Altered steroid hormone and prostaglandin metabolism during chlamydial infection in sheep

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Chlamydial infection is associated with premature labour in sheep, and causes necrosis of placential tissue. The effect of Chlamydia infection on the synthesis of progesterone was studied, by measuring serum progesterone in seven infected and seven control animals. Progesterone inhibits the release of prostaglandin (PG) E2 and PGF2α, from the pregnant uterus (Taylor et al., 1978). Local inflammation at the site of infection may also stimulate intrauterine PGE2 synthesis (Thorburn & Challis, 1979).

The amniotic and allantoic sacs and the utero-ovarian vein were cannulated in seven Chlamydia-infected sheep and seven uninfected controls. Infection was by subcutaneous inoculation of 103 infectious particles of an ovine abortion strain of Chlamydia psittaci. PGE2 was detected by radioimmunoassay of uterine fluids (Leaver & Seawright, 1982) without prior extraction, as parallel binding curves were obtained in the presence and absence of fluid. However, PGE2 in utero-ovarian plasma was immediately extracted, as an interfering factor, and PGE2 degradation, were detected in plasma. Anti-PGE2 antiserum was purchased from the Institut Pasteur, Paris, and anti-rabbit IgG was donated by the Scottish Antibody Production Unit.

The plasma progesterone of Chlamydia-infected sheep was not significantly different from control values until day 120 of gestation (see Fig. 1). The concentration of progesterone decreased significantly between day 119 and day 135 of gestation in Chlamydia-infected animals, but not in control, uninfected sheep. The decline in plasma progesterone in control sheep occurred between day 139 and 145 of pregnancy. Therefore, the decline in circulating progesterone was observed 20 days earlier in Chlamydia-infected sheep, and 16 days before delivery.

The concentration of PGE2 in amniotic and allantoic fluids in control animals was low (2-4 ng/ml), between day 115 and day 120 of gestation. In contrast, an elevated concentration of PGE2, and pulsatile release of this prostaglandin, was observed in Chlamydia-infected sheep from day 119, which gradually increased until delivery. The release of PGE2 into the utero-ovarian vein just before parturition, observed in control animals, was impaired or inhibited in Chlamydia-infected sheep. The magnitude of the increase in PGE2 observed in Chlamydia-infected amniotic fluid (over 2 ng/ml), suggested that the PGE2 was of uterine, rather than leucocyte, origin. The relation of the timing of PGE2 release, to the decline in plasma progesterone, also suggested an endocrine control of this PGE2 release.

In summary, changes in circulating steroid hormones, and intrauterine PGE2, were detected in Chlamydia-infected sheep. These changes may precipitate the premature labour associated with Chlamydia infection.

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Fig. 1. Plasma progesterone in Chlamydia-infected (a) and control (b) sheep during late pregnancy

The mean date of lambing is indicated with an arrow. Progesterone was extracted from plasma using diethylether, and results were corrected for efficiency of solvent extraction (70 ± 6%), and detected using the antiserum of Scaramuzzi et al. (1974).