
Quantifying the Welfare Impact of Cardiorespiratory Problems in Broilers

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I. Background

Modern day broiler chickens are the result of intense genetic selection for rapid growth, efficient feed conversion and large breast muscle size. These economically driven changes in genetics have resulted in significant physiological changes – many of which are associated with important welfare challenges. To achieve fast growth, birds divert energy primarily towards muscle deposition, causing development of critical organs to lag behind. Fast-growing strains have a proportionally smaller heart and lung [3]. Additionally, low thyroid hormone activity has been shown to accompany selection for rapid growth, impairing lung development [4]. The increased oxygen demand from large muscles places an additional strain on the already compromised cardio-respiratory system of these birds, leading to discomfort, reduced activity levels, and potential death from heart failure. As put forward by Wideman and collaborators [5] *“a broiler chick weighs 40 g at hatch and is capable of growing to 4,000 g in 8 wk. If humans grew at a similar rate, a 3 kg (6.6 lb) newborn baby would weigh 300 kg (660 lb) after 2 mo. Doubling and redoubling of the body mass almost 7 times in 8 wk cannot be sustained without equally dramatic increases in the functional capacities of the heart and lungs”*.

Two conditions commonly present in commercial flocks, pulmonary hypertension (leading to ascites) and sudden death syndrome (SDS), are a result of cardiorespiratory insufficiency from intense selection for rapid growth. The quantification of their impact on the welfare of fast- and slower-growing broiler chickens (average daily gain of, respectively, ~60 and 45-46g/day) is the subject of this chapter. Their pathophysiology, and association with fast growing rates, are described next.

Understanding cardiorespiratory insufficiency in broiler chickens

Because of the increased demand for oxygen associated with fast growth, coupled with insufficient lung and heart capacity, growing broilers have their pulmonary vessels continuously filled with blood, forcing the right ventricle (which pumps blood out into the body) to develop increasingly higher

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pressures to be able to propel an increasingly higher volume of blood as the bird grows [5]. Such a high pressure will lead to the hypertrophy of the right ventricle and faster blood flow rates in the lungs, leaving too short of a time for the capture of O₂, causing a low pressure of O₂ in the blood (hypoxemia) and high pressure of CO₂².

Hypoxemia, in turn, leads to an increase in the production of red blood cells (enhancing the O₂ carrying capacity), increasing blood viscosity and flow resistance, which in turn contributes to the development of pulmonary hypertension [5]. Hypoxemia also increases the dilation of systemic arteries to increase blood flow and restore adequate O₂ delivery. The dilation of arteries causes systemic blood pressure to drop and heart rate to rise, increasing the rate at which blood returns to the right ventricle. This forces the right ventricle to develop even higher pressure to pump the higher volume of blood out [5], escalating pulmonary hypertension and ventricular hypertrophy.

With time, the ventricular muscle will become weak and dilated, blood will start flowing backwards into the atrium, marking the onset of congestive heart failure [5] (the term used for the impaired pumping power of the heart muscle). Low blood pressure will also cause the kidneys to retain sodium and water, increasing the volume and pressure within the systemic veins. Together with hypoxemia, this will also impair the flow of blood from the intestines to the liver, eventually causing hepatic cell death and cirrhosis (scarring - fibrosis - of the liver tissue), and plasma transudation from the liver into the abdominal cavity [5] (or “water belly” [7]).

Ascites (the term used for the abnormal accumulation of fluid in the abdominal cavity) can cause prolonged suffering before becoming severe enough to result in death, which will often happen due to a combination of hypoxemia, congestive heart failure, starvation and respiratory distress caused by pulmonary edema and compression of the air sacs in the abdomen by the accumulation of fluid in the abdominal cavity [5][8]. Live birds experiencing ascites present with visibly swollen abdomens and apparent respiratory distress [9] (see for instance: <https://youtu.be/IMh4zjv-FE8>). Males seem to be affected at higher rates than females, presumably due to their larger body mass [10]. Blood pH can also be predictive of ascites, with male broilers often having lower pH values, and females having higher. While genetic selection for fast growth is the main cause of ascites, environmental conditions such as poor ventilation, cold ambient temperatures and high altitudes may exacerbate it [10,11].

