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# **Bone development on the distal phalanx of New Zealand dairy cows: The link to lameness**

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the degree of

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## Abstract

Claw horn disruptions are important cause of lameness and affects cattle around world, causing major economic and welfare problems for dairy industry. Inflammation from an active CHD can lead to bone growth (exostoses) on the distal phalanx, which predisposes the animal to further CHD in future and damages the internal anatomy of the hoof. Previous quantitative studies have evaluated, using  $\mu$ -CT, bone development on the distal phalanx of cows which are housed for most or all of the year, and identified that bone growth is associated with age, lifetime history of CHD and chronic lameness. The aim of the present study was to evaluate whether digital photography could be used instead of  $\mu$ -CT to obtain linear measurements of exostoses and to find whether bone development was associated with age, claw type and lameness history in the lactation prior to culling in pasture-based cattle. Distal phalanxes (160) were collected from the hind feet of 40 dairy cows after slaughter. Digital photographs were then taken, and bone growth measurements were made at 4 locations near the flexor tuberosity (A to D) guided by the method using  $\mu$ -CT. Modelling was then used to identify whether age, claw type and lameness history were associated with the average of the sum of measurements A to D across all four claws of an individual cow (Ave BG) and the highest sum of A to D for an individual claw within a cow (BG Max). Only minor modifications were necessary to the measurement method to account for the differences between  $\mu$ -CT and digital photography, thus digital photography provides an economic alternative to the use of  $\mu$ -CT to study bone development in the distal phalanx. An association was found between age and BG Max and Ave BG with a one-year increase in age being associated with BG Max increasing by 1.12 (95% CI: 1.03-1.21) times and Ave BG increasing by 1.09 times (95% CI: 1.04-1.14). Outcomes for history of lameness were also compatible with lameness increasing bone growth, with the point estimate for BG Max being 1.61 (95 CI: 0.88-2.95) (equivalent to 5 years of age effect) and for Ave BG being 1.25 (CI:0.89-1.78) (equivalent to 3 years of age effect). However, the data were also compatible with no biologically important association between lameness and bone growth. The majority of the differences between claws in BG Max and Ave BG were driven by differences in measurement D as 136/160 claws had zero values for measurements A, B and C but all claws had values  $> 0$  mm for D. In all claws where the only measurement  $>0$  was measurement D, the bone development appeared to be normal being smooth in appearance without any pointed edges or rough surfaces. Analysis of the effect of age on measurement D (in claws with A, B, C= 0), and on the sum of A, B and C was consistent with the hypothesis that the principal effect of age was through its impact on measurement D, as the

effect of age on measurement was similar to its impact on BG max, and the analysis of age on sum of A, B and C was compatible with no effect. However, these are preliminary data, and this hypothesis needs more investigation.

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# Literature Review

## Introduction

Dairy farming contributes 3.2% of New Zealand's GDP, and dairy products accounts for 25% of the value of all exports (Anonymous. 2024a). New Zealand is the tenth largest milk producer in the world supplying 14% of the world's dairy exports (Anonymous. 2024a). As of 2023/24 there were 4.7 million dairy cows in New Zealand with 60 % of these being New Zealand Friesian/ Jersey cross ("Kiwi Cross") cows, with Friesian and Jersey cows accounting for 24% and 8%, respectively (Anonymous. 2024b).

On most farms in New Zealand, dairy cattle are kept at pasture all year-round. This means that they are principally fed on grass with limited use of concentrates and have lower average milk yields than intensive housed cows. They also have limited contact with concrete and similar surfaces except when they are being milked, as they spend their dry period at grass as well as calving at grass.

This pasture-based system has major benefits in terms of cow welfare, particularly in relation to biological functioning and natural behaviour (Mee and Boyle 2020). One key welfare benefit is a reduction in lameness; in a recent study Mason *et al.* (2023) reported that the median prevalence of lameness on 120 farms was 2.8 %, much lower than any of the 53 studies of lameness prevalence reported by Thomsen *et al.* (2023).

Lameness is an abnormality in gait or posture. In cattle, lameness is usually associated with tissue damage, pain and discomfort (O'Callaghan 2002). This, in combination with high rates of persistence and recurrence means that lameness is one of the most important welfare issues in modern dairy cattle (Whay and Shearer 2017). Lameness is also associated with significant economic loss (Urban-Chmiel *et al.* 2024).

Lameness is not one disease, but a clinical sign produced by many diseases. In dairy cattle, claw horn disease, where the principal lesion is in the horn of the claw, is the most important cause of lameness partly because of their relatively high prevalence of lameness compared to other causes of lameness (Murray *et al.* 1996; Chesterton *et al.* 2008; Sadiq *et al.* 2017), but also because of their high level of persistence and recurrence (Leach *et al.* 2012).

The lesions which cause claw horn lameness are often described as claw horn disruption lesions (CHDL). The most important of these are hoof horn haemorrhages, sole ulcer and white line disease (Bicalho and Oikonomou 2013).

Claw horn disease is the end stage of long process. The aetiology of claw horn diseases and the CHDL which produce it, is far from fully understood, but it is clear that CHDL principally arise from contusions of the corium (the tissue which produces and nourishes the hoof horn) by the distal phalanx (pedal bone) (Newsome *et al.* 2016). The damage to the corium by the distal phalanx results in staining of the horn surrounding the damaged corium by blood. Four to 6 weeks later (when this horn comes into wear), this staining is seen as hoof horn haemorrhages (Laven and Laven 2024). Hoof horn haemorrhages are indicators of mild to moderate corium damage, and as such are precursors to the more severe lesions that result in white line disease and sole ulcer. Once damaged the corium may never heal completely with keratinocytes being replaced by fibrosis (Blowey 2016). This means that cows with CHDL are subsequently at increased risk of CHDL even when the lesions are apparently healed. Other factors can also increase the risk of lameness in cattle which have had lameness before. One of the most important of these is the development of bony exostoses on the distal phalanx, which develop in response to the inflammation secondary to corium damage (Newsome *et al.* 2016). Clearly distal phalanges with exostoses are likely to cause more damage to the corium than bones without such exostoses, so the development of exostoses will significantly increase the risk (and perhaps severity) of subsequent lameness. In addition, once formed, there is no treatment for these exostoses so they will persist. Preventing the formation of these exostoses is thus likely to have significant benefits in terms of reducing lameness. However, research has shown that increasing age may be as important for the development of exostoses as lameness (if not more) (Tsuka *et al.* 2012; Newsome *et al.* 2016). If this is the case, then early effective treatment may only delay rather than prevent the development of lameness exacerbating exostoses.

However, both Tsuka *et al.* (2012) and Newsome *et al.* (2016) studied exostoses in housed cattle with limited or no access to pasture. It is thus possible that the age-related exostoses are actually produced by recurrent minor damage which is too small to produce clinical lameness but sufficient to cause localised inflammation. If this is the case, then it is likely that the minor damage is caused by recurrent interaction with hard flooring, and it may not occur or be much reduced in cows kept at pasture. The aim of this study was thus to assess whether this was the case.

## Pathogenesis of claw horn disruptions

Claw horn disruption (CHD) is a comprehensive term that encompasses a group of non-infectious causes of lameness in which disruption of hoof horn production is the principal feature. The key lesions associated with CHD are white line and sole haemorrhages, sole ulcer, and white line disease (WLD) (Griffiths *et al.* 2020). Together these lesions are a major cause of lameness in the modern dairy cow (Bicalho and Oikonomu 2013)

