infliction Point of tissue rupture	Hemostasis (coagulation)	Acute inflammation	Inflammation + proliferation
Excruciating				
Disabling	50%	20%		

Hurtful Annoying	50%	80%	70%	20%
			30%	60%
	0.5 - 2 min	5 - 15 min	0.5 - 2 days	1 - 2 wks

Pain-Track 4.2 is divided into five phases, named after the wound healing phases described in the previous section. At the point of skin rupture, there is often a short-lasting, but abrupt reaction by a feather-pecked bird, compatible with the Hurtful and Disabling categories of pain. This is expected from an evolutionary perspective, given the adaptive value of preventing further tissue damage very rapidly. The painful nature of tissue rupture is also confirmed by the observation that cutaneous nociceptive stimulation triggers an increase in blood pressure with an amplitude and duration similar to that recorded following the forceful removal of feathers (Woolley & Gentle 1987). In the following phases we assume that the pain progressively subsides. During the inflammatory phase we propose that the intensity of pain does not reach disabling levels given the local nature and small size of the wound. After the first 12-48 hours, coinciding with the observation of the highest levels of proinflammatory cytokines, pain should subside further until it is predominantly of an annoying nature. We assume that pain will last as long as there is residual inflammation (one to weeks), hence less than the entire proliferation phase (which should last about three weeks; section *'The avian skin and wound healing'*). We assume no pain in the maturation phase given the absence of inflammation (or very reduced levels).

Next, we consider the case where the wound gets infected. In this case infection is local and resolves naturally. While uninfected wounds will gradually improve until fully healed, infected wounds are painful for greater lengths of time, with both swelling and heat sensations in the affected area. In addition, infected wounds that are not treated will take longer to heal. Therefore, in Pain-Track 4.3 we increase the intensity, as well as the duration of the inflammatory phase relative to that proposed in the previous Pain-Track. These values should be revised as knowledge becomes available.

Pain-Track 4.3. Proposed temporal evolution of the pain associated with a skin wound (<2 cm) that becomes infected, but infection resolves spontaneously.

	i. Wound infliction Point of tissue rupture	ii. Hemostasis (coagulation)	iii. Acute inflammation (infected)	iv. Inflammation + proliferation (after infection)
Excruciating				
Disabling	50%	20%		
Hurtful	50%	80%	100%	30%
Annoying				70%
	0.5 - 2 min	5 - 15 min	2 - 3 days	2 - 3 wks

Pain associated with vent wounds

To explore the pain associated with vent damage, we first consider the description of how severe typical vent wounds present. The following is a description from a commercial farm in Sweden (Yngvesson et al. 2004): *“In this paragraph we describe typical types of wounds observed at catching... Victim number 4... represented a typical attack at the cloaca. The left half of the upper cloacal lip was gone and the middle of the wound was just above the cloaca on the left. In total the wound was approximately 3.5 cm in diameter, swollen, with dried blood, a haematoma and uric acid... The second typical attack at the cloaca was combined with a prolapse. Victim bird ... was 28 weeks old at catching. From the upper cloacal lip, 10 mm 3 mm of the tissue was missing and the edge around the cloaca looked like a ‘flower’ because it was pecked all around. The ring musculature appeared to be destroyed and what was left was ‘loose’ and the cloaca looked open. The cloacal tissue was prolapsed (extended about 30 mm outside the body). She had uric acid mixed with blood coming out of the cloaca.”* .

Vent wounds are often more severe than those in other parts of the body. They are larger, deeper, and may even involve loss of tissue. Accordingly, the case fatality ratio of vent-pecked hens (~30%) is substantially greater than that associated with tissue damage (~1%) in other parts of the body (Chapter 8 (Schuck-Paim & Alonso 2021c)). The hypothesis proposed in Pain-Track 4.4 considers, therefore, a wound of approximately 3-4 centimeters, with part of the cloacal lip missing.

Pain-Track 4.4. Proposed temporal evolution of the pain associated with a vent wound that heals spontaneously.

