Journal of Andrology, Vol 16, Issue 1 5-11, Copyright © 1995 by The American Society of Andrology

JOURNAL ARTICLE

Prenatal in vivo bulbourethral gland development is not affected by prostaglandin E2 inhibition

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Investigation of bulbourethral gland (BUG) development is useful to study genitourinary (GU) tract growth and differentiation. Understanding GU tract growth and differentiation is relevant to testing the hypothesis that the initial lesion of human benign prostatic hyperplasia involves focal re-expression of inductive processes in the periurethral region of the prostatic transitional zone. Prostaglandins play a role in regulating growth and morphogenesis of different organ systems. Previous reports have proposed that prostaglandin E2 (PgE2) mediates the masculinizing effects of testosterone in the developing neonatal male GU tract. We have previously shown that androgens lower rather than raise BUG PgE2 levels. Further studies led us to conclude that PgE2 does not play a major role in postnatal BUG growth and morphogenesis in vitro. In order to investigate the possible role of PgE2 in prenatal BUG development, indomethacin (INDO, 1.0 mg/kg- day, subcutaneously) was administered to pregnant BALB/c mice on gestational days 12-18. Control pregnant mice were either untreated or injected with dimethylsulfoxide vehicle. Anogenital distances were measured within 12 hours after birth in male and female offspring on day 19. In male neonatal mice, BUGs were examined histologically and PgE2 levels were measured by radioimmunoassay in BUGs and whole genital tracts. We observed no significant morphological differences in INDO-exposed BUGs compared to controls. No significant differences in mean anogenital distances of INDO-exposed male offspring or controls were detected. Mean anogenital distances of female offspring were similar in the three respective groups. Mean BUG PgE2 levels in INDO-exposed neonates were significantly lower ($P < 0.05$) than in untreated neonates.(ABSTRACT TRUNCATED AT 250 WORDS)

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