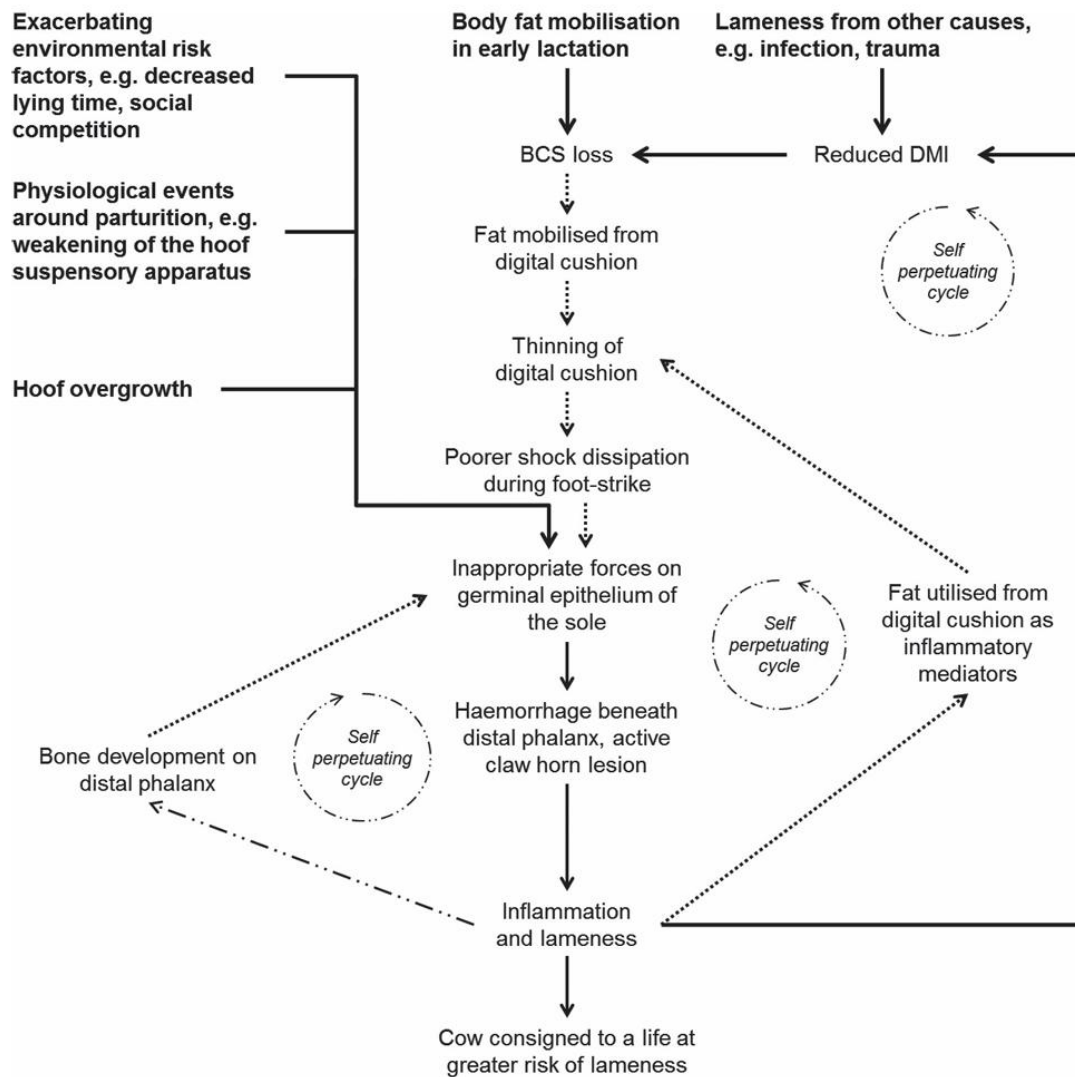
CHD is a term based on the presentation of the lesions rather than their aetiopathogenesis (Griffiths *et al.* 2020), which is useful when our understanding of the causes of CHD has changed and improved significantly since the first use of the term ‘claw horn disruption’ in the 1990s. CHD is a multifactorial disease with a large number of associated risk factors (Knott *et al.* 2007); however it is clear that the initiating cause of the lesions is contusion of the hoof horn-producing and sustaining tissue (the corium) (Räber *et al.* 2004).

Contusion of the corium initially results in capillary damage and haemorrhage. If the contusions continue, or are sufficiently severe as a one-off insult, the damage and haemorrhage will progress to thrombosis, cellular inflammatory reactions and, finally, ischaemic necrosis (Lischer and Ossent 2002). All of these changes interfere with the normal nutrient exchange of the corium and the basal epidermal cells. If they are severe enough, they will lead to the degeneration of the basal epidermal cells and defective horn production (Bergsten 1994).

The initial corium damage is not usually detectable from the outside of the hoof, but the haemorrhage is incorporated into the hoof horn and, as the horn grows and wears, the haemorrhage becomes visible at the hoof surface (as white line or sole haemorrhages); a process that normally takes at least 6 weeks or more (Laven and Laven 2024). If the damage is more severe then necrotic tissue can also be incorporated into the horn and once this horn comes into wear, the necrotic area will be visible as CHD. Sole ulcers develop when the impact on the corium is sufficient to result in the development of a full-thickness horn defect in the sole horn (Shearer and van Amstel 2017) while WLD occurs secondary to damage of the corium producing the horn of the white line (the soft horn layer that connects the horn of the wall to the horn of the sole). Damage to the white line results in less resistance to the shearing forces that occur when the cow places its foot on the ground, result in separation of the sole and wall horn. This separation leads to formation of fissures which can allow the entry of bacteria and foreign bodies which further increase the separation and can lead to abscessation often accompanied by further corium damage.

# Causes of CHD

Newsome *et al.* (2016) identified that inappropriate forces on the epithelium of the corium were the key factor leading to CHD and identified three groups of factors which were principally responsible for these inappropriate forces (see **Figure 1**): environmental factors, systemic events around parturition and hoof overgrowth.



**Figure 1:** Pathogenesis of CHD. From Newsome *et al.* (2016)

## 1. Environmental factors

Hard surfaces, which increase ground reaction forces at the claw-floor interface, have long been identified as associated with the development of CHD (Bazeley and Pinsent 1984). Cows with more exposure to concrete have a higher risk of lameness. For example, in the UK study

by Haskell *et al.* (2006), they observed higher lameness score in cows which were kept in free-stall barns compared to cows kept in straw yards. Similarly in the US Adams *et al.* (2017) observed a higher prevalence of lameness in farms with free-stall barns and dry lots (predicted 3.2 and 2.5%) compared with pasture (0.5%), while recent study in New Zealand by Mason *et al.* (2024) observed that farms which used concrete stand-off pads during harsh weather conditions to house cows had 1.45 times higher risk of lameness than the farms that did not use concrete stand-off pads.

These epidemiological associations between increased exposure to hard surfaces have also been seen in controlled studies. For example, Vermunt and Greenough (1996) identified that pre-partum heifers kept in cubicle yards had a higher incidence of hoof horn haemorrhages (of greater severity) than heifers maintained in a dry lot during the same period. Frankena *et al.* (1992) observed that heifers kept in concrete yards had 5 times increased odds of counteracting sole haemorrhages than heifers kept in straw yards. Similar differences were also reported when pre-partum heifers reared in cubicle yards were compared to those reared in straw yards (Webster 2001). Nearly all cows show signs of haemorrhage in early post-partum period, these haemorrhages resolves spontaneously when the effect of external stress on foot is minimum i.e., when cows are kept in pasture or straw yards. But when the effect of external stress on foot is higher, i.e. when animals are housed in concrete, when lying time is reduced these lesions progress to clinical lameness and CHD (Leach *et al.* 1998; Webster 2001).

Risk factors within the hard surface associated with CHD include the design and space of feeding troughs, stall configuration, stocking density (Cook and Nordlund 2009). All these factors increase the risk of CHD by altering the contact time between the hard surface and the hoof and therefore altering the risk of corium contusion.

## 2. Systemic Events around Parturition

It has long been recognised that most cases of lameness due to CHD are seen in the early post-partum period (Rowlands *et al.* 1985), with Sanders *et al.* (2009) observing that the incidence of CHD was highest in the first 60 days of lactation. This was commonly linked to changes in management, i.e. changes in housing and nutrition during the time around parturition, rather than parturition itself. Webster (2001) evaluated haemorrhage score in heifers by keeping them in two different housing (Cubicle Vs Straw yard) and two different feeding regimens (Wet vs Dry) from 4 weeks prepartum to 24 weeks postpartum. All groups of animals had a low haemorrhage score at 4 weeks before calving but after calving the haemorrhage score of all

heifers increased. The highest haemorrhage scores after calving were observed in cubicle reared heifers, with wet feeding producing higher haemorrhage scores only in animals reared in cubicles. Webster stated that his study showed that “systemic factors associated with calving and onset of lactation have a central role in the aetiology of claw horn lesions” and that the impact of these factors were modified by the environment in which an animal was kept.

The first study directly testing this claim was by Tarlton *et al.* (2002). They used histological, biochemical and biomechanical evidence to show that changes around parturition weakened the connective tissue of the hoof suspensory apparatus. They observed widened and distorted laminae in heifers beginning around from 2 weeks before calving and persisting to at least 12 weeks after calving. This process was not secondary to inflammation. The changes in the laminae led to lower rigidity, and higher laxity (displacement to initial support) of the supporting structures of the distal phalanx, with these mechanical changes reflecting alterations in the biochemistry of the connective tissue. Tarlton *et al.* (2002) suggested that the processes around parturition had a primary role in the development of hoof horn lesions after calving.

Knott *et al.* (2007) examined the effect of housing, feed and parturition on biomechanical, biochemical and pathological changes in claws. They identified that only housing and parturition were associated with haemorrhage score, consistent with Webster’s claim. The biomechanical and biochemical changes they observed were consistent with those reported by Tarlton *et al.* (2002) and they identified that both housing and parturition were associated with these changes. Knott *et al.* (2007) concluded that their results indicated that “parturition/lactation causes non-inflammatory changes in [connective tissue] that impair resilience of the feet to external stresses associated with poor housing”.

