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Copper toxicosis in Bedlington terriers

SIR, – Copper toxicosis (CT) in the Bedlington terrier breed is an autosomal recessive disorder in which excess copper accumulates in the liver with an ensuing hepatopathy. The liver biopsy test, initially performed in young adulthood, is based on analysis of liver copper content together with a histological assessment of copper localisation and hepatocellular injury. Although largely superseded by genetic tests, it still remains today the best evaluation of phenotype.

Insight into the genetic basis of the disease came in 1997, when US workers found a microsatellite marker C04107, with allele 2 closely linked to the disease-causing mutation (Yuzbasiyan-Gurkan and others 1997). A comparable study broadly supported the US findings (Holmes and others 1998). A caveat emerged by both groups limited the deriv-ative test to 90 to 95 per cent accuracy and recommended it to be used together with a supportive pedigree to establish marker linkage. Subsequently, we at the Department of Veterinary Pathology, Faculty of Veterinary Science, University of Utrecht reported the identification of a microsatellite marker C04107, in which a deletion in both the mutant form (van de Sluis and others 2001) and the non-mutated form (Yuzbasiyan-Gurkan and others 1997). Recently, however, some CT-affected Bedlington terriers have been identified without the homozygous COMMD1 deletion in both the UK, USA and Australia (Coronado and others 2003, Hyun and others 2004), reinforcing earlier voiced concerns. Since no other COMMD1 mutation could be found in them, (Haywood and van de Sluis) have hypothesised that a second as yet unknown disease gene is involved in Bedlington CT and have received funding from the Kennel Club to try to identify this gene.

While we have collected DNA from many of these dogs, we are aware that in the UK Bedlington population there exists dogs that exhibit signs of liver disease associated with excess copper storage, despite having been identified with DNA marker as categories 1-1 or 1-2.

It is of the utmost importance that these dogs are identified, particularly as animals exhibiting DNA marker 2-2 have been largely removed from the breeding pool, which has left a sense of complacency among breeders. In a relatively small breeding population, such as Bedlington terriers, the impact in time of an unsuspected carrier of an unrecognised second mutant copper gene could be devastating, and the undeniable progress made over the years in eliminating COMMD1 from the gene pool halted. Identification of the second gene could also help the canine population at large, as several other breeds, including West Highland white terriers, Skye terriers, dobermanns, dalmatians and keeshonds, have been identified with copper-associated liver disease, which preliminary screenings have shown not to be associated with COMMD1 deletion. It could be that a second gene is responsible.

The purpose of this letter is to inform colleagues in small animal practice as to the current situation with regard to Bedlington terrier CT, and to alert them to Bedlington terriers with signs of liver disease irrespective of their DNA marker status. I would like very much to be informed about these dogs. Also, I would like brought to my attention those Bedlington terriers that might be undergoing a routine blood test in which the parameters indicate a possible hepatopathy. I would be willing to offer a free liver biopsy test to the dogs of compliant owners.

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References