	Wound infliction Point of tissue rupture	Hemostasis (coagulation)	Acute inflammation	Inflammation + proliferation
Excruciating				
Disabling	100%	100%	60%	5%
Hurtful			40%	70%
Annoying				25%
	15 - 75 sec	5 - 15 min	2 - 3 days	2 - 3 wks

To the authors knowledge, no published description of how many pecks a victim typically receives during vent pecking attacks, or the duration of these attacks, is available to date. Therefore, we have made necessary assumptions based on the damage type descriptions available in the Swedish farm, estimating a range of 3 to 6 pecks during the infliction of wounds, spaced by 2 to 15 seconds (uncertainty range), during which the pain is sharp and acute (of a disabling nature). Given the larger extent of the damage, we assume that pain intensity is kept at a Disabling level in the minutes immediately after the injury (hemostasis phase). Swelling can be substantial, pushing against sensitive nerve endings, which can result in sustained pain at a greater intensity level. Even when vent-pecked birds do not endure subsequent attacks, vent injuries are disrupted on a daily basis by the process of egg laying, which likely slows healing and causes peaks of pain.

Indeed, vent pecking has been associated with decreased egg production, which may be explained by reduced feeding behaviour following pain in the victims (Prunier et al. 2013). Pullets with fresh vent wounds are also more stressed and fearful, exhibiting greater heterophil to lymphocyte ratios (a

measure of stress), and longer tonic immobility durations (Campo et al. 2008), a classic metric of fearfulness. Thus, we assume that the acute inflammatory process (during which pain is likely more intense) persists for two to three days, and it may take up to 3 weeks for inflammation to completely subside. A longer period is not assumed given evidence that survivors of vent pecking “*recovered and had no visible external wounds (although some had chronic scars) after 3 weeks*” (Yngvesson et al. 2004). The intensity of the pain during the last phase is most likely of a Hurtful nature, compatible with the observation of reduced feeding due to pain.

Next, we consider the time course and intensity of a vent wound that becomes infected and resolves spontaneously without treatment. The hypothesis proposed is illustrated in Pain-Track 4.5. The same rationale that we applied to infer the temporal evolution of pain in the case of an infected skin wound is applied here.

Pain-Track 4.5. Proposed temporal evolution of the pain associated with a vent wound that becomes infected, but infection resolves spontaneously.

	Wound infliction Point of tissue rupture	Hemostasis (coagulation)	Acute inflammation (infected)	Inflammation + proliferation (after infection)
Excruciating				
Disabling	100%	100%	60%	20%
Hurtful			40%	70%
Annoying				10%
	15 - 75 sec	5 - 15 min	2 - 3 days	2 - 4 wks

Finally, we consider the case of a pecked bird that survives a cannibalistic attack, but the wounds provide a route for infection that becomes systemic, leading to septicemia and death (Pain-Track 4.6).

Pain-Track 4.6. Proposed temporal evolution of the pain associated with a vent wound that becomes infected, leading to sepsis and death.

	Wound infliction Point tissue rupture	Hemostasis (coagulation)	Acute inflammation	Sepsis (1st symptoms)	Severe sepsis (organ failure)	Septic shock
Excruciating					30%	
Disabling	100%	100%	60%	90%	40%	
Hurtful			40%	10%	30%	80%
Annoying						20%
	15 - 75 sec	5 - 15 min	3 - 4 days	0.5 - 1 days	5 - 10 hrs	2 - 4 hrs

When infected wounds do not heal, ascent of the bacteria to internal organs can happen and can lead to sepsis. In Chapter 5 (Schuck-Paim & Alonso 2021a), we discuss the clinical symptoms and evolution of sepsis, only briefly summarized here. As with other welfare challenges, there seems to be no

published studies on the clinical evolution of sepsis in poultry. This lack of information is particularly surprising in light of the observation that septicemia is typically the main cause for carcass condemnation in broiler processing plants in the U.S. (Kentucky University n.d.). Based on research in animal models (mice), death due to sepsis, including the development of severe sepsis and septic shock, is estimated to take 24 to 48 hours, from the onset of symptoms until death following septic shock.

In animal models of sepsis, systemic and local inflammation is often regarded as a major source of pain (Carpenter et al. 2019). In mice, the first signs of sepsis include hunched posture, piloerection, lethargy, and diarrhea. The Centers for Disease Control and Prevention lists “*extreme pain or discomfort*” as a potential sign of sepsis, with an estimated 70% of patients developing polyneuropathy and neuropathic pain (Carpenter et al. 2019). Intravenous morphine is routinely used for relief, giving an indication of the level of pain intensity endured. Systemic inflammation has been also linked to generalized hyperalgesia (Carpenter et al. 2019), hence the proposed increase in the likelihood that pain is experienced at a Disabling level in phase iv of Pain-Track 4.6.

