A METABOLISM-BASED SCENARIO FOR PCDD AND PCDF EXCRETION IN MILK FROM PRIMIPAROUS TRANSITION DAIRY COWS

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Introduction The EU has recently established provisional maximum and alert levels for PCDDs+PCFDs in cow milk at respectively 3.0 and 2.0 pgTE g–1 (WHO-TEFs), lipid base (lb), to prevent unacceptable high human exposures.1 These levels are basically referred to bulk milk resulting from milking dairy cows that differ each other with respect to the metabolic and physiological statuses (i.e., lactation period, age, number of labours). It may be envisaged that milk from primiparous cows be more contaminated than bulk milk due to the following concurrent risk factors: (a) PCDD+PCDF body burden, determined by progressive bioaccumulation over 26 months (on average, from birth to the age of first labour) from environmental and feedingstuff contributions (on average, 5500-kg dry matter intake); (b) the metabolic impairment between the energetic intake and the needs represented by body weight maintenance and the quantity of milk yield.2 Metabolic impairment is particularly evident in primiparous cows — affected by a severe appetite drop just after labour — that determines a status of “transition” cow, where up to 5 % of body weight can be lost mainly due to adipose tissue mobilization. Such status is clinically evaluated on a routine basis by the body condition score (BCS) index.3 Several studies were carried out to evaluate the transfer factors from contaminated feedingstuffs to dairy products.4,5 However, there is a sparseness of data concerning body burden contribution to contamination of the aforesaid products.2 In this paper a metabolism-based scenario for the evaluation of such contribution is proposed, to exploit possible animal management options to reduce the overall contamination of bulk farmed milk. Material and methods Nutritional and animal husbandry data have been derived by routine farming management practice referred to a Holstein-Fresian dairy farm, in Italy. Feedingstuff intake (in kg day–1 of dry matter (dm)) and the daily milk yield were derived from three primiparous cows observed during the winter of 2005. Daily nutritional needs were calculated according to theoretical values derived from good farming management practices and accounting for 7.80 Mcal for a 500-kg body weight and 0.73 Mcal for each kg of 3.8%-lipid milk produced. Effective energy intakes were calculated on the basis of the real feed (dm) amount ingested, assuming that 1-kg feed was equivalent to 1.15 Mcal. PCDD+PCDF body burdens were obtained from a EU inventory dealing with contamination levels in beef:6 0.5, 2.1, and 3.8 pgTE g–1 lb were considered to be representative of the mode, 95%, and 99% percentile values (Q.95 and Q.99, respectively). As a worst case assumption, we considered a body burden of 22.6 pgTE g–1 lb, recently reported for a female beef exposed to treated wood facilities.7 It was assumed that, in a 26-month-old pre-lactating cow, a steady state is reached between concentration in flesh and metabolic adipose tissues.2 The energy value derived from 1-g tallow metabolization was estimated 8.72 cal. The theoretical milk contamination of primiparous cows during their transition period was estimated by taking into account the amount (grams) of tallow to be catabolized to cope with the energy deficit and the reference contamination body burden levels selected. The absolute contamination derived from body burdens was then plotted against the total lipid amount (3.8 %) in milk on a daily yield, to recover the final amount of PCDDs+PCDFs (pgTE g–1 lb) daily released in milk. In this model we have assumed a quantitative end up of catabolized lipids in the mammary gland, to support milk production and the subsequent trasfert of the body burden-derived dioxins. Results and discussion The relationships between milk production, the real feed intake, and the metabolic needs are reported in Figure 1 during cow’s transition period. No data are reported for the first three days of lactation due to secretion of colostrum. Figure 1. Metabolic pattern model for primiparous Holstein-Fresian transition cows. In Figure 2, the milk (3.8% lipids) contamination is modelled according to the default body burdens values considered (0.5, 2.1, 3.8, and 10 pgTE g–1 lb). Lipid mobilization was assumed to be the main contributor; no concurrent contribution from feeds was considered. Figure 2. Contamination contributions from primiparous transition cows according to different body burdens and