Effects of Feeding Fish Meal and n-3 Fatty Acids on Ovarian and Uterine Responses in Early Lactating Dairy Cows

A. R. Heravi Moussavi,* R. O. Gilbert,† T. R. Overton,‡ D. E. Bauman,‡ and W. R. Butler‡1
*Department of Animal Science, Ferdowsi University, Mashhad 91775-1163, Iran
†Department of Clinical Sciences, and
‡Department of Animal Science, Cornell University, Ithaca, NY 14853

ABSTRACT

The study was designed to test the effects of dietary supplementation with fish meal or specific n-3 fatty acids on ovarian activity and uterine responses in early lactating cows. From 5 to 50 d in milk (DIM), cows were fed diets that were isonitrogenous, isoenergetic, and isolipidic containing none (control), 1.25, 2.5, or 5% menhaden fish meal (FM) or 2.3% Ca salts of fish oil fatty acids (CaFOFA). Ovarian follicular dynamics were monitored along with plasma concentrations of estradiol and progesterone. Beginning at 23 DIM, cows were induced into a synchronized ovulatory cycle. On d 15 after ovulation (49 DIM), cows were injected with oxytocin and blood samples were collected to monitor uterine release of PGF2α (measured as 13, 14-dihydro-15-keto PGF2α; PGFM). Uterine endometrial biopsies were collected for fatty acid analysis and cyclooxygenase-2 (COX-2) protein measurement. Ovarian follicular activities as well as plasma estradiol and progesterone concentrations were similar across diets. Endometrial fatty acid composition of eicosapentaenoic acid (C20:5, n-3) and docosahexaenoic acid (C22:6, n-3) were increased as much as 3-fold by supplementation with fish meal and CaFOFA. Conjugated linoleic acid (C18:2 cis-9,trans-11) in the endometrium was also increased; conversely, arachidonic acid (C20:4, n-6) percentage was decreased by 5% FM. Plasma PGFM response to oxytocin injection was not different among diets and endometrial COX-2 protein abundance did not differ. Results from this experiment demonstrate that dietary supplementation with fish meal or n-3 fatty acids in early lactating dairy cows significantly increased uterine n-3 fatty acid concentrations, but had no apparent effect on endometrial COX-2 or PGF2α, production in response to oxytocin challenge.

Key words: cow, fish meal, cyclooxygenase-2 protein, n-3 fatty acids

INTRODUCTION

Prostaglandins influence many processes throughout the body including ovarian activity and follicle development and are well known for their effects on corpus luteum (CL) function (Robinson et al., 2002). Arachidonic acid (ARA), an essential fatty acid present in membrane phospholipids, is the primary precursor of prostaglandins. Cyclooxygenase-2 (COX-2), a rate-limiting enzyme, oxidizes ARA to prostaglandin H2 (PGH2), which is the precursor of all other prostaglandins including PGF2α and PGE2. Bovine endometrium secretes PGF2α during the estrous cycle (Danet-Desnoyers et al., 1995) and pulsatile secretion seems to mediate CL regression (McCracken et al., 1999). Expression of COX-2 mRNA and protein were found to be at low and high levels on d 1 to 12 and 13 to 21 of the estrous cycle, respectively (Arosh et al., 2002). Polysaturated fatty acids such as linoleic, linolenic, eicosapentaenoic (EPA), and docosahexaenoic (DHA) may inhibit uterine PGF2α synthesis through mechanisms such as decreased availability of the precursor ARA, increased competition by these fatty acids with ARA for binding to PGH synthase, and inhibition of PGH synthase synthesis or activity (Mattos et al., 2000). In vitro studies showed that the n-3 fatty acids EPA and DHA altered prostaglandin biosynthesis in a number of cells and tissues (Weber and Sellmayer, 1991; Mattos et al., 2003). The most potent COX-2 inhibitor was EPA followed by DHA (Ringbom et al., 2001). In another study, isomers of conjugated linoleic acid (CLA) also inhibited PGF2α synthesis and the effect was independent of the concentration of linolenic acid and n-6:n-3 ratio (Harris et al., 2001).

Fish meal (FM) has relatively large concentrations of 2 polyunsaturated fatty acids, EPA (C20:5, n-3) and DHA (C22:6, n-3) and so FM or fish oil fatty acids in the diet may alter uterine PGF2α synthesis. Inhibiting uterine secretion of PGF2α by feeding EPA and DHA may delay regression of the CL and increase fertility by improving embryo survival (Burke et al., 1997; Staples et al., 1998; Mattos et al., 2000).

Evidence for the incorporation of DHA (Mattos et al., 2004) and EPA (Burns et al., 2003; Mattos et al., 2004)