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Guest Editorial

Greyhound sports injuries: Racing careers fractured by anatomical imperfections?

Greyhound sports injuries are typically of an indirect nature, and as such there are site-specific injuries which are well known within the greyhound world. Good examples are fractures of the accessory carpal bone, central tarsal bone and dorsal metacarpal stress fractures. Investigation of the injury mechanics is facilitated as the location of injuries can be predicated, and thus comparisons can be made between racing and non-racing dogs (and even comparisons within each individual animal over a racing career). Thus there are two arms to the investigations, namely, specific analysis of particular injuries, and the overall patterns of injuries during training and racing.

The paper by Dr. Gabriela Galateanu of the Leibniz Institute for Zoo and Wildlife Research and her colleagues, published in this issue of *The Veterinary Journal*, shows that the development of more specific dynamic anatomy can help to determine why certain fractures are more likely (Galateanu et al., 2013). The common failure of the right central tarsal bone has been linked with the loading experienced by the right hindlimb during anti-clockwise cornering, and the overall injury patterns also suggest that the right hindlimb is more heavily loaded than the left hindlimb, since 90% of all hindlimb injuries are right-sided. This includes the higher prevalence of sand-burns on the right hind foot (sand burns are skin abrasions on the plantar web skin).

There is also the issue of the asymmetric anatomy of the tarsus, meaning that the right central tarsal bone is compressed in a way that is different from the fourth tarsal bone of the left tarsus (these bones being on the ‘rail’ side of their respective limbs). Radiographic examination of the left and right tarsi also show the differences in terms of a higher bone density of the right central tarsal bone (Bergh et al., 2012; Hercock et al., 2011; Johnson et al., 2000). These pieces of observational evidence together help to explain the left–right loading asymmetry and the within-limb loading asymmetry. There are usually several factors to consider when attempting to explain the mechanics of an injury and for greyhound injuries these also include the basic fitness of the musculoskeletal system, track quality and collisions.

It is widely accepted that many sporting fractures are the end result of the coalescence of stress fractures (Bergh et al., 2012; Muir et al., 1999), and that proper exercise regimens to encourage the adaptation of bones to exercise are an essential part of injury prevention (Goodship and Smith, 2008). The various studies of bone density give us evidence of Wolff's Law[1] at work. There is a precise link between the mechanical forces in bone and the remodelling events that enable the bone to cope with these stresses in the future. At the molecular level within a bone, apatite crystals are bonded to collagen by a pair of copper ions. This creates a positive–negative (p–n) junction, or diode, within the bone crystals. Loading the bone generates an electric charge by a piezoelectric effect across the bone crystals, which is converted into a one-way current by the p–n junction. Compressive loading generates a net negative charge across the apatite–collagen junction, and it is this charge which attracts osteoblasts to lay down new bone at the areas of greatest stress. This process underlies the responses seen as Wolff's Law, and dictates how bone is remodelled.

Even with all this skeletal adaptation, we must not forget that neurological control of the body and proprioception are integral to this. The nervous system can and does adapt to exercise. The proper coordination of gait and the rapid reaction to changes in the racing environment (i.e. track surface, corners and interference) are the keys to maintaining balance and physiological loading of the musculoskeletal system.

In the wider field of injury data analysis, as with any epidemiological analysis, there must be data with which to generate some useful statistics. In the case of greyhound racing, one may use readily available race form and from that decide whether there are any patterns. A major issue is the amount of variance in the outcome measure. In the analyses performed for the Greyhound Board of Great Britain (GBGB), the outcome measure of individual race time would seem to be the least biased of all the race form data. Initial forays into measures of performance yielded mean race times as expected, but with a variance much larger than reality. The advent of multilevel modelling using computer software has allowed the analysis of complex datasets at different levels, thus splitting the variance into packets related to each level. One can thus determine differences between stadia, gender, distance, age and weight, to name but a few, within a single model, giving marked advantages over both ANOVA and general linear models. Recent analyses undertaken for the GBGB on the effect of spaying and dioestrus used multilevel modelling on large datasets to extract subtle changes in performance that previously were masked by excessive variance.

As far as injuries go, the available data are more sparse and difficult to collect, but my own injury data show that certain patterns are evident. One of the most elusive has been to link the syndrome of muscle cramping in greyhounds to the findings of the GBGB Sudden Death Survey. Post mortem examinations show that multiple small bleeds from the blood supply within the iliopsoas muscle lead to fatal blood loss (either retroperitoneal or into the peritoneal cavity). Some cases had previous episodes of bleeding which had either healed, or were in the process of healing when the fatal bleed occurred. Determining when the bleed first occurs is very difficult, owing to a lack of specific injury data, but personal observations suggest that these greyhounds experience mild tearing of the iliopsoas during the acceleration phase (i.e. the first

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[1] The principle that changes in the form and function of a bone are followed by changes in its internal structure. Mosby's Medical Dictionary, 8th edition, 2009, Elsevier.

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30 m) of a race, but do not display clinical signs until the race has finished (typically 30 s for about 500 m), at which point hypovolaemic hypotension and related hypoxia produce marked ataxia and rapid collapse.

It is interesting that the male gender proportion for cramping is of the same order as the gender proportion for acute abdominal haemorrhage (AAH; 72% ± 11% are male in both groups, $n = 280$). The normal male gender proportion for racing greyhounds is 55% (±1%, $n = 11{,}175$), meaning that males are 2.1 times more likely to cramp (or suffer AAH) than females. The other similarities are that both cramp and AAH are more common in the colder months, which lead to the suggestion that AAH has its roots in muscular cramping. The proposed mechanism is unequal muscle fibre contraction, leading to local sheering forces and muscle tearing. This is an example of analysis where the cause is not known, but comparisons with known patterns can help to elucidate an unknown cause.