The next stage, often referred to as severe sepsis, starts when organs begin to malfunction. Sepsis-associated inflammation has been linked to pulmonary, cardiovascular, renal, haematological, hepatic and neurological dysfunction. In broiler chickens, septicemic carcasses are frequently associated with hemorrhages on the heart, liver, kidneys, muscles and serous membranes. The kidneys, liver and spleen are often swollen and contain excess blood. Muscles are also compromised, as illustrated by this passage: “*Very few birds with septicemia survive catching and transportation to make it to the processing plant alive. If the bird prior to catching has had a septicemia event, often the muscles have wasted away and have broken down because of changes in muscle metabolism*” (Russell 2012). In humans, this stage is marked by severe muscle pain and pain described as extreme (“I feel like I might die”) (Rees 2018; MacGill 2018). Thus, we consider a 30% probability that pain, at this stage, is felt at the Excruciating level. These estimates may be also understood as the probability that, during this phase, pain is experienced at the Excruciating level 30% of the time. That said, one of the consequences of severe sepsis may be neurological dysfunction, with some individuals experiencing reduced pain perception, as sepsis-associated encephalopathy is implicated in brain damage of regions related to nociceptive modulation. Because the resulting decrease in cognitive and nociceptive sensitivity may attenuate pain responses (Nemzek et al. 2008) in advanced stages of severe sepsis, we attribute a 30% probability to a less intense (Hurtful) category.

In the final phase before death (septic shock) symptoms of severe sepsis are still present, as well as very low blood pressure (Hotchkiss et al. 2016). At this stage, systemic inflammation is believed to profoundly alter the local synaptic circuitry, resulting in reduced nociception (Cazuza et al. 2020). Additionally, sepsis can also lead to a reduction in the perception of pain due to the release of endogenous analgesics (Gentle & Corr 1995). Accordingly, we consider that pain is more likely felt at lesser intensity levels during this final phase.

Pain from Fatal Cannibalistic Attacks

Tissue pecking can ultimately result in victim death due to excessive blood loss and serious tissue damage. In the case of vent pecking, which is the most common cause of fatal cannibalism in hens, pecking starts at the cloaca, surrounding skin and tissues, and can progress to evisceration and death. We refer to fatal tissue pecking events as fatal cannibalistic attacks.

Pain-Track 4.7. Proposed temporal evolution of the pain associated with vent pecking leading to a fatal cannibalistic attack.

	Initial pecks (before immobility)	Pecking attack (immobile phase)
Excruciating		30%
Disabling	60%	40%
Hurtful	40%	30%
Annoying		
	0.3 - 4 min	10 - 20 min

Unfortunately, ethological information on cannibalistic attacks is extremely scant - we were unable to find any formal description of these attacks, such as information on their duration, time course, or how long a victim takes to die or become unconscious. Therefore, we base our assessment on the following descriptions: (1) *“When attacking a victim, the cannibal typically approached from behind with the head slightly lowered. The pecks were vigorous and directed at the wound. The pecked bird usually vocalised and moved away, or was aggressive towards the pecking bird. If the pecked bird did not escape or show aggression, the cannibal might continue to peck and, if the victim moved, she was often followed”* (Yngvesson et al. 2004); (2) *“Cannibalism has been observed as starting with the pecking of fresh wounds in the back or excretory channels of a hen by nearby birds. The attacked birds are usually silent and make little attempt to escape. Pecking often continues until part of the intestine is obtained. Death follows, usually in 10 minutes, but pecking of the corpse continues”* (Anon n.d.).

These descriptions are different in that, in the first, victims vocalize vigorously and move away, whereas in the second description attacked birds make little attempt to escape. Leaving aside differences in individual personalities (modulated by genetic and/or environmental factors), we assume here that targeted birds initially react to the attackers (i), but then surrender if the attack continues. Chickens are known to react to a number of fear- and pain-eliciting stimuli by going into a state of complete motor paralysis, and it is not different with feather and tissue pecking events. In the experiment described previously (section ‘Pain Associated with Feather Removal’), where feathers were removed from White leghorn chickens (Gentle & Hunter 1991), successive feather removals led hens to crouch with the tail and head lowered in a sustained immobile state. The response observed in this experimental setting parallels that observed both in more natural settings and in commercial conditions, where victims of pecking bouts by flock mates are observed to go into crouching immobility or make little attempt to escape.

