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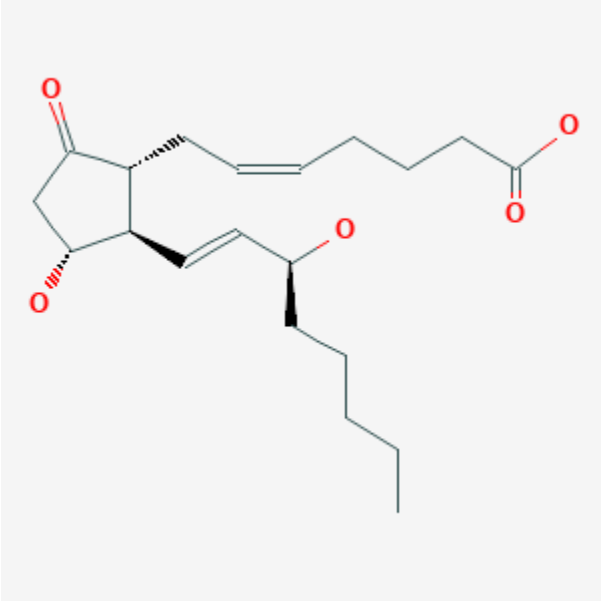
NLM Citation: Drugs and Lactation Database (LactMed) [Internet]. Bethesda (MD): National Library of Medicine (US); 2006-. Dinoprostone. [Updated 2018 Dec 3].
Bookshelf URL: https://www.ncbi.nlm.nih.gov/books/

Dinoprostone

Revised: December 3, 2018.

CASRN: 363-24-6



Drug Levels and Effects

Summary of Use during Lactation

Dinoprostone (prostaglandin E2) has not been measured in human milk after exogenous administration, but it is a normal component of breastmilk in small amounts where it may help protect the infant's gastrointestinal tract.

Use of vaginal dinoprostone to induce labor appears to have a negative effect on breastfeeding. Given orally in the first few days postpartum, dinoprostone can suppress lactation. Whether