### 3. Hoof Overgrowth

Hoof overgrowth alters weight distribution causing changes in the pressure applied by the distal phalanx on the corium, increasing the risk of CHD (Shearer and van Amstel 2001). Animals housed in hard surfaces have hoof overgrowth because the pressure on the corium results in increased proliferation of hoof horn, resulting in increased horn growth (Blowey 2005). As this increase in horn growth is greater than the increased wear produced by contact with hard surfaces, cattle on hard surfaces generally develop hoof overgrowth (Toussiant Raven 1989).

Preventive hoof trimming was developed to restore the normal shape of the hoof and thus correct weight distribution, thereby reducing the risk of further corium damage (Toussaint

Raven 1989). Although it is far from clear which preventive trimming method is optimal (Stoddard and Cramer 2017), it is clear that hoof trimming can reduce the risk of lameness. Manske *et al.* (2002) conducted a two-year study in Swedish dairy cattle which compared the effect of an additional autumn trimming on claw health and lameness score (measured at spring trimming). They found that the additional trimming at autumn reduced the odds of lameness in spring by 1/3. Other studies which have shown that restoring hoof shape can reduce CHD, include study by Thomsen *et al.* (2019) who found that hoof trimming at dry-off diminished the odds of sole ulcer by 20 % in the subsequent lactation and Sadiq *et al.* (2021) found that preventive hoof trimming delayed the onset of lameness in non-lame cows by approximately one month in Malaysian dairy cows with and without access to grazing.

#### 4. The Digital Cushion and its significance for CHD risk

Having identified the key factors that directly increased the risk of inappropriate forces on the corium, Newsome *et al.* (2016) moved to factors which indirectly increased that risk, focusing especially on factors that reduced shock dissipation when the hoof and floor came into contact (foot-strike). In cows, the key structure responsible for shock dissipation during foot strike is the digital cushion (Räber *et al.* 2004). The digital cushions consist of three parallel layers of fat pads situated below the flexor tuberosity of distal phalanx. As the main component of the digital cushion is fat, it is highly likely that fat mobilisation will influence digital cushion thickness. All dairy cows mobilise their body fat in early lactation to meet the increased demands of energy to support milk production (Rastani *et al.* 2001).

Bicalho *et al.* (2009) in a cross-sectional study, showed that stage of lactation was strongly associated with the thickness of the digital cushion, with the mean thickness of the digital cushion decreasing from calving until the 4<sup>th</sup> month of lactation and body condition score (a measure of fat coverage) also showing similar reduction. This study thus showed that fat mobilisation in early lactation led to a reduced digital cushion which is likely to provide less protection of the corium against contusions.

This hypothesis has been confirmed by studies evaluating the association between digital cushion thickness and the risk of CHD. Bicalho *et al.* (2009) reported that cows in the lowest quarter of digital cushion thickness had an adjusted CHD prevalence of 24 % whereas those in the highest quarter had an adjusted prevalence of 8 %. This cross-sectional association was also seen in a prospective study by Machado *et al.* (2011). They showed that the thickness of digital cushion at dry off was associated with the incidence of CHD in the subsequent lactation. Cows

in the lowest quarter of digital cushion thickness had an adjusted CHD incidence above 20 %, whereas cows in highest quarter had an incidence of <10 %.

Meeting the demands of early lactation is not the only reason for increased body fat mobilisation. Newsome *et al.* (2016) also identified that anything that reduced dry matter intake such as other causes of lameness was also a risk factor for the development of CHD. Lame cows spend less time for feeding and thus reduced DMI (Bach *et al.* 2007; Sepúlveda-Varas *et al.* 2018), which leads to a loss in BCS and digital cushion thickness, with this perpetual cycle further thinning the digital cushion.

However, loss of BCS is not the only reason for thinning of digital cushion of claws. Newsome *et al.* (2017a) in a longitudinal cohort study observed the association between digital cushion thickness and back fat thickness and showed that the timing of the post-partum nadir were different for the two measurements (first week and ~9 weeks post calving, respectively). In addition, although Newsome *et al.* (2017a) found a positive correlation between back fat thickness and digital cushion thickness, the effect was very small (10 mm change in back fat thickness produced 0.13 mm change in digital cushion thickness). In a subsequent study, Newsome *et al.* (2017b) identified that both digital cushion thickness and back fat thickness could independently affect the risk of CHD.

Newsome *et al.* (2016) identified inflammation as a potential reason for a difference between back fat thickness and digital cushion thickness. They pointed out that inflammation was an outcome of corium damage and proposed that this inflammatory reaction could utilise the fat tissue from the digital cushion to produce prostaglandins (mediators of inflammation) as arachidonic acid (a key precursor of prostaglandins) is present in the digital cushion (Räber *et al.* 2006). Newsome *et al.* (2016) hypothesised that utilisation of the fat in the digital cushion in this way would result in the thinning of the digital cushion and therefore a reduction in its effectiveness. Although plausible, there is little published evidence to support this proposal. Räber *et al.* (2006) reported that the digital cushion in heifers contained higher proportion of arachidonic acid than that of adult cows. As heifers are much less likely to have encountered CHD than older cows, and, therefore, less likely to have had local utilisation of the digital cushion's arachidonic acid, this is consistent with Newsome's hypothesis, especially as Räber *et al.* (2006) found no similarly age-related difference in fats from perirenal and subcutaneous adipose tissue. However, the study by Räber *et al.* (2006) was a small-scale preliminary study

and more data are needed on fatty acid composition of the digital cushion and the importance of these fatty acids in modulating the response to CHD.

## CHD and exostosis

The final factor identified by Newsome *et al.* (2016) as increasing the risk of subsequent lameness was that the inflammation associated with lameness resulted in bone development on the distal phalanx and that this bone development could increase the risk of inappropriate forces on the germinal epithelium of the sole.

Bony exostoses of the third phalanx have been long been considered to occur as a response to hoof pressure. Rusterholz (1920) suggested that deformation of the distal phalanx, due to increased bony tissue resulting from chronic inflammation, could be a principal driver of sole ulcer, while Maclean (1970) identified exostosis at the level of the extensor process and flexor tuberosity of the distal phalanx in beef cows with laminitis. Toussaint Raven (1989) reported that the surface of the distal phalanx of the hind limbs became rougher with age, particularly those of the lateral claws. He stated that the age-related changes were the result of the accumulation of the forces applied over time and that the increased change in lateral claws reflected the more irregular and greater forces on those claws.

Lischer *et al.* (2002) in an anatomical study, reported “variable degrees of excessive bone formation” on the surface of the distal phalanx where the suspensory apparatus was attached to the bone. They also reported that cows with sole ulcers had more pronounced exostoses. As displacement of the distal phalanx into the corium is the main cause of the contusions of the corium which ultimately lead to CHD (Lischer *et al.* 2002; Newsome *et al.* 2016), exostoses on the distal phalanx will increase the degree of damage produced by distal phalanx displacement. The sites on the distal phalanx where exostoses are likely to be most important are on the palmar/plantar surface of the flexor tuberosity, and on the abaxial edge of the distal phalanx. This is because it is contact between these sections of the phalanx and the corium which are the principal drivers of sole ulcer and white line disease, respectively (Lischer *et al.* 2002).

However, despite this long-assumed association between exostoses and lameness it was not until 2012 that a quantitative study of bone development of the distal phalanx of hindlimbs was first undertaken.

## Quantitative evaluation of bone development of the distal phalanx of the cow hind limb using computed tomography (Tsuka *et al.* 2012)

The aim of this study was to quantitatively evaluate the bone developments using  $\mu$ -CT and to represent horizontal and vertical progression; and to study the effects of housing, claw and age on these measurements.

Tsuka *et al.* (2012) collected data from 100 pairs of hind limbs that were collected from the abattoir. The data they had available for this convenience selection of cows were age (23 to 143 months), housing (67 from tie stalls and 33 from free stalls or group housing) and breed (all Holstein-Friesian). No data were available on previous history of lameness, although Tsuka *et al.* (2012) did recorded the presence/absence of sole ulcers (observed in 7 cows and 9 claws, all from tie stall farms).

Three measures of bone development on the third phalanx in the region of the flexor tuberosity were investigated: vertical bone development; the ratio of this vertical development to the thickness of the corium and the subcutis; and the ratio of horizontal bone development to the width of the flexor tuberosity. All measurements were made using  $\mu$ -CT.