There is some evidence that chickens may experience reduced pain while in this paralysis state (Gentle 2011). In the experimental setup described (Gentle & Hunter 1991), the electroencephalogram showed a characteristic high-amplitude low-frequency (HALF) activity during this period of immobility similar to that seen in sleep and in catatonic states like tonic immobility (see Box 4.2). A similar pattern has been reported for general anesthesia in humans, where a progressive increase in HALF activity is observed as anesthesia deepens (Brown et al. 2010). These observations would suggest that chickens may not experience pain, or may experience reduced pain, when immobile in the later stages of a cannibalistic attack (Gentle 2011). In general, the altered electroencephalogram patterns persist for short intervals after the triggering event, alternating with periods of normality

(Gentle & Hunter 1991). This is likely an adaptive response to ensure the animal has the possibility of regaining awareness of the situation in order to escape from an attacker, but it may also indicate that analgesia may be frequently interrupted during an attack.

Analgesia in situations of tonic immobility has been reported for other species (Carli et al. 1981; Porro & Carli 1988). In guinea pigs, for example, tonic immobility has been shown to induce endogenous analgesia involving opioid synapses, which increased their threshold to noxious stimuli such as heat (Leite-Panissi et al. 2001). In fact, analgesia induced by stress and severe pain has been documented in many species (including humans (Butler & Finn 2009)). In addition and presumably as a mechanism to protect the organism from being overwhelmed by pain during dangerous situations, analgesia has also been observed in situations of tonic immobility and others, such as uncontrollable footshock, restraint, inescapable swimming in cold water and body pinching (Gregory 2004).

Still, the analgesic effects of tonic immobility are not entirely uniform across context or species, with a few cases (e.g., lizards) of tonic immobility being associated with hyperalgesia instead (Mauk et al. 1981). Despite evidence to suggest that chickens have a remarkable ability to suppress intense pain (Gentle & Corr 1995), conclusive evidence as to whether tonic immobility has analgesic effects in chickens is still lacking. Additionally, it is not possible to completely rule out the possibility that the immobility observed in victims of cannibalistic attacks emerges as a result of tonic immobility or learned helplessness (Gentle 2011), a phenomenon in which an animal exposed repeatedly to an inescapable stressor gives up reacting, with no attempts of withdraw or protection, even when escape becomes possible. Thus, the possibility that pecking attacks are extremely painful for victims cannot be completely discarded.

Probability descriptions in the Pain-Track (immobile phase) consider two possibilities: (1) that pain of lower intensity (hurtful) is experienced while immobile for some time (30%), but interspersed with periods of disabling pain (based on the observation altered electroencephalogram patterns persist for short intervals after the triggering event, alternating with periods of normality), and (2) that immobility is associated with learned helplessness, hence pain is experienced at very high levels of pain intensity (hence a 30% probability of excruciating pain).

Box 4.2. The adaptive nature of tonic immobility

From an evolutionary perspective, tonic immobility (also referred to as catatonia, thanatosis, playing dead and even animal hypnosis) is believed to have evolved as a response to predator attacks after flight and fight responses have failed - a last resort in face of inescapable danger. Accordingly, some studies (Thompson et al. 1981; Sargeant & Eberhardt 1975) have shown that tonic immobility can increase prey survival by reducing the time of handling by the predator (during which escape is possible) or by increasing the likelihood that predators will drop or cache the individuals for consumption at a later time, providing them with further opportunities to flee. Tonic immobility is observed in a wide variety of vertebrate and invertebrate species, from ants and spiders to fish, rodents and even humans, such as victims of sexual assaults (Volchan et al. 2011). In chickens, tonic immobility has been shown to be elicited by both fear-inducing and painful stimuli (Gentle & Hill 1987). However, this strategy is counterproductive in commercial systems where individuals cannot escape, and are in fact making themselves available to attackers (Gentle 2011).

Burden of Pain in Affected Individuals

Figure 4.4 shows the expected time that affected individuals endure pain at each intensity level as a result of the non-fatal pecking injuries investigated. The estimated duration and intensities of pain are based on the corresponding Pain-Tracks (Pain-Tracks 4.1 to 4.6). Error bars represent the 95% confidence in the time estimates, when the uncertainty in the duration of the time segments depicted in each Pain-Track are taken into account. The time in pain as a result of skin wounds considers an average frequency of wounds over the laying cycle of approximately 1 to 3 wounds per affected bird (Chapter 8 (Schuck-Paim & Alonso 2021c)). For all other wounds (except feather removal), the time in pain depicted in Figure 4.4 represents the pain associated with one episode only. Although the prevalence of pecking injuries may differ across housing conditions (Chapter 8 (Schuck-Paim & Alonso 2021c)) and breeds, the results concern the pain endured by an average affected individual (independently of the prevalence of individuals enduring pecking injuries in the population). Only hours spent awake (16 hours per day) are considered.