Different from pulmonary hypertension and ascites, sudden death syndrome (SDS) seems to occur without warning, with seemingly healthy birds suddenly flipping over and dying. This condition is believed to be caused by a lack of oxygen to the myocardium, leading to acute heart failure. Fast-growing broilers are highly predisposed to abnormal heart rhythm disturbances, with abnormalities detectable in birds as young as a week old [12]. In studies in which broilers were feed-restricted to slow down growth, prevalence of arrhythmia was reduced to less than 2% [13]. Accordingly, the incidence of arrhythmia in breeds selected for slow growth is thought to be less than 1% [13].

When birds with arrhythmia are exposed to stressful conditions, the risk of sudden death increases. High stocking densities, in which many commercial broilers are housed, have been shown to be a chronic stressor on broilers and lead to increased mortality rates by SDS [14]. Prior to death, birds are observed violently flapping their wings, extending their neck, emitting a vocalization (“squawk”), and collapsing [15].

² These measurements can be used to determine which birds are at a heightened risk of ascites later in life [6].

II. Prevalence of cardiorespiratory challenges in commercial breeds

Fast-growing breeds

Determining the prevalence of cardiorespiratory conditions in commercial breeds is challenging. Data owned by commercial companies predominantly remain confidential and conditions between commercial facilities vary widely. Many studies rely on survey data from producers, however accurate diagnoses of the underlying causes of mortality can be difficult for conditions such as SDS, where no specific post-mortem lesions are present. Also, prevalence data from surveys at the slaughter line disregard on-farm and transport mortality, underestimating prevalence figures in the production cycle.

About 15 years ago, mortality from ascites was estimated to vary from 5% to 8% of total deaths in various populations, increasing up to 30% in heavier broiler flocks [16]. In US flocks, ascites was similarly estimated to cause up to 30% of deaths at about the same time (reviewed in [7,17]). In a review of the financial impact of production diseases in poultry, the authors report, however, a wider range of mortality from ascites, from 15 to 68% of total mortality [18], with the extreme values corresponding to flocks fed pelleted food and raised in high altitudes. There is no recent data on the prevalence of mortality from ascites in commercial flocks, but it is generally accepted that it may have decreased as a result of selective breeding aimed to correct the condition. Therefore, we tentatively assume that ascites may currently underlie, on average, 5 to 40% of deaths in commercial broiler flocks. Considering that cumulative mortality in commercial flocks kept until 42 days ranges between 4 and 6% [18,19], this translates into **0.3 to 2%** of the commercial broiler population dying from ascites.

Still, many more birds are affected by ascites than those that die from this condition. In studies involving post-mortem inspection of birds that died from ascites and those that were presumed healthy, abnormal changes in cardiac muscle and valve dilation were observed in both groups of birds [21], indicating the condition was substantially more prevalent than suggested by mortality figures only. Indeed, many condemnations at slaughter plants, of birds that reach the slaughter weight alive, are due to ascites [7]. About two decades ago, the global average incidence of ascites in live broilers was estimated at 4.7% [7]. A more recent review also reports an average incidence of 5% [18]. Considering the wide variability in the prevalence of the disease as a function of differences in nutrition, management and environmental conditions, we estimate the number of birds affected by non-fatal cases of ascites (during which birds experience the symptoms of pulmonary hypertension, before it causes de transudation of fluid into the abdomen and progresses to death) to range, on average, from **2.5 to 7.5%** (i.e. $\pm 50\%$ of the reported values).

Different from ascites, SDS is an acute condition that develops in seemingly healthy individuals. Although birds that succumb to SDS frequently have a history of cardiac rhythm disturbances [13] - a problem estimated to affect 17-30% of fast-growing broiler populations two decades ago [12,22] - there is no evidence to suggest the presence of distress or discomfort prior to the onset of death. Frequently, apparently normal broilers in good body condition die suddenly without any discernible pre-existing clinical signs. Therefore, here we consider only the potential pain associated with the SDS event.

SDS used to account for most mortality in commercial broiler flocks in the end of the 1990's [12]. Currently, veterinary manuals report a typical mortality rate from ascites ranging from 0.5 to 4% [23], yet some authors report narrower ranges, from 1.5 to 2% in healthy flocks of fast-growing birds [24]. Here we conservatively assume a wider range, from **0.5 to 4%**, as reported by Merck, in birds grown to 6 weeks (the median value is the same as in the range 1.5-2%, only the uncertainty is larger).