to the amount of tallow metabolized to cope with energy impairment. For modelling, it was assumed that the all the lipid catabolism end up in the mammary gland. If only a fraction of the lipids contributes to milk production, the PCDD+PCDD concentration in milk will be lowered by a proportional amount. The impact of primiparous transition cow contributions to PCDD+PCDF contamination of bulk milk has to be evaluated according to cow turnover in the farm. Primiparous rate in high producing farms is roughly 33 % and labours distributed the year round: in other words, of 100 animals present in the farm, on average 33 cows are yearly primiparous, with some three labours per month. In the light of the above, it was estimated that primiparous transition cows in their first lactation

month can contribute up to 3 % of total farm milk production (es., some 81 kg of milk of a daily total of 2666-kg milk), with the individual contributions described in Figure 2 . These critical contributions can therefore undergo dilution in bulk milk, and thus may not appear as a factor relevant to reach the alert and maximum levels. According to the proposed model and accounting for the above reported dilution factor, a rise of 0.3–1.0 pgTE g–1 lb in bulk milk may be expected from individual milk of transition primiparous cows under body burden worst-case conditions. The aforesaid contribution is even less relevant for those animals with mode, Q.95, and Q.99 body burdens (<0.2 pgTE g–1). On the contrary, in some dairy farms (basically “organic”), labours could be concentrated in the most favourable seasons, such as spring, to allow offspring to graze on grasslands of the best quality. Generally, under such situations cow turnover in farms is reduced to 20 %: In this case, we could expect a change of the dilution factor, with a seasonal critical contribution from primiparous transition cows of up to 10 % of the bulk milk. This could lead to a rise in contamination levels up to 3.3, 0.6, 0.3, and 0.1 pgTE g–1 lb, for worst case, Q.99, Q.95, and mode body burdens, respectively. To conclude, as risk management options the following critical control points were identified: (a) to prevent excessive PCDD+PCDF body burdens in non-lactating cows, all possible exposure risk factors (i.e., quality of the environment, treated wood-based facilities and beddings, feedingstuffs) must be taken into account; (b) obesity of non-lactating cows (fat cow syndrome) must be prevented and lipid mobilization during the transition period limited with adequate nutritional requirements coupled with a clinical monitoring (BCS index to be kept within 3.5 and 2.5); (c) because contributions from primiparous transition cows are also a direct consequence of the turnover rate in the farm, it is advisable to prolong the dairy cow economic life for more than three lactations; (d) in dairy farms where labours are seasonally scheduled, adequate sampling procedures should be taken to monitor bulk milk contaminantion levels while reducing possible seasonal biases; (e) when high body burdens are suspected, it could be advisable to avoid to pool in the bulk the milk from primiparous cows during the transition period. In the near future, and with respect to bulk contamination, our group will address further investigations on the analytical determinations of TEQ levels in milk from transition animals to verify the fitness of the proposed scenario. Acknowledgements: Work granted by Ministry of Health, Project ARACNA 2002-2005. Authors wish to thank Mrs Fabiola Ferri for the technical assistance. References 1. Council Regulation 2375/2001/EC of 29 November 2001 Official Journal of the European Communities. L321/1, 6 December 2001. 2. Sweetman A.J., Gareth O.T, and Jones K.C. (1999). Environmental Pollution 104, 261–270. 3. Dechow C.D., Rogers G.W., and Clay J.S. (2001). Journal of Dairy Science 84, 266–275. 4. Malisch R. (2000). Chemosphere 40, 1041–1053. 5. European Commission (2000). Opinion of the Scientific Committee on Animal Nutrition on the dioxin contamination of feedingstuffs and their contribution to the contamination of food of animal origin. Available at: http://europa.eu.int/comm/food/fs/sc/scan/out55_en.pdf. 6. European Commission (2000). Assessment of dietary intake of dioxins and related PCBs by the population of EU Member States. Available at: http://europa.eu.int/comm/dgs/health_ consumer/library/pub/pub08_en.pdf. 7. Huwe J.K., Davisony K., Feily V.J., Larseny G., Lorentzseny M., Zaylskiey R., and Tiernanz T.O. (2004). Food Additives and Contaminants 21, 182–194.