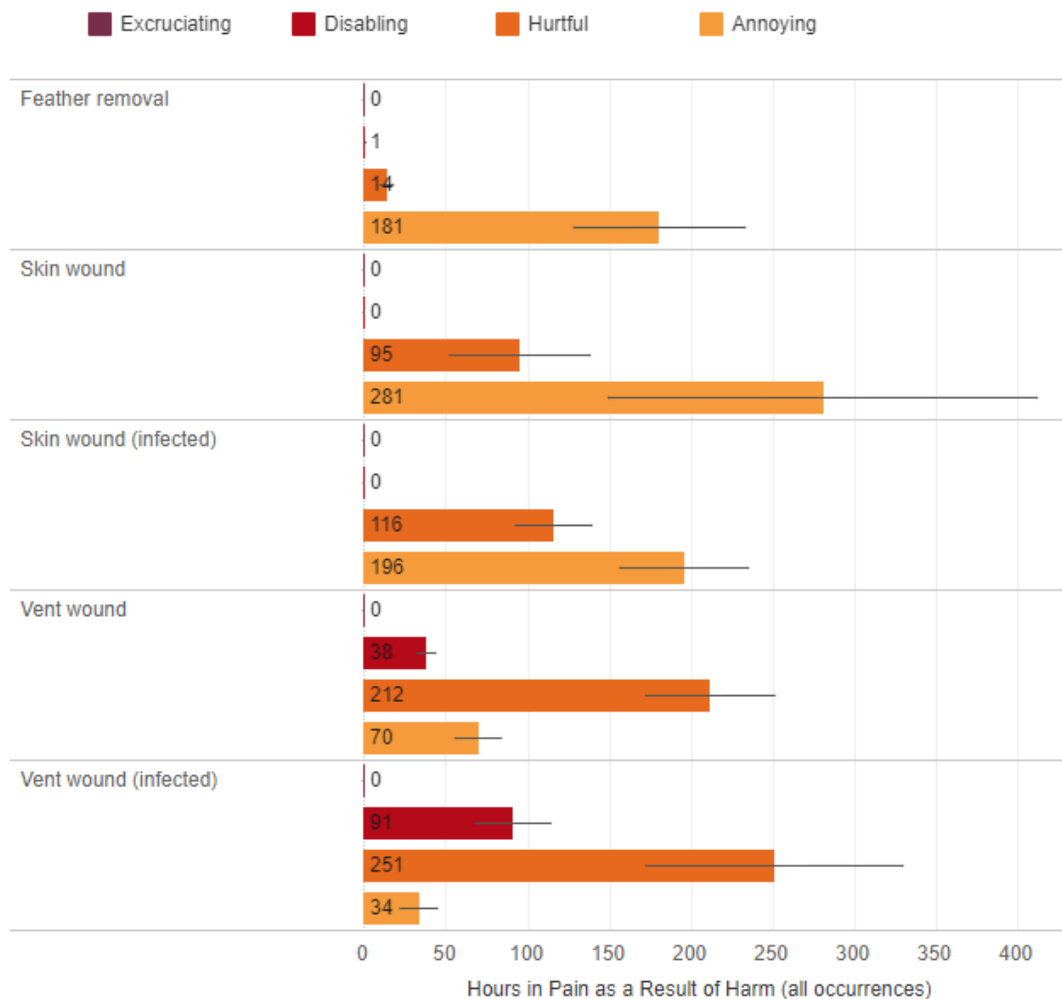


Figure 4.4. Time endured at each level of pain intensity (hours) as a result of pecking injuries. Error bars represent 95% confidence intervals. To determine the pain endured over the entire laying cycle as a result of forceful feather loss, an average plumage score of 30-50% at the end of lay was assumed (the rationale to determine the number of feathers lost is specified in the Chapter 8 (Schuck-Paim & Alonso 2021c)).

Apart from the initial pain (at the Hurtful level) associated with tissue rupture and inflammation (longer in the case of infection), skin wounds are associated predominantly with long hours of discomfort. Vent wounds represent, as expected, a greater source of more intense pain, leading to 38 [33-44] hours on average of Disabling pain when not infected, and 91 [68-114] hours if infection occurs (and resolves spontaneously). An average of 212 [173-251] and 251 [173-329] hours in Hurtful pain (corresponding to approximately 12-15 days) are also expected as a result of one of these wounds.

