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SHORT COMMUNICATIONS

Changes in Litter Size in Kerry Blue Terrier Dogs with Abnormal Dentition

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Abstract—The pleiotropic effects of mutations resulting in abnormal dentition were analyzed in Kerry Blue Terrier. A decrease in litter size was demonstrated for dogs with dentition anomalies. The mean litter size was 5.72 puppies when both parents had normal dentition and 3.64 puppies when the parents had hypodontia. Analysis showed that the decrease in litter size cannot be fully explained by the effect of inbreeding and is most probably associated with the pleiotropic effect of the genes controlling teeth development on the embryonic viability.

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Deviations from normal dentition occur in many mammals. The genetic control of these abnormalities has been best studied in mouse and human. In these species, the major role in dentition abnormalities is played by mutations of the *PAX9* and *MSX1* homeobox genes [1–6]. Homeobox genes control the key ontogenetic events in many systems and functions, and it is not surprising that their mutations has a broad range of pleiotropic effects [7, 8].

Maldevelopment of teeth is often observed in dogs of various breeds. In some breeds, a lack of several teeth (hypodontia) is not considered impairing the breeding value of the dog and, consequently, this abnormality is not eliminated by selection. Kerry Blue Terrier is one of such breeds. We have previously shown that agenesis of premolars in Kerry Blue Terrier dogs is determined by mutations of two different genes, which have been termed *LP1* and *LP2* [9–11].

In this work, we studied the pleiotropic effects of these mutations on the litter size in Kerry Blue Terrier.

We examined the same pedigree that was used to analyze hypodontia. Its structure has been described in detail previously [9–11]. We analyzed 98 matings of 111 dogs, including 59 males and 52 females. Of these, 77 dogs (45 males and 32 females) had complete dentition and 34 (14 males and 20 females) had various abnormalities of dentition, consisting in the lack of one to four premolars.

All matings were divided into three groups. Group 1 (N × N) included 46 matings where both parents had normal dentition. In group 2 (N × A), hypodontia was observed only in one parent (a female in 38 matings and a male in 5 matings). In group 3 (A × A), both parents lacked one or several premolars (nine matings).

The mean litter size in the three groups is characterized in the table. One-way analysis of variance showed that the intergroup difference is significant ($F = 9.33$, $p \ll 0.0001$, $d.f._1 = 2$, $d.f._2 = 95$). When only one parent had incomplete dentition, the litter size was 11% lower than in group N × N ($P = 0.033$). When both parents had dentition abnormalities, the litter size was 36% lower than in group N × N ($P = 0.004$) and 28% lower than in group N × A ($P = 0.016$). The difference between reciprocal matings in group N × A was nonsigificant ($P = 0.421$).

The abnormal phenotype was characteristic of a female in most of the N × A matings. Hence, the decrease in litter size in group N × A compared with group N × N is to a great extent due to a maternal effect. However, the decrease in litter size in group A × A compared with group N × A testifies that the paternal genotype is as important in determining the litter size as the maternal genotype.

The effect of the paternal genotype on the litter size has been observed in many studies. The most common explanation is that males affect both the number of ovu-

Mean litter size and coefficient of inbreeding between the parents in matings of dogs with normal (N) or abnormal (A) dentition

Mating type	Number of matings	Litter size	Coefficient of inbreeding
N × N	46	5.72 ± 0.221	0.039 ± 0.006
N × A	43	5.08 ± 0.223	0.069 ± 0.010
A × A	9	3.64 ± 0.737	0.070 ± 0.019

lating eggs in females and the pre- and postimplantation survival of embryos [12–14]. The first factor cannot be effective in the case of house dogs, whose ovulation occurs spontaneously rather than being male-induced [15]. It is probable that an increase in embryonic lethality plays a main role in this case. Embryonic lethality may result from the pleiotropic effect of mutations of the *LP1* and *LP2* genes or from inbreeding, which is observed in the pedigree under study. We have previously shown that a lack of premolars is inherited as a recessive trait. Consequently, inbreeding increases the likelihood of hypodontia. It is in inbred matings that the litter size should be decreased to the maximum extent. As shown in the table, the coefficient of inbreeding in group $N \times N$ was significantly lower than in the other groups. Thus, inbreeding did contribute to the decrease in litter size. However, the coefficient of inbreeding was much the same in groups $N \times A$ and $A \times A$, and the significant difference in litter size between these groups is not fully explained by the effect of inbreeding. These findings make it possible to assume that the pleiotropic effects of mutations of the *LP1* and *LP2* genes are the major cause of the decrease in litter size in dogs with dentition abnormalities.

A lack of particular teeth is not under negative selection in Kerry Blue Terrier, and dogs with incomplete dentition have the same probability of producing a litter as dogs with normal dentition. Our results demonstrate that dentition abnormalities can serve as a marker of genetic defects decreasing the litter size in dogs of this breed. This fact should be taken into account in developing breeding programs in Kerry Blue Terrier.

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