Slower-growing breeds

Ascites can be virtually eliminated from broiler populations through the use of slower growing breeds, given their reduced metabolic demand [26]. In a recent trial conducted at the farm level, which compared one fast-growing (ADG: 58g/d) with three slower-growing breeds (ADG: 44-47 g/d), 0.16% of fast-growing birds were rejected at the slaughter line due to the presence of ascitic oedema, but none from the slower-growing strains [27]. Accordingly, low energy intake through feed restriction at an early age can similarly drastically reduce the incidence of ascites and SDS given the slower growth rate [28].

Additionally, the use of slower growing breeds tends to be accompanied by improvements in other environmental inputs that are likely to reduce even further the likelihood of these disorders. For example, the Better Chicken Commitment (EU Broiler Council Directive 2007/43/EC) sets limits on maximum stocking densities within broiler facilities at 30kg/m². Reduced stocking densities can improve litter quality and air flow, leading to better air quality parameters within the house, reducing respiratory stress on the birds [29]. The EU directive also mandates a required dark period of at least 6 hours per day. By requiring a period of rest, growth and strain on the cardiovascular system can be reduced through decreasing oxygen utilization in the body [9,30]. Even in fast-growing strains, these lighting schemes have been shown to substantially reduce the incidence and severity of circulatory disorders and mortalities due to ascites by roughly 30-40% [31–33].

The risk of SDS can be similarly virtually eliminated in slower growing strains due to their lower metabolic demands, especially under improved housing conditions. Less metabolic stress, lower stocking densities, and improved lighting schedules can virtually eliminate many of the stressors that lead to the onset of SDS.

Therefore, here we conservatively assume that the risks of ascites (fatal and non-fatal) and SDS in slower-growing strains will range from 0 to 10% of those reported for fast-growing strains. In the next sections we investigate the possible intensity (Box 1), and duration, of pulmonary hypertension and SDS in fast- and slower-growing breeds. In all scenarios, we conservatively assume that no negative affective state as a result of these conditions is experienced when individuals are sleeping (Chapter 1 describes the rationale for this assumption, and for estimating the time birds spend awake per day).

Box 1 Definition of Pain Intensity Categories

Annoying: experiences of pain perceived as aversive, but not intense enough to disrupt the animal's routine in a way that alters adaptive functioning or affects the behaviours that animals are motivated to perform. Similarly, Annoying pain should not deter individuals from enjoying pleasant experiences with no short-term function (e.g., play) and positive social interactions. Sufferers can ignore this sensation most of the time. Performance of cognitive tasks demanding attention are either not affected or only mildly affected. Physiological departures from expected baseline values are not expected to be present. Vocalizations and other overt expressions of pain should not be observed.

Hurtful: experiences in this category disrupt the ability of individuals to function optimally. Different from Annoying pain, the ability to draw attention away from the sensation of pain is reduced: awareness of pain is likely to be present most of the time, interspersed by brief periods during which pain can be ignored depending on the level of distraction provided by other activities. Individuals can still conduct routine activities that are important in the short-term (e.g. eating, foraging) and perform cognitively demanding tasks, but an impairment in their ability or motivation to do so is likely to be observed. Although animals may still engage in behaviors they are strongly motivated to perform (i.e., exploratory, comfort, sexual, and maintenance behaviors), their frequency or duration is likely to be reduced. Engagement in positive activities with no immediate benefits (e.g., play in piglets, dustbathing in chickens) is not expected. Reduced alertness and inattention to ongoing stimuli may be present. The effect of (effective) drugs (e.g., analgesics if pain is physical, psychotropic drugs in the case of psychological pain) in the alleviation of symptoms is expected.

Disabling: pain at this level takes priority over most bids for behavioral execution, and prevents all forms of enjoyment or positive welfare. Pain is continuously distressing. Individuals affected by harms in this category often change their activity levels drastically (the degree of disruption in the ability of an organism to function optimally should not be confused with the overt expression of pain behaviors, which is less likely in prey species). Inattention and unresponsiveness to milder forms of pain or other ongoing stimuli and surroundings is likely to be observed. Relief often requires higher drug dosages or more powerful drugs. The term Disabling refers to the disability caused by 'pain', not to any structural disability.