It is generally accepted that the level of significance be pre-set at an alpha of 5%, and that the only assessment required is whether the *P* value is greater or lesser than alpha. There is a growing perception that this general value often does not fairly reflect the nature of the data and that always setting alpha at 5% affects the power of the statistical tests and biases their interpretation in favour of minimizing Type I errors, with little regard for power and effect size. The result is that for small genuine differences there is a greater chance of committing a Type II error (a false negative), together with a lower power for a given sample size. What the analysis should be aiming for is an equal (or weighted) chance of either error, and in so doing the power of the test in question is increased. Essentially, alpha needs to be set at an optimal value to minimize both Type I and Type II errors (Mudge et al., 2012). Thus where it is suspected that small genuine differences exist, a power analysis can be performed to ensure an adequate sample size to detect the predicted effect size and to calculate the optimal alpha.

The other advantage of choosing the correct sample size is that the confidence limits will aid in the interpretation of the results, since as sample size increases, the confidence limits become more precise and allow the detection of smaller differences between test groups. The follow-on from this is that the results from statistical analysis can be interpreted in a more 'real world' fashion, as opposed to the familiar method of striving for low *P* values (Mudge et al., 2012; Verrill and Durst, 2005). There should be statements about effect size, which is essentially the description of results in a meaningful way, such as gender ratio, weight difference, height difference, relative risk, and correlation coefficient (Nakagawa and Cuthill, 2007).

I used this approach in analysing the performance of racing greyhound bitches during dioestrus – there was an obvious pattern but setting alpha at 5% would have rejected this as chance, even given the fact that greyhound races are won and lost over 0.01 s for a 30 s race. Performing a power analysis using a desired effect size to calculate an appropriate sample size, together with setting a non-standard alpha to give a balance in the Type I and Type II error rates, allowed a better realization of the performance changes due to dioestrus. Additionally, the large sample size required also made the estimates more precise (Payne, 2013).

There are many examples of where statistical significance does not relate to real significance, most especially in cases where statistically non-significant differences are actually part of a real effect, thus risking a Type II error. For example, race times, or the effect of aspirin on reducing deaths due to strokes in humans, are cases where real effects are rejected as chance by fixing alpha at 5%. To combat this, there is a good rationale for setting alpha to minimize the combined probabilities of making Type I or Type II errors at a chosen a priori critical effect size, with the alpha being specific to that analysis. This can keep the overall error rates (the combination of Type I and Type II errors) lower than with the standard alpha of 5%. This requires a round of calculations using the a priori probabilities of H_0 and H_1 being true, the weights (costs) of Type I and Type II errors, the critical effect size, together with the available sample size and variance (Mudge et al., 2012). Although this creates another task for the analyst, its benefit is in a better description of the problem under investigation.

So, there is a need for an extension of the science behind specific injury into the realm of statistics and epidemiology so that the real patterns of injuries related to gender, race length, age, etc. can be linked to the background science. Alongside this, there should be a proper appraisal of power and effect size, with the aim of balancing Type I and Type II errors rather than focussing purely on avoiding Type I errors. These strategies will provide a better and more valid understanding of the patterns and mechanics of injury and, coupled with better decision-making based on unbiased statistical analysis, will lead to the development of evidence-based training and racing strategies.

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References

Bergh, M.S., Piras, A., Samii, V.F., Weisbrode, S.E., Johnson, K.A., 2012. Fractures in regions of adaptive modeling and remodeling of central tarsal bones in racing Greyhounds. American Journal of Veterinary Research 73, 375–380.

Galateanu, G., Apelt, D., Aizenberg, I., Saragusty, J., Hildebrandt, T.B., 2013. Canine tarsal architecture as revealed by high-resolution computed tomography. The Veterinary Journal 196, 374–380.

Goodship, A.E., Smith, R.K.W., 2008. Skeletal physiology: Responses to exercise and training. In: Equine Exercise Physiology: The Science of Exercise in the Athletic Horse, First Ed. Saunders Elsevier, Philadelphia, PA, USA, , pp. 81–105.

Hercock, C.A., Innes, J.F., McConnell, F., Guilliard, M.J., Ness, M.G., Hodson, D., Young, I.S., 2011. Observer variation in the evaluation and classification of severe central tarsal bone fractures in racing greyhounds. Veterinary and Comparative Orthopaedics and Traumatology 24, 215–222.

Johnson, K.A., Muir, P., Nicoll, R.G., Roush, J.K., 2000. Asymmetric adaptive modeling of central tarsal bones in racing greyhounds. Bone 27, 257–263.

Mudge, J.F., Baker, L.F., Edge, C.B., Houlahan, J.E., 2012. Setting an optimal α that minimizes errors in null hypothesis significance tests. PLoS ONE 7, e32734.

Muir, P., Johnson, K.A., Ruaux-Mason, C.P., 1999. In vivo matrix microdamage in a naturally occurring canine fatigue fracture. Bone 25, 571–576.

Nakagawa, S., Cuthill, I.C., 2007. Effect size, confidence interval and statistical significance: A practical guide for biologists. Biological reviews of the Cambridge Philosophical Society 82, 591–605.

Payne, R.M., 2013. The effect of the oestrus cycle on the racing performance of greyhounds. Greyhound Board of Great Britain Internal Research Papers, GBGB, London.

Verrill, S., Durst, M., 2005. The decline and fall of Type II error rates. American Statistical Association 59, 287–291.