In the case of those birds that die as a result of pecking injuries, the burden of pain is substantially greater when the pecking attack does not immediately lead to death, but the bird instead dies of infected vent pecking wounds (Figure 4.5). For a hen that dies from an infected vent wound, 53 [46-60] hours of Disabling pain, and over 2 hours [1.5-3] of Excruciating pain are expected on average. To have a sense of scale, an estimated 5% cumulative mortality at the end of lay, and 5% of all

deaths being due to infected vent wounds, would translate into approximately 300 hours of excruciating pain as a result of this injury in a flock of 50,000 hens.

If the reader wants to change any of the assumptions regarding the durations and intensity of the pain depicted in the Pain-Tracks, or the prevalence of the injuries, an interactive web application can be used for this purpose, available at <https://pain-track.org/hens>. In the web application, changes in any of the parameters will immediately update the estimates on the time in pain endured as a result of skin wounds, vent wounds, cannibalistic attacks and multiple events of feather removal.

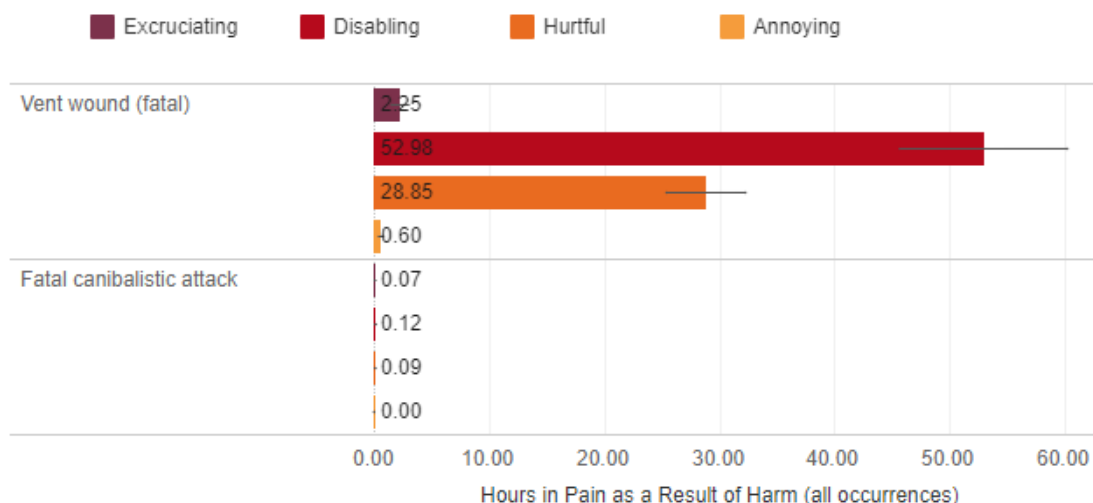


Figure 4.5. Time endured at each level of pain intensity (hours) as a result of fatal pecking injuries. Error bars represent 95% confidence intervals. To determine the pain endured over the entire laying cycle as a result of forceful feather loss, an average plumage score of 30-50% at the end of lay was assumed (Chapter 8 (Schuck-Paim & Alonso 2021c)) An interactive comparison with other harms is available at <https://www.hen-welfare.org/charts>.

Research Gaps

- ❑ Despite many years of research on injurious pecking, ethological data describing the time course of vent pecking events and fatal cannibalistic attacks is extremely scant. Little information is published on the duration of these events, how many pecks a targeted bird typically receives, or how long she takes to die.
- ❑ Similarly, there seems to be no information on the prevalence of skin wounds in the population, their distribution in terms of size, the likelihood of infection or estimates of the number of skin wounds a laying hen can typically sustain over the production cycle. Longitudinal studies on potential changes in the behavior of wounded birds are also still missing.
- ❑ Published information on the wound healing process in poultry, or how long it lasts, is scant, being predominantly restricted to anecdotal reports by backyard keepers.
- ❑ There seems to be no published studies on the clinical evolution of sepsis in poultry. This lack of information is particularly surprising in light of the observation that septicemia is typically the main cause for carcass condemnation in poultry.
- ❑ Despite evidence to suggest that chickens have a remarkable ability to suppress intense pain, conclusive evidence as to whether tonic immobility has analgesic effects in chickens is still lacking.

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