Excruciating: all conditions and events associated with extreme levels of pain that are not normally tolerated even if only for a few seconds. In humans, it would mark the threshold of pain under which many people choose to take their life rather than endure the pain. This is the case, for example, of scalding and severe burning events. Behavioral patterns associated with experiences in this category may include loud screaming, involuntary shaking, extreme muscle tension or extreme restlessness. Another criterion is the manifestation of behaviors that individuals would strongly refrain from displaying under normal circumstances, as they threaten body integrity (e.g. running into hazardous areas or exposing oneself to sources of danger, such as predators, as a result of pain or of attempts to alleviate it). The attribution of conditions to this level must therefore be done cautiously. Concealment of pain is not possible.

III. Pain-Track: Pulmonary Hypertension (Ascites)

In Pain-Track 1 we propose a tentative hypothesis for the intensity and duration of the pain associated with non-fatal cases of pulmonary hypertension. In Pain-Track 2 we consider its progression to ascites and cardiac failure. The first two stages (hypoxemia, pulmonary hypertension) are common to Pain-Track 1 and 2. The justification for the parameter values in the pain-tracks are given next. The criteria defining the intensity categories (Annoying, Hurtful, Disabling and Excruciating) are available in Chapter 1.

Pain-Track 1. Hypothesis for the temporal evolution of the pain endured by commercial broilers affected by **non-fatal cases** of pulmonary hypertension (ascites) that do not progress to ascites. Estimated sleeping time: 4 to 10 hours/day.

	i. Hypoxaemia	ii. Pulmonary Hypertension		TIME IN PAIN (hrs)
Excruciating			Excruciating	
Disabling	10%	25%	Disabling	84 [42-130]
Hurtful	80%	50%	Hurtful	280 [170-390]
Annoying	10%	25%	Annoying	84 [42-130]
	98-280 h	98-420 h		
(duration in weeks)	(1-2)	(1-3)		

Phase i. Hypoxemia

Low blood oxygen is commonly associated with feelings of lethargy, weakness and increased heart rates. Respiration rate will also increase in an attempt to bring in more oxygen, which is likely accompanied by feelings of shortness of breath or difficulty breathing. Humans experiencing hypoxemia often report dyspnea (difficulty breathing), lethargy, and difficulty drinking [38].

Given the likely fatigue, breathlessness and weakness typically associated with hypoxemia, it can be presumed that the majority of broilers experiencing hypoxemia experience pain at the “Hurtful” level, since this is not a sensation that can be ignored most of the time (as it would be required under the Annoying level). However, birds are still able to perform tasks required for survival, though their frequencies (e.g., drinking) may be reduced. Male broilers are likely to experience more severe symptoms than females due to their accelerated growth and increased oxygen demands. These extremely fast-growing birds, especially those kept in poor environmental conditions, may experience elevated respiratory distress. For some of the heaviest individuals of flocks, we cannot rule out the possibility that pain reaches a Disabling level of intensity given the severe distress associated with severe shortness of breath. While most clinical symptoms are not apparent at this stage to caretakers, birds in the “Disabling” category may be observed panting and their movements may be greatly reduced. On the other side of the scale, lighter birds with hypoxemia may experience considerably less severe symptoms, with the distress experienced consistent with the definition of the “Annoying” category of intensity. Cardiac abnormalities have been detected in broilers as young as 12 days old, suggesting birds may experience cardiac insufficiencies related to hypoxemia for one to two weeks before developing more severe symptoms [22].