The initial analysis of vertical bone development excluded all claws with a sole ulcer and analysed the effect of housing type and of medial vs lateral claw. This analysis was then repeated for claws with a sole ulcer. The correlations between age and each of the four bone development measures were then calculated, as were the correlations between horizontal and vertical development ratios (separately for both lateral and medial claws).

The first analysis identified that vertical development was influenced by claw. Mean vertical development across all medial claws was 2.38 mm while that for all lateral claws without sole ulcer was 2.73 mm (mean difference 0.35 mm; 95%CI<sup>1</sup> 0.12 to 0.58). The impact of housing was less clear. Mean vertical development across medial claws was very similar in both housing types (2.37 vs 2.39 mm for tie stalls and free stalls/group housing, respectively). For all non-ulcerated lateral claws, vertical development was 0.14 mm greater (2.78 mm vs 2.64 mm) in cows housed in tie stalls (95%CI<sup>2</sup> -0.26 to 0.54). Thus, although Tsuka *et al.* (2012) claimed they had found no effect of housing, their data are actually compatible with the difference in

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<sup>1</sup> Assuming no correlation between claws within each cow and that bone development in the medial claw is independent of that in the lateral claw

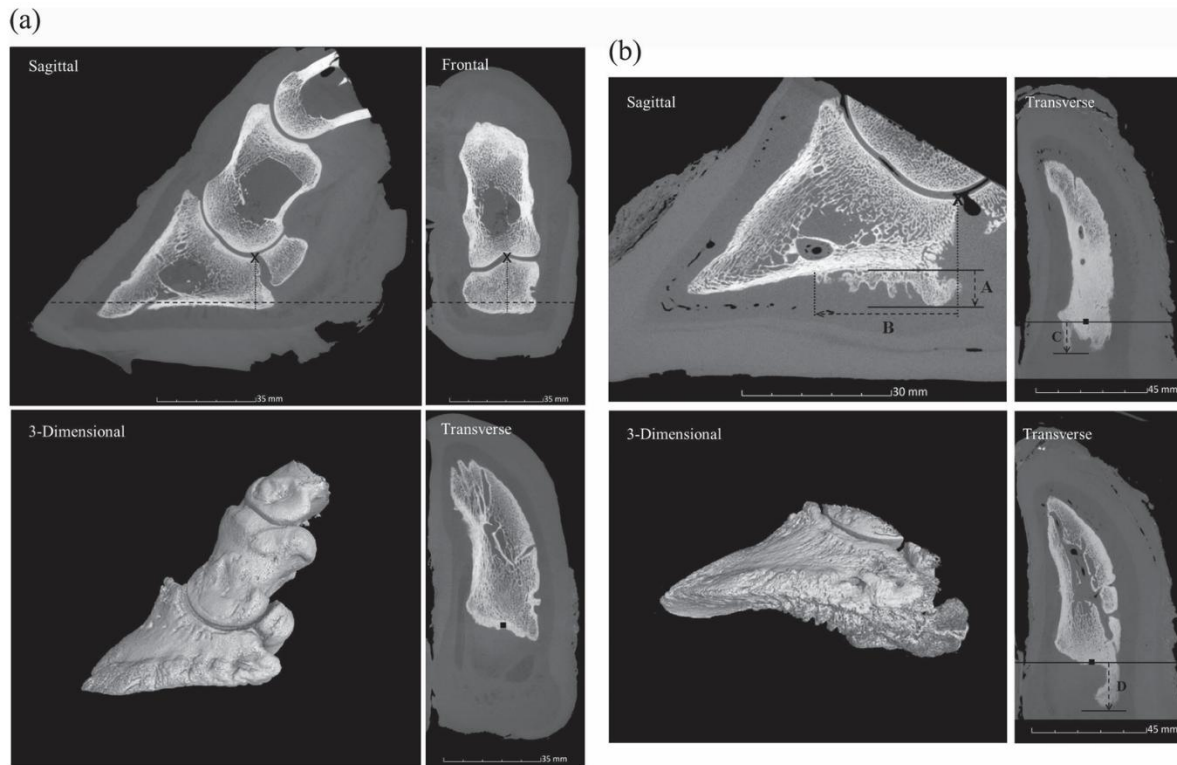
<sup>2</sup> Assuming no correlation between claws within each cow

vertical bone development between the lateral claws of the cows in the two housing groups being similar to the difference between medial and lateral claws across all cows. Claws from cows with sole ulcers had markedly increased vertical development compared to claws from cows without sole ulcers. Mean vertical development in lateral claws (without sole ulcers) of cows kept in tie stalls was 2.78 mm, whereas that for claws with sole ulcers (all in lateral claws of cows kept in tie stalls) was 7.06 mm (mean difference 4.28 mm; 95%CI<sup>2</sup> 3.03 to 5.53). For their ratios, Tsuka *et al.* (2016) reported that the presence of a sole ulcer and coming from a tie stall farm both increased their vertical bone ratio (greater in cows from tie stalls) while for horizontal bone ratio they only identified an effect for the presence of a sole ulcer. However, the large variance in these ratios within groups and the lack of accounting for repeat measures means that it is difficult to simply test the robustness of their claims for these ratios (whether they found an effect or not) except for the presence of sole ulcer.

The issues with the statistical analysis continued for their correlation of age and bone development. They simply reported Pearson's *r*, with no confidence intervals or accounting for the likelihood that age will affect hind claw bone development similarly in the left and right hind claw. The latter means that calculating confidence intervals from their data are not simple. This, combined with the choice of correlation rather than regression-based analysis, means that, although their study does support the suggestion that age increases vertical bone development, vertical bone ratio and horizontal bone ratio, it provides little detail on the impact of age on those outcomes.

### Linking bone development of the caudal aspect of distal phalanx with lameness during life (Newsome *et al.* 2016)

The aim of this study was to identify whether bone developments on the caudal aspect of the distal phalanx were associated with history of CHD. They evaluated, using  $\mu$ -CT, claws from 38 Holstein-Friesian cows which all originated from one farm. The data available for them were management (partly housed vs. fully housed); age; genetic line; culling reason; and history and cause of lameness, as well as weekly locomotion scores for the 12 months prior to culling. Four bone development measurements (A to D) were collected from all hind claws of each cow (see **Figure 2**). Two outcome variables were created: (1) BD-Max (largest sum of the four measures for an individual claw), and (2) BD-Ave (mean sum across all 4 claws).



**Figure 2:** Measurements of bone growth at 4 locations (A to D). From Newsome *et al.* (2016). A is the vertical bone development from base of distal phalanx. A vertical line is drawn trochlear ridge of interphalangeal joint downwards and this landmark became the base for all other measurements B, C and D. The vertical line drawn culminates in square dot (■) in transverse plane. B is the bone development from the base of distal phalanx towards toe taken in sagittal plane. C is axial bone development from caudal to vertical line and D is the abaxial bone development from the vertical line taken in transverse plane.

The results of three models used to estimate the effect of lameness history on BD-Max were reported: one which included any history of CHD (yes (12 cows) vs no (26 cows)), one which used a cow's individual percentage of weekly locomotion scores with severe lameness in the 12 months prior to slaughter, and one which categorised that percentage. The same models were reported for BD-Ave. None of the predictor variables included alongside lameness history were retained in the final model, except for age, so for all six models, the final model included only age and lameness history. For the model with a history of CHD as the lameness history predictor, a history of CHD increased BD-Max by 7 mm (95%CI 2.7 to 11.7) and BD-Ave was by 3.6 mm (95%CI 0.9 to 6.2), whereas an increase of 1 year in age was associated with an increase in BD-Max of 2.5 mm (95%CI 0.8 to 4.1) and BD-Ave of 1.2 mm (95%CI 0.2 to 2.2). The balance between the impact of lameness history and age on bone development was similar across all models and Newsome concluded that “age explained the majority of the variation in bone development”, with lameness history modifying that development. They also

concluded that bone development was specific to CHD, as history of infectious lameness was not retained in the final model.

Thus, taken together, the results of Tsuka *et al.* (2012) and Newsome *et al.* (2016) strongly support the hypothesis that bone development on the distal phalanx is a consequence of age, and that lameness history is also important. The findings of the two studies are thus consistent with the claim by Newsome *et al.* (2016) that bony development is an inflammatory response either to direct trauma “due to insufficiencies in the force-dissipating structures surrounding the flexor tuberosity” (probably the route by which age increases bone development) or to “macrophage action in surrounding tissues” in response to inflammation during active CHD. All of the cows in these two studies were principally housed in stall systems with persistent exposure to concrete which increases the risk of direct trauma and development of CHD. Cows kept permanently at pasture have much less exposure to concrete and would therefore have less risk of direct trauma and therefore a reduced association between age and bone development. Therefore, studying the association between age and bone development on the distal phalanx in pasture-based dairy cows could provide a useful test to determine whether direct trauma is likely to be the cause of the association between age and bone development. In addition, both previous studies used  $\mu$ -CT to measure bone development. This is an extremely costly method of measurement; in contrast digital photography is a much cheaper method which may provide similar outcomes.

