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Short communication

Screening of Polish Holstein-Friesian bulls towards eradication of Complex Vertebral Malformation (CVM) carriers

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Abstract

The effectiveness of a program aimed at eradicating carriers of the recessive disorder Complex Vertebral Malformation (CVM) from the population of Holstein-Friesian bulls is reported. Among 1823 bulls, 1268 young and 555 proven bulls were examined. Three hundred and three bulls appeared to be CVM carriers (16.62%). The highest number of carriers occurred in the sons of a CVM sire, 55.51% and 61.90%, for proven and young bulls, respectively. This very high incidence of CVM carriers forced us to implement a strategy of screening young bulls offered by individual breeders to insemination centers. In effect, the number of CVM carriers dramatically dropped among proven bulls born in 2004 and disappeared in bulls born in 2006.

Key words: complex vertebral malformation, Holstein-Friesian cattle, lethal defect

Introduction

Complex Vertebral Malformation syndrome (CVM) is a hereditary lethal disorder, determined by a recessive gene. It was discovered in a Danish population of Holstein cattle in 1999 (Agerholm et al. 2001) and confirmed in several countries (reviewed by Rusc and Kaminski 2007, Zhang et al. 2012). CVM causes embryo death, frequent abortions and stillbirth (Agerholm et al. 2001, Nielsen et al. 2003). Heterozygotes (carriers) do not show symptoms. In 2002 we started a program aimed at eradicating CVM carriers among Holstein-Friesian bulls used in the national breeding program in Poland. The aim of this paper is to present the effectiveness of culling the CVM carriers from the active population of bulls.

Materials and Methods

The present study includes 1823 Polish Holstein-Friesian Black-and-White bulls born between 1991 and 2012 which were the property of 4 domestic insemination companies, or individual breeders offering young bulls to these companies. Genomic DNA was isolated from 100 µl of commercial semen straw using a Genomic DNA Purification Kit (Promega) or from 0.4 ml of blood using a MasterPure DNA Purification Kit (Epicentre). CVM carriers were identified using the PCR-SSCP method described earlier (Rusc and Kaminski 2007).

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Table 1. CVM carrier frequency among Polish Holstein-Friesian bulls examined in 2001-2012 (with regard to sire genotype). International symbols were used: TV for tested animals free of CVM, and CV for carriers of CVM.

Group of bulls	Number of bulls	Carrier bulls	
		no.	%
Proven bulls	555	94	16.94
– TV sire	291	34	11.68
– CV sire	74	41	55.41
– non-tested sire	190	19	10.00
Unproven bulls	1268	209	16.48
– TV sire	860	141	16.40
– CV sire	21	13	61.90
– non-tested sire	408	55	13.48
All bulls	1823	303	16.62

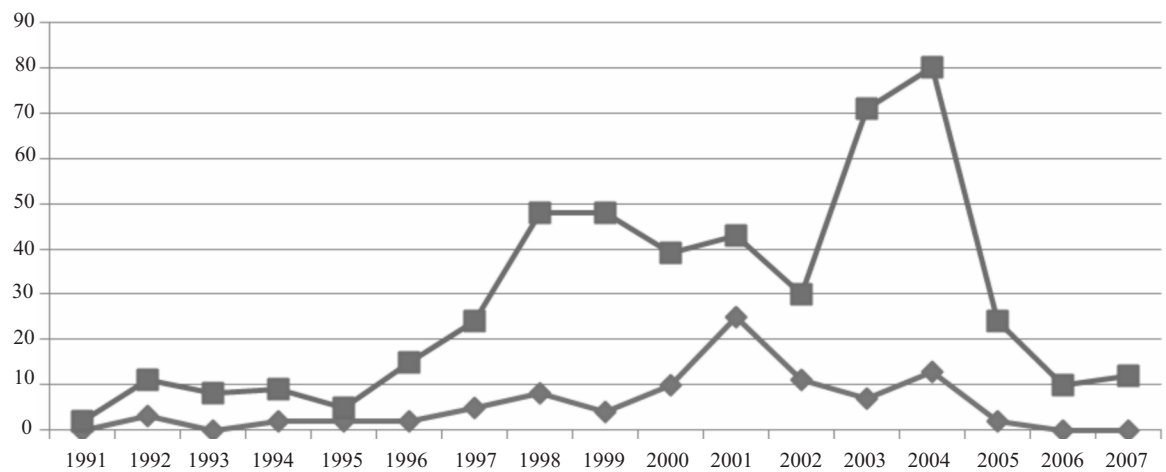

Fig. 1. Changes in the frequency of CVM carriers in proven bulls in relation to their birth year. Upper line (with square) indicates TV bulls, lower line (with diamonds) indicates CV bulls.

Results and Discussion

Among 1823 bulls, 1268 young and 555 proven bulls were examined (Table 1). Three hundred and three bulls appeared to be CVM carriers (16.62%). The highest number of carriers occurred in the sons of a CV sire, 55.51% and 61.90%, for proven and young bulls, respectively. The percentage of identified carriers after a TV sire in both groups of bulls was also relatively high (11.68% and 16.4%, proven and young, respectively) and indicated an alternative pathway of recessive allele transfer, i.e. through the bull's dam. Thomsen et al. (2006) indicate that in many countries the number of heterozygous bulls (CV) may reach the alarming level of 20-30%. Indeed, in our first report (Rusc and Kaminski 2007) the frequency of CVM carriers was very high (30.27% among young bulls). The very high incidence of CVM carriers forced us to implement a strategy to screen all young bulls offered by individual breeders to insemination centers and recommend eradication of carriers from progeny testing programs. In effect, the number of CVM carriers dramatically dropped among proven bulls born in 2004 and disappeared in bulls born 2 years later (Fig. 1). This does not mean that no CVM carrier will appear in the future. Because of the economical significance of the CVM mutation (Kearney et al. 2005) and its recessive mode of inheritance, attention has to be paid to any case of a bull having in its origin any known CVM carrier. In countries keeping detailed records of cases of congenital defects and reducing their occurrence, their frequency is generally low. VanRaden and Miller (2006) have shown that, in the USA, the frequency of CVM carriers among bulls

born after 2002 decreased to 1% after implementation of testing. This illustrates the urgent need to organize a system of permanent monitoring of inborn defects of calves, in particular in Polish Holstein-Friesian cattle, which is the second largest population (2.4 mln milking cows) in the world, and a breeding program mostly based on imported semen. Since Holstein-Friesian cattle is the most inbred dairy cattle breed, closer international integration of national associations of Holstein cattle breeders and coordination of genetic defects discovery programs is necessary to decrease the risk of new recessive disorders and their prevalence.

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