Phase ii. Pulmonary Hypertension

Broilers experiencing right ventricular failure (section ‘Understanding cardiorespiratory insufficiency in broiler chickens’) display reduced growth and may be smaller than their penmates [9]. This reduction in growth rate is likely due to the combination of reduced feed intake, either from

discomfort or decreased appetite, with nutrient absorption insufficiencies as a result of altered metabolism as is evident in humans affected by this condition [40]. Under the hypothesis of reduced feed intake and activity due to discomfort, the intensity of the distress experienced would be compatible with both the Disabling and Hurtful categories. Conversely, under the possibility of altered metabolism leading to decreased appetite (such as that experienced as a result of liver cirrhosis in humans, [41]) and poor nutrient absorption, pain would be compatible with the “Hurtful” and Annoying categories (see Chapter 1). In humans, the underlying cause of weight loss is poorly understood. It has been concluded that it is likely a combination of malabsorption, gastrointestinal discomfort, and appetite loss, with unclear weight given to any condition [42]. Since data is not available that enables determining whether reduced feed intake derives from a lack of appetite or from the discomfort associated with the condition, we distribute probabilities equally to each of the proposed hypotheses. The duration of pulmonary hypertension for birds that experience it may be estimated to be around 2-3 weeks based on (1) knowledge of when cardiac abnormalities appear (around two weeks) and (2) the typical onset of symptoms of ascites (fluid transudation), which are most frequent at around 4-5 weeks of age [43].

Pain-Track 2. Hypothesis for the temporal evolution of the pain endured by commercial broilers affected by **fatal cases** of pulmonary hypertension, which progresses to ascites and death. Estimated sleeping time: 4 to 10 hours/day.

	i. Hypoxaemia	ii. Pulmonary Hypertension	iii. Fluid Transudation	iv. Severe Cardiac Failure (death)		TIME IN PAIN (hrs)
Excruciating			1%	50%	Excruciating	39 min [18-60]
Disabling	10%	25%	74%	50%	Disabling	130 [81-180]
Hurtful	80%	50%	25%		Hurtful	290 [180-400]
Annoying	10%	25%			Annoying	84 [42-130]
	98-280 hrs (1-2 wks)	98-420 hrs (1-3 wks)	28-100 hrs (2-5 days)	~1 minute		

Phase iii. Fluid Transudation

The late stages of pulmonary hypertension result in increased intravascular pressure, causing the transudation of fluid into abdominal cavity (ascites). Pressure on the air sacs from fluid accumulation causes severe respiratory distress [44,45]. The tell tale sign of ascites is the distended abdomen, often referred to as “water belly”. Visually, birds appear dull and depressed and may be reluctant to move [30,46]. They may experience bradycardia (slower than normal heart rate) and tachypnoea (rapid breathing), which may be accompanied by open beak panting [30,46,47]. Lack of circulating oxygen may cause cyanosis (blue tint to the skin) that may be most noticeable on the bird’s comb and wattles, which may also have a sunken appearance [30]. As a result of the high pressure, superficial veins may become dilated. It has been reported that birds may survive for a few days with visible ascites symptoms such as the distended abdomen [9]. It is very likely that the level of discomfort birds experience is correlated with the amount of fluid build-up that has occurred, but predominantly at a Disabling level. This is consistent with the report by some authors that death from ascites is usually preceded by prolonged agony [12]. As time progresses, birds likely go from the “Disabling” level of

pain as they begin to experience pressure in their abdomen, to “Excruciating” levels of pain as they become less able to breathe. In Pain-Track 2 we consider the possibility that, in the final moments preceding cardiac failure, the symptoms associated with ascites are of an Excruciating nature given the highly distressful nature of breathlessness, hence the 1% probability attributed to this category. Similarly, because we cannot rule out the possibility that pain is less severe in the first stages of fluid transudation, we also attribute a 25% probability to the Hurtful category.

Phase iv. Severe Cardiac Failure

Death from ascites often occurs late in the production cycle (mostly after 5 weeks of age), sometimes during handling or transport to slaughter [30,43,48]. Dead birds are often found on their backs. Cause of death can be attributed to profound hypoxemia, terminal congestive heart failure, severe respiratory distress as a result of pulmonary oedema, ascitic compression of the air sacs, or starvation [5]. Upon post mortem examination, accumulation of ascitic fluid and clots of fibrin can be observed in the abdomen, in addition to hydropericardium, right ventricular hypertrophy, and generalized venous congestion [49].

It may be presumed that, in the final minute of life before death, abdominal and chest compression and, particularly, respiratory distress are so severe that the intensity of the pain is of an Excruciating nature, as expected for conditions in which individuals are unable to breath. We conservatively assume a similar probability that the pain is only Disabling at this stage.