The aim of this study was to assess the effect of age and previous lameness history on bone development (as measured using digital photography) in cows kept at pasture and to assess the practicalities of using digital photography to measure bone development.

# Material and Methods

Hind feet were collected after slaughter from a convenience sample of 40 culled cows from 5 farms in Taranaki, New Zealand. Age and lameness history in the current lactation were recorded for all cows prior to slaughter. After collection, the distal phalanxes were separated from the hind feet by simmering in water for 90 minutes and then allowed to cool. The distal phalanges were then rinsed and dried before being placed into separate labelled bags

## 1. Measurement of Bone Growth

Digital photographs of each distal phalanx were then taken with each phalanx in a standardised position within a bespoke photographic box (as per Laven *et al.* 2015), which allowed the bone to be positioned and clamped at a constant distance from the mounted camera (Sony A6400, Sony Inc, Tokyo, Japan). A ruler, placed vertically near the bone sample was used for calibrating the measurement and setting the scale in ImageJ (NIH, MD, USA). Images were taken of abaxial and axial aspects of the distal phalanx (see **Figure 3** and **Figure 4**). All measurements and images were taken by the same observer. Four linear measurements (A to D) were then taken of the maximum extent of bone development at four locations, based on the approach by Newsome *et al.* (2016) (See Fig. 3 to 4).

- 1) Measurement A: Vertical bone development measured from a line parallel to the normal bone contour of the distal phalanx (see **Figure 3**).
- 2) Measurement B: Extent of abnormal bone development distally (toward the toe) along the base of the distal phalanx, starting at a vertical line drawn from the lateral peak of the articular surface with the distal sesamoid bone (See **Figure 3**).
- 3) Measurement C: Caudal bone development on the axial aspect of flexor tuberosity measured from a vertical line drawn from the caudal most point of the trochlear ridge (See **Figure 4**).
- 4) Measurement D: Caudal bone development on the abaxial aspect of the flexor tuberosity measured from the same vertical line as measurement B (See **Figure 3**).

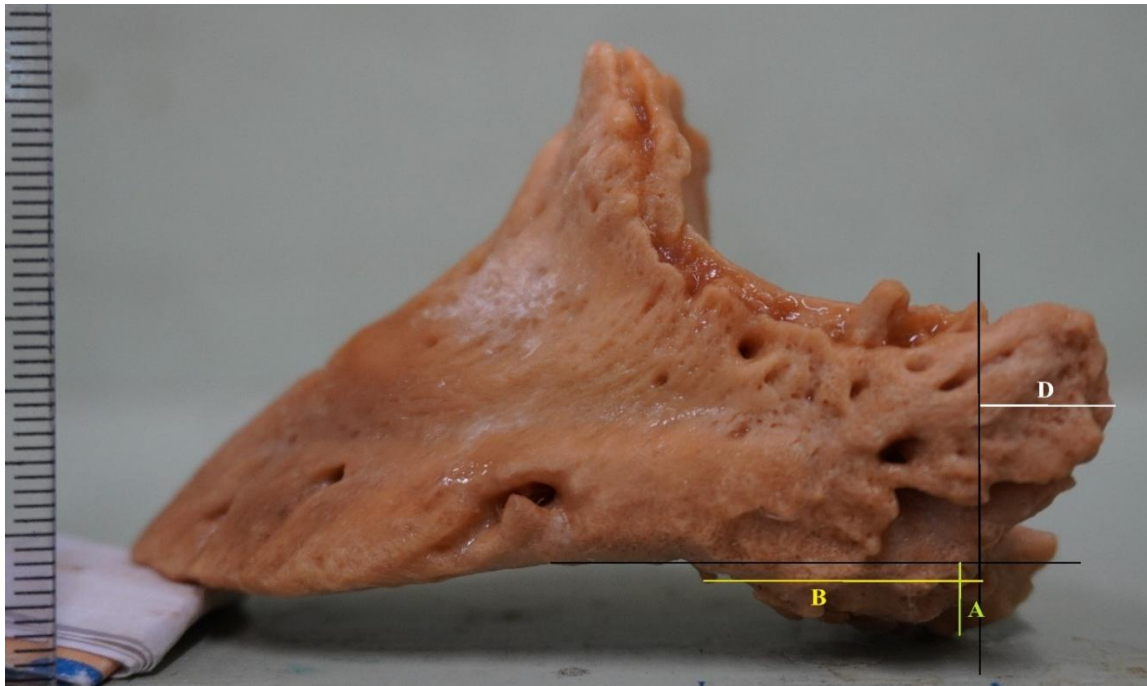


Figure 3: Abaxial image of distal phalanx. Measurement A: From the contour of the distal phalanx (horizontal black line) to the greatest extent of bone (green line). Measurement B is the distance from a line drawn from the lateral peak of the articular surface with the distal sesamoid bone (vertical black line) to the most cranial point of bone development (yellow line). Measurement D is the greatest extent of caudal bone development from the vertical black line (white line).

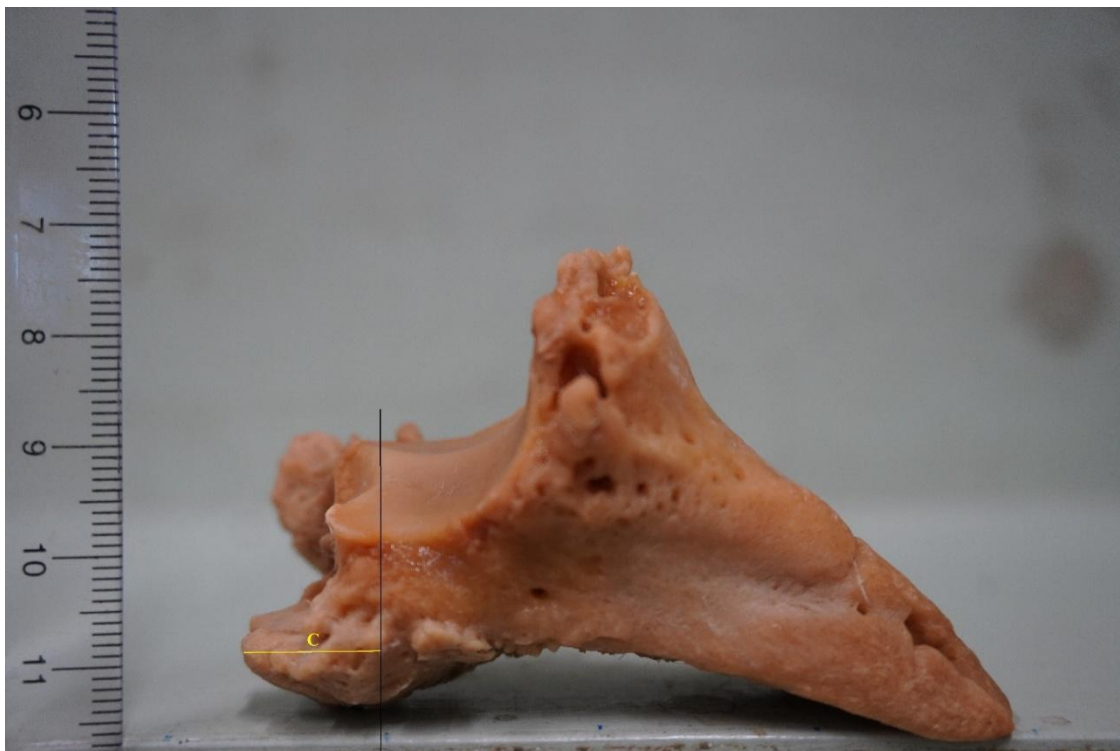


Figure 4: Abaxial image of distal phalanx. Measurement C is the distance from a line drawn from the caudal point of the trochlear ridge (black vertical line) to the most caudal point of bone development.

## 2. Exploratory Data Analysis

All records, i.e. Cow ID, claw, limb, age and history of lameness as well as measurements A, B, C and D were entered into an Excel spreadsheet (Microsoft, Redmont, USA). The sum of the measurements A to D were then calculated for every claw, followed by the average sum of A to D for each pair of medial claws and pair of lateral claws within a cow (medial Ave BG and lateral Ave BG, respectively) and the largest score for an individual claw within a cow (BG max).

## 3. Association between Ave BG/BG max and age and lameness history

This was analysed using two linear mixed models with either Ave BG or BG max as the outcomes. For BG max, the included predictor variables were lameness history, and age, while for Ave BG, claw was included as a predictor variable alongside age and lameness history, with cow being included as a random variable to account for the multiple measurements within each cow. Both outcome variables were log transformed before inclusion in the model, with the residuals being checked for normality using Q-Q plots and histograms. Back transformed exponentiated coefficients are reported for all variables.

## 4. Factors affecting measurement D and sum of A, B and C

Two separate models (one for lateral claws and one for medial claws) were created to assess the effect of exostoses in sites A, B or C and bone development at site D. For both models the outcome was natural log bone development at site D with the presence or absence of exostoses (i.e.  $A + B + C = 0$  or  $>0$ ) as the predictor variable. Cow was used as a random effect to account for clustering and residuals being checked for normality using Q-Q plots and histograms. Back transformed coefficients are reported.

A linear mixed model was then used to assess the effect of age on measurement D in claws without other exostoses (i.e.  $A, B, C = 0$ ). The outcome of the model was log transformed D measurements, with the predictor variables being age, claw type and lameness history with cow being included as random variable to account for clustering. Back transformed coefficients are reported for continuous variable (age).

A two-stage hurdle model was then used to assess the effect of age and lameness history on the sum of A, B and C. The first stage was a binary logistic regression model with  $A + B + C = 0$

or  $>0$  as the binary outcome variable and age and claw type as the predictor variables and cows as a random variable. The second stage was a linear mixed effect model which included only cows where  $A + B + C >0$ , with log transformed sum of A, B and C as the outcome variable and age and claw as the predictor variables and cows as the random variable. Lameness history was not included in both models as no claws from cows without lameness history had  $A+B+C >0$ . Back transformed coefficients are reported.

All analyses were undertaken using RStudio Version 4.5 (Posit Software, Boston, USA).

## Results

Across all 40 cows, median age was 8 years (range: 3 to 12 years), and 34/40 had a history of lameness in the lactation prior to culling. **Table 1** presents the descriptive statistics for measurements of A, B, C and D for all 160 distal phalanges along with Ave BG and BG Max. Out of the 160 distal phalanges measured, 136 had zero values for measurements A, B and C. Of these 136 phalanges, 77 were from medial claws and 59 were from lateral claws, i.e. medial claws were 1.3 (95%CI 1.1 -1.5) times more likely of having zero values for A, B and C than lateral claws. Of the 40 cows, 24 had no A, B or C measurements >0 mm, including all six cows with no history of lameness during the previous lactation. In contrast, measurement D was >0 mm for all 160 phalanges. The claw with the greatest bone development (BG Max) was a lateral claw in 27 cows and a medial claw in 13 cows, i.e. the proportion of claws with the BG Max that were lateral claws was 0.65 (95%CI 0.48 – 0.79). The highest value of BG Max observed among the whole group was 58.3 mm (See **Figure 5** and **Figure 6**).

**Table 1:** Descriptive data of bone development measures (in mm) at the claw level ((A to D) and BD-Ave) and at the cow-level (BD-Max), for 160 digital phalanges from 40 cows.

Measure ment	Claw	Mean	Minimum	Lower Quartile	Median	Upper Quartile	Maximum
A	Lateral	0.37	0	0	0	0	4.5
	Medial	0.05	0	0	0	0	1.82
B	Lateral	2.52	0	0	0	0	18.1
	Medial	0.43	0	0	0	0	14.8
C	Lateral	1.26	0	0	0	0	24.8
	Medial	0.08	0	0	0	0	6
D	Lateral	7.59	2.48	4.8	7.55	9.35	18.1
	Medial	7.15	2.7	5.05	6.82	8.97	12.8
BG Max		15.92	3.69	6.16	10.74	21.99	58.3

Measurement	Claw	Mean	Minimum	Lower Quartile	Median	Upper Quartile	Maximum
Ave BG	Lateral	11.77	2.85	5.44	8.70	14.87	41.7
	Medial	7.71	2.81	5.33	7.2	9.67	18.8

Measurement A: vertical bone development on plantar aspect of flexor tuberosity. Measurement B: bone development on base of distal phalanx measured towards toe. Measurement C: axial bone development caudal to trochlear ridge of interphalangeal joint. Measurement D: abaxial bone development caudal to of the lateral peak of the articular surface with the distal sesamoid. BG Max is the highest value for an individual claw for sum of A to D across all four distal phalanges. Ave BG is the average sum of A to D for each pair of medial claws and pair of lateral claws within a cow.



**Figure 5:** Abaxial image of left lateral distal phalanx of cow 168. Highest BG Max was obtained for this claw. The measurements A, B, C & D observed for the claw was 3.95, 14.19, 24.84 and 15.27mm respectively. BG max is the maximum value obtained for an individual claw within a cow for the sum of all four bone development measurements (i.e. A, B, C and D; see **Table 1** for definitions).

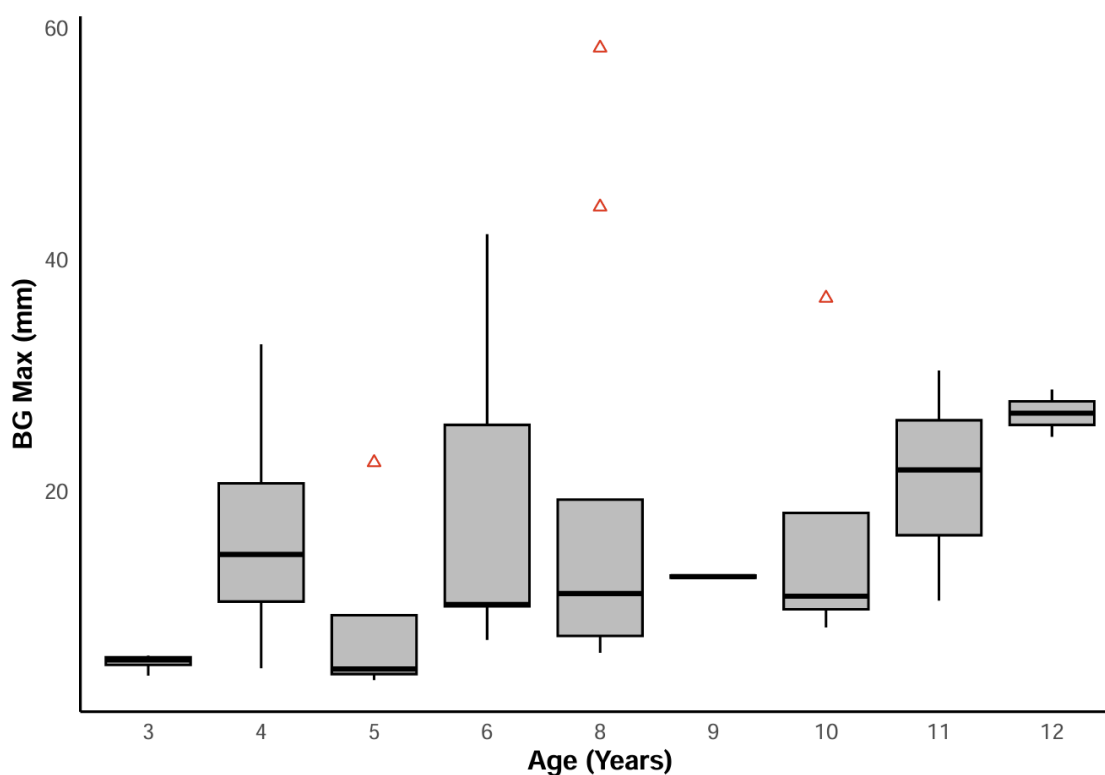


**Figure 6:** Solar image of left lateral distal phalanx of cow 168 showing exostosis on caudal aspect of flexor tuberosity

## Statistical Modelling

### BG Max

Boxplots of the raw data for BG max by age are presented in **Figure 7**. The outcomes from the linear model for  $\ln$  BG Max along with the back transformed estimate of the effect size are presented in **Table 2**. Our model suggests that increased age was associated with an increase in BG Max, with an increase of 1 year in age increasing BG Max by 1.12 times (95 % CI: 1.03-1.21). For lameness history, although the point estimate of our effect of lameness on BG Max (1.61 times higher than cows with no lameness history) was equivalent to our point estimate of a 5-year increase in age ( $1.61 \approx 1.12^5$ ), our data were also compatible with lameness being associated with a small, biologically unimportant decrease in BG Max (95 % CI: 0.88 to 2.95).



**Figure 7:** Boxplot showing BG Max across different age groups of animals. Box represents first quartile (25<sup>th</sup>) to third quartile (75<sup>th</sup>). Line inside the box represents median and whiskers represents 1.5 times the interquartile range. Red markers represent values that falls outside the 1.5 times interquartile range. BG max is the maximum value obtained for an individual claw within a cow for the sum of all four bone development measurements (i.e. A, B, C and D; see **Table 1** for definitions).