IV. Pain-Track: Sudden Death Syndrome

In the case of sudden death syndrome, birds appear to be perfectly healthy prior to death [50]. Immediately preceding death, birds engage in normal behaviors such as feeding, standing, walking, and lying [51]. It can be presumed that birds do not experience any discomfort leading up to death as there is no observed reduction in activity levels, no difference in behavioural patterns between healthy birds and those that succumb to SDS, no difference in appetite or feeding behavior, no reduction of maintenance activities such as preening, and no diminished responsiveness to external stimuli [51].

Onset of death occurs suddenly. Birds can be observed violently flapping their wings and extending their necks [15,51]. Birds experience strong muscle contractions and a severe loss of balance, causing them to fall over [51]. Some birds may produce a “squawk” type vocalization [15,51]. The vast majority of birds are found deceased lying on their back, hence SDS sometimes being referred to as “flip-over syndrome”, however birds that died from SDS may also be found in other postures such as on their sternum. Cause of death is believed to be ventricular fibrillation [52,53].

Fast growing broilers are predisposed to arrhythmia of the myocardium. When exposed to stress, whether from metabolic strain from rapid growth or poor environmental conditions such as overcrowding, the risk of death from SDS is increased [54]. Onset of SDS has been observed as early as 2 days of age [25] and can continue until birds reach market weight. Peak mortality rates have been reported between 9-22 days [9] and from 21-27 days [25]. The duration of the SDS episode, from the

first signs of distress until death, has been shown to range from 37 to 69 seconds [51]. Humans experiencing ventricular fibrillation report acute chest pain and breathing difficulty [55]. Therefore, we assume that pain experienced from ventricular fibrillation will fall under the Disabling category, although it is not possible to completely rule out an Excruciating intensity for a few seconds or for a proportion of individuals.

Pain-Track 3. Hypothesis for the temporal evolution of the pain endured by commercial broilers dying from SDS.



V. The Burden of Pain from CardioRespiratory Challenges

Birds affected by pulmonary hypertension (ascites) endure very long hours in pain, as shown in Pain-Tracks 1 and 2. If the condition evolves to ascites and cardiac failure, the time in Disabling will reach on average 130 hours ‘awake’ (a week to 10 days), with an expected 3 hours in Excruciating pain. For birds affected, the bird of pain is, therefore, substantial, on par with that experienced by individuals affected by infectious diseases that progress to sepsis and septic shock [56].

However, when the time spent in pain at each intensity level is weighted by the prevalence of each condition in the population, the burden of pain from these conditions becomes considerably shorter, since a relatively small proportion of individuals is affected (section II). Figure 1 shows the total time endured at each level of pain intensity by an “average” broiler chicken.

Box 1 Time in Pain endured by the Average Population Member

Although pain inherently concerns individuals, we operationally accept that the collective welfare of the members of a population can also be determined. Measuring cumulative pain at the population level is also necessary to account for the heterogeneity in the exposure of population members to different challenges. For example, while lameness is experienced by a large fraction of broiler chickens (Chapter 2), events such as fatal cases of ascites are only experienced by a few individuals.

Therefore, measurement efforts must consider the prevalence of each welfare challenge, so that pain is determined for the average member of the population (which may not necessarily correspond to any real organism). At the population level, the time spent at each level of pain intensity as a result of each challenge is determined by multiplying it by its prevalence. For example, if a condition causes 10 hours of Disabling pain and 70% of the population are affected, then the average member of this population could be said to experience 7 hours of Disabling pain due to the condition. Measurements at the population level enable comparing the impact of different practices and conditions across demographics, geographies and time.