**Table 2:** Results from the linear regression model for the effect of age and history of lameness in the lactation preceding culling on BG Max (mm) in 40 cows.

Predictor	Estimate	SE	P-Value	95 % CI	Back transformed estimate of effect (95%CI)
Intercept	1.27	0.36	0.001	0.55 to 2.0	
Age	0.11	0.038	0.006	0.034 to 0.19	1.12 (1.03 - 1.21)
Lameness History: N	Referent				
Lameness History: Y	0.478	0.29	0.12	-0.12 to 1.08	1.61 (0.88 – 2.95)

BG Max is the maximum value obtained for an individual claw within a cow for the sum of all four bone development measurements (i.e. A, B, C and D; see **Table 1** for definitions). For the analysis, BG Max was natural log transformed, with the effect size being calculated by back transforming the model estimate. Interpretation of effect size: For age, every increase of 1 year in age increases BG max by 1.12 (1.03 -1.21) times, while a history of lameness increases BG max by 1.61 (0.88 -2.95) times.

### Ave BG

The outcomes from the linear model for ln Ave BG along with the back transformed effect estimates (and CI) are presented in **Table 3**. Similar to BG Max, our model for Ave BG suggests that increased age was associated with an increase in Ave BG, with an increase of 1 year in age increasing Ave BG by 1.09 times (95 % CI: 1.05-1.15). For lameness history, although the point estimate of lameness history on Ave BG (1.25) was equivalent to a 3-year increase in age ( $1.25 \approx 1.09^3$ ), our data were also compatible with lameness being associated with a small, biologically unimportant decrease in Ave BG (95 % CI: 0.89 – 1.78).

**Table 3:** Results from the linear mixed model for the effect of age, claw (lateral vs medial) and history of lameness in the lactation preceding culling on Ave BG (mm) in 40 cows.

Predictor	Estimate	SE	P-Value	95 % CI	Back transformed estimate of effect (95%CI)
Intercept	1.39	0.22	<0.001	0.97 - 1.82	
Age	0.091	0.024	<0.001	0.045 - 0.13	1.09 (1.05 -1.15)
Claw: Lateral	Referent				
Claw: Medial	-0.281	0.086	0.002	-0.45 to -0.1	0.76 (0.64-0.90)
Lameness history: N	Referent				
Lameness history: Y	0.229	0.18	0.21	-0.12 to 0.58	1.25 (0.89-1.78)

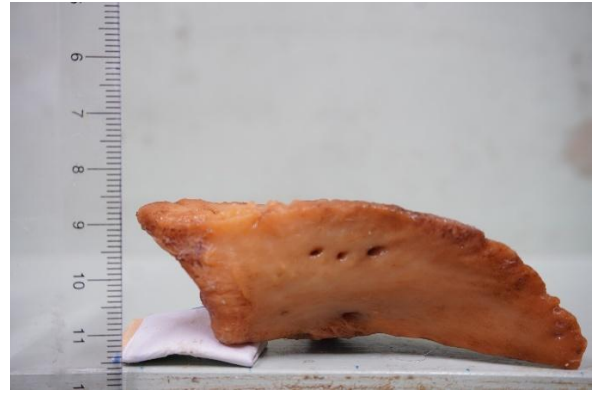
Ave BG is the average value obtained for an individual cow for the sum of all four bone development measurements (i.e. A, B, C and D; see **Table 1** for definitions) for each claw. For the analysis, Ave BG was natural log transformed, with the effect size being calculated by back transforming the model estimate. Interpretation of effect size: For age, every increase of 1 year in age increases Ave BG by 1.09 (1.05 -1.15) times, while a history of lameness increases BG max by 1.25 (0.89 – 1.78) times.

## Measurement D

As noted earlier, measurement D was greater than zero in all of the measured distal phalanges despite most phalanges (136/160) having measurements A, B and C all being 0. **Figure 8** and **Figure 9** shows the abaxial and solar image of a left medial distal phalanx with a D-value of 12.3 mm and zero values for all other measurements. Even though the D value was large, the appearance of the bone associated with the protrusion was smooth without rough surfaces or pointed edges.

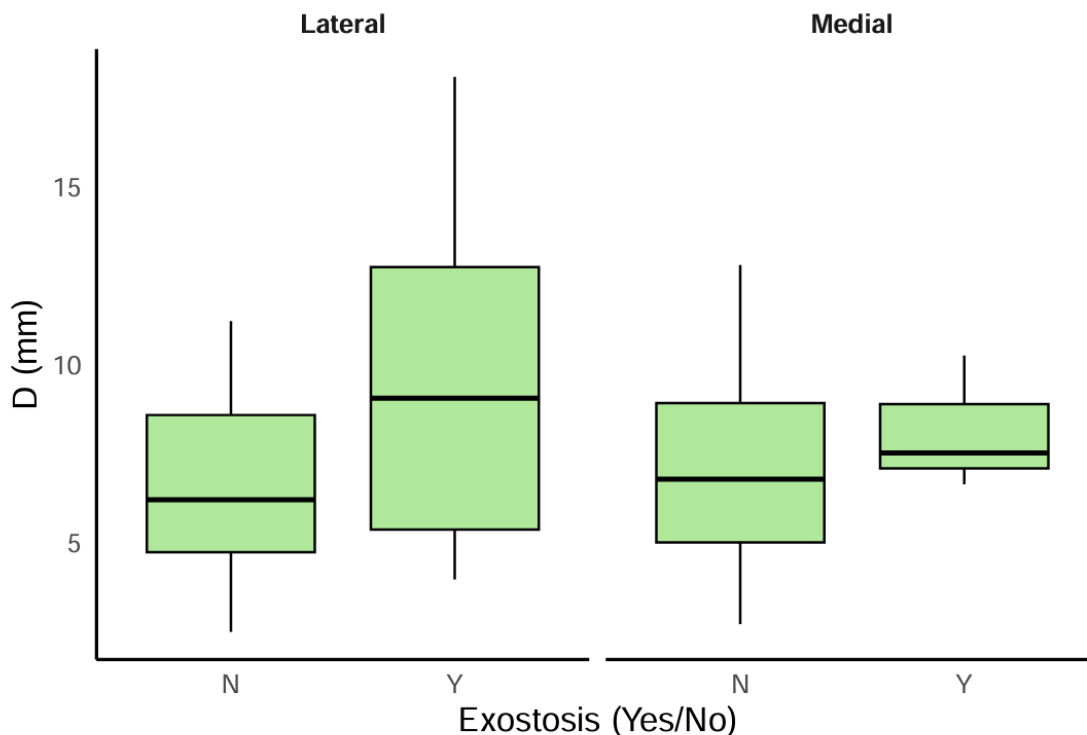


**Figure 8:** Abaxial view of Left Medial distal phalanx of animal 82 showing caudal bone development with smooth surface.



**Figure 9:** Solar view of left medial distal phalanx of animal 82 showing smooth appearance without any bony outgrowths.

**Figure 10** presents observed D values for lateral and medial claws with and without bony development at sites A, B or C. For lateral claws, D-values in claws with bone development at sites A, B or C were 1.34 (95%CI 1.12–1.61) times greater than in those with no bony development. The equivalent figures for medial claws were 1.17 (95%CI 0.92 -1.50) times greater. In claws without bone development at sites A, B or C, the effect of age (after accounting for claw and lameness history) was that every increase of 1 year in age increased D measurements by 1.09 (95%CI 1.05 – 1.11) times.



**Figure 10:** Observed D values for animals which had either measurements greater than zero for A, B or C on lateral / medial distal phalanx (Lateral/Medial exostosis: Y) or zero values for A, B or C in lateral / medial distal phalanx (Lateral/Medial exostosis: N). Box represents first quartile (25th) to third quartile (75th). Line inside the box represents median and whiskers represents 1.5 times the interquartile range. See **Table 1** for definitions of measurements.

### Effect of Age on sum of A, B and C

The binary logistic regression identified that an increase of 1 year in age was associated with an increase in the odds of the sum of A, B and C >0 (OR 1.15; 95%CI 0.97 to 1.37), although our data are also compatible with no biologically meaningful association. Medial claws were associated with lower in odds of having sum of A, B and C >0 (OR 0.11; 95%CI 0.03 to 0.33) compared to lateral claws.

The second model identified that age and claw were all associated with an increase in the sum of A, B and C in claws where the sum was >0, with a 1-year increase in age being associated with increase in sum of ABC by 0.96 (95 % CI: 0.84 to 1.1) times, though as for the odds of the sum A, B and C >0, our data were also compatible with no biologically meaningful effect. Medial claws were having estimated 0.78 times sum of A, B and C (95% CI 0.42 to 1.7) compared to lateral claws, although our data were also compatible with no biological meaningful effect.

## Discussion

The two key aims of this study were to identify whether digital photography instead of  $\mu$ -CT could be used to measure the bone development on the distal phalanx identified by Newsome *et al.* (2016) and, then to assess whether, as identified by Newsome *et al.* (2016) in housed cows, whether age, and lameness history also influence bone development on the distal phalanx of cows kept on pasture.

Whereas Newsome *et al.* (2016) used three cross-sections (sagittal, transverse, and frontal) from the  $\mu$ -CT image to ensure landmarks and measurements were consistent; we used two digital photographs to duplicate their measurements. For measurement A, B and D we used an abaxial image of the distal phalanx (**Figure 3**), while for measurement C we used an axial image (**Figure 4**). The principal issue with transferring the measurements made using  $\mu$ -CT to a digital photograph was in ensuring that there was consistency in the starting points of the measurements, as unlike with  $\mu$ -CT, we could not identify a landmark in one view and then transfer this to other views to ensure a consistent starting point for measurement.

For measurement A, Newsome *et al.* (2016) measured the vertical development of the bone from the “contour of the cortical bone, on the plantar aspect of the flexor tuberosity”. This can be directly visualised in the sagittal cross-section of a  $\mu$ -CT image, but not in an abaxial view of a digital photograph. Our line was therefore drawn from the normal bone contour visible in the centre of the distal phalanx (see **Figure 3**). It is thus likely that our results are marginally different from those we would have obtained using  $\mu$ -CT; however, the vertical differences in these two points in normal hooves is generally small ( $\sim 1$  mm).

For the other three measurements (B, C and D), Newsome *et al.* (2016) used the caudal most point of the trochlear ridge (identified in the frontal cross-section) as the consistent landmark from which to start their measurement. With  $\mu$ -CT, this point could be identified in the frontal cross-section and then transferred to each viewpoint. However, with digital photography this is not possible, and we were reliant on being able to visualise the caudal most point of the trochlear ridge in order to use it as a consistent landmark. This applied particularly to the solar view, so whereas C and D were measured using the transverse cross-section by Newsome *et al.* (2016), we used the axial view for C and the abaxial for D.

For measurement C we were able to consistently identify the caudal most point of the trochlear ridge using the axial view (see **Figure 4**), so we drew our vertical line from this point. As such,

it is likely that our results for measurement C are close to those that would have been obtained had we used a  $\mu$ -CT. In contrast for measurement D, we were not able to consistently identify the caudal most point of the trochlear ridge using the abaxial view (see **Figure 3**). Instead, we used the highest point of the articular surface of the distal phalanx with the distal sesamoid, i.e. the lateral most point of that articular surface (Sisson 1921) (**Figure 11**). This point is caudal to the caudal point of the trochlear ridge, so, on average our measurement D is likely to be 2.8 mm (95% CI 2.5 to 3.1 mm) less than it would have been had we used  $\mu$ -CT (data not shown). As we used the same landmark for measurement B (see **Figure 3**), the impact on that measurement compared to using  $\mu$ -CT is the same as that on measurement D, but in a positive direction. Thus, for claws with a value of measurement B  $>0$ , our choice of starting point would have no effect on the sum of measurements for individual claws or on BG Max or Ave BG. However, where B = 0 (138/160 claws), the sum of all measurements for a claw would be reduced as would Ave BG and, potentially, BG Max.

The difference between using the caudal end of the trochlear ridge and our landmark is a relatively high proportion of measurement D ( $\sim 40\%$  at our mean measurement D of 7 mm). Nevertheless, we chose not to adjust our figure to replicate that of Newsome *et al.* (2016) by measuring the distance between the two landmarks for each individual claw and then reducing measurement D by that distance for two reasons. Firstly, to do so would require an additional photograph (dorsal view) and more measurements, increasing complexity and the time taken. Secondly, we question whether the caudal point of the trochlear ridge is the optimal site to measure caudal bone development especially on the abaxial surface of the claw. As in all cases our landmark was caudal to the end of the trochlear ridge, measuring from that ridge would have resulted in normal bone being included in measurement D. We thus believe that our measurements of B and D are probably more closely related to actual abnormal bone development than those of Newsome *et al.* (2016).



**Figure 11:** Dorsal View of distal phalanx showing landmarks identified by Newsome *et al.* (2016) and the present study. The point indicated by blackline shows the caudal aspect of trochlear ridge identified by Newsome *et al.* (2016) used for measurement B, C & D. The white line indicates lateral peak of the articular surface with the distal sesamoid bone (landmark for measurement B & D in present study). The green line shows the distance between two landmarks (See **Table 1** for definitions of measurements).

Nevertheless, this study has shown that it is possible to (relatively closely) duplicate the measurements made by Newsome *et al.* (2016) using  $\mu$ -CT by using digital photography. The significant advantage in cost of digital photography is not outweighed by the greater simplicity of obtaining the measurements once  $\mu$ -CT images have been made.

We thus believe that we have obtained a set of results that are comparable with those that we would have obtained had we used  $\mu$ -CT. Furthermore, our findings in relation to the effect of age and lameness history on maximum and average outcomes of bone development are consistent with those reported by Newsome *et al.* (2016). Direct comparison is not simple as we used log-transformed outcomes while Newsome *et al.* (2016) used untransformed outcomes and our lameness variable (any lameness in the lactation prior to culling) was not the same as any of those used by Newsome *et al.* (2016) which were based on locomotion scoring or any history of CHD. Nevertheless, comparisons are possible. Newsome *et al.* (2016) reported that, in their model with history of CHD as the lameness variable, a 1-year increase in age increased BD Max by 2.46 mm (95%CI: 0.81 - 4.1), while we found that an increase of 1 year in age resulted in an increase in BG max of 1.12 (95%CI: 1.03 - 1.21) times. Thus, our model predicts that, for a cow with BG Max of 10 mm (close to the median value in our population and that of Newsome *et al.*, 2016), an increase of 1-year in age will increase BG max by 1.2 mm (95%CI: 0.3 – 2.1). Given the difference in outcome measures, measurement methods, and that both populations were a convenience selection, our data provide only limited support for the hypothesis that age-related bone development is different in pasture-based and housed cattle. Further data are required.

For lameness, Newsome *et al.* (2016) reported that, in their model with history of CHD as the lameness variable, previous history of CHD increases BD Max by 6.98 mm (95%CI: 2.73 – 11.2), whereas we found that a history of lameness in the lactation prior to culling increased BG Max by 1.61 times (95%CI: 0.88 – 2.95), or compared to a cow with a BG Max of 10 mm and no history of lameness, an identical cow with a history of lameness would be expected to have a BG Max 6.1 mm higher (95%CI: -1.2 to 19.5). These data thus provide very little support for the hypothesis that the impact of lameness on BG Max is different between housed and pasture-based cows. However, this analysis is limited by the small number of cows in our population that had no lameness history. Again, further data are needed to better establish whether the impact of lameness on bone development is the same in housed cows and pasture-based ones.

Consistent with Newsome *et al.* (2016), in our population most claws 136/160 distal phalanges had no bony development at A, B or C, despite all claws having bone caudal to the caudal end of the trochlear ridge. In most cases this bony development was smooth and had no pointed or rough edges (see **Figure 8** and **Figure 9**). We thus suspect that much of this caudal bone development is normal bone and not bone produced in response to inflammation associated

with lameness. This is not to say that abnormal bone development does not occur at D; the distal phalanx shown in **Figure 5** and **Figure 6** clearly has abnormal bone development at that site, while D-values in claws with bony development at sites A, B or C were greater than those with no such development (1.34 times (95%CI 1.12–1.61) in lateral claws). Nevertheless, in the 136 claws without values >0 for any measurement other than D, we suspect that the increase in D with age is simply normal development. This is supported by our finding that the effect of age on D (1.09 times per increase of 1 year in age; 95% CI 1.05- 1.11) is very similar to the effect of age on BG Max (1.12 times; 95%CI 1.03 - 1.21), and that our model of the effect of age on the odds of A + B +C being >0 was compatible with no biologically meaningful effect (OR 1.14; 95 CI: 0.97-1.37) as was the effect of age on that sum if it was >0 (95 % CI:0.84-1.1). However this conclusion is far from definitive and further data are required to properly test this hypothesis.

## Conclusion

The present study demonstrates that, with minor modifications, digital photography can be used to quantify the measures of bone development assessed by Newsome *et al.* (2016). The advantages of cost mean that this method is much more suitable for measuring bone development in all systems including those in low-moderate income countries. In this study both age and lameness history were positively associated with bone development, with lateral claws having more bone development than medial ones. Our results were thus consistent with previous studies of age and lameness history and bone development in housed cows. However, this was small convenience sample of cattle with only six cows with no history of lameness, so these conclusions are preliminary, and more data are needed.

## References

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