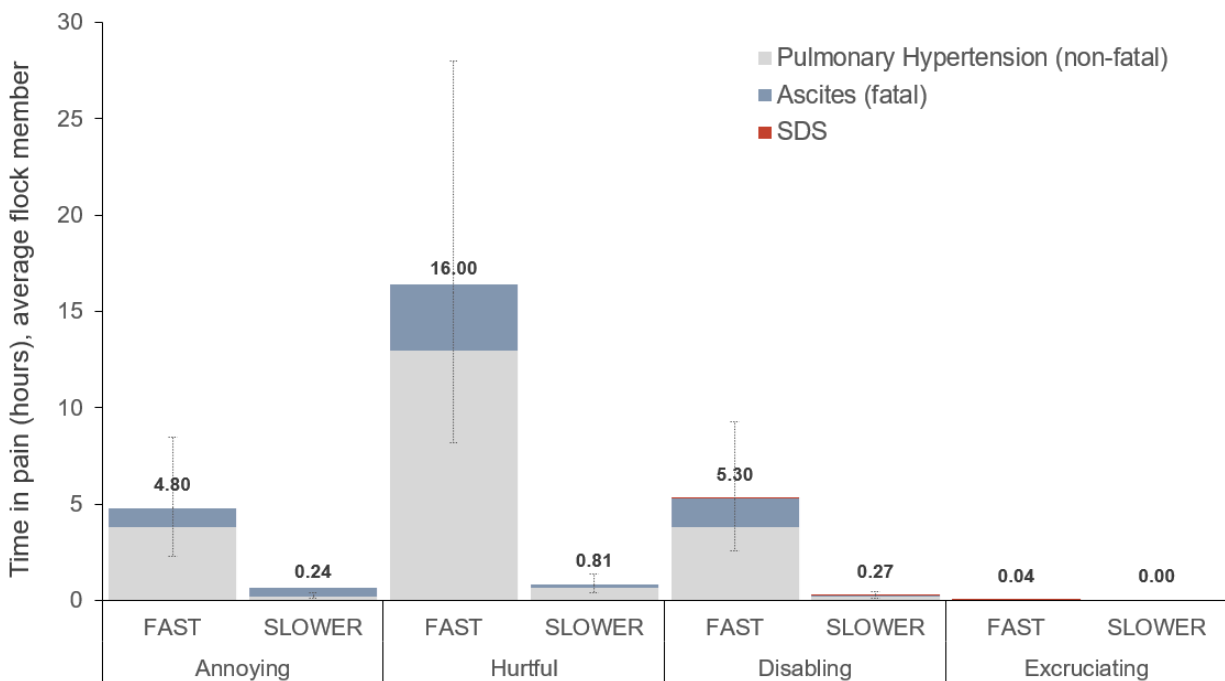


Figure 1. Total time endured at each level of pain intensity (hours) by an “average” broiler chicken as a result of pulmonary hypertension (fatal and non-fatal) and Sudden Death Syndrome (SDS). Error bars represent 95% confidence intervals.

Since time in pain is concentrated in a relatively small proportion of individuals, at the flock level the time in pain as a result of cardiopulmonary conditions is relatively short. This is particularly the case for SDS.

These findings are also interesting in that they show the poor correlation between the welfare impact of a condition and its cost to producers. Despite the relatively short time spent in pain by the average broiler as a result of these conditions, SDS and ascites still remain among the main causes of mortality in broiler flocks. Despite some success in the reduction in the incidence of these cardiopulmonary challenges through selective breeding, their incidence is still substantially higher than in slower-growing individuals. Once again, it indicates that as with other welfare issues present in conventional genotypes, the extent to which cardiopulmonary risks can be mitigated without reducing growth rates is limited [57]. Indeed, as shown in Figure 1, the estimated time spent in pain at all levels is substantially shorter for slower-growing strains despite their longer lifetime.

VI. Research Gaps and Opportunities

1. There is no recent data on the prevalence of mortality from ascites in commercial flocks, either from fast-growing or slower growing strains. Commercial datasets are private, with research currently relying on experimental studies, small and old datasets or surveys in the slaughter plant, which disregard on-farm and transport mortality, underestimating prevalence figures in the production cycle.
2. Improved feeding strategies and environmental conditions may mask or delay the progression of ascites or sudden death syndrome. Recent information on the incidence of non-fatal cases of pulmonary hypertension in commercial flocks is currently lacking.
3. Likewise, the extent to which the onset of cardiac abnormalities are delayed in slower-growing birds is currently unknown.
4. The underlying cause of reduced feed intake in birds experiencing pulmonary hypertension is not fully understood. Whether this is due to a lack of appetite driven by metabolic changes or due to feelings of discomfort, or a combination of factors, make it difficult to estimate the extent of suffering experienced by individuals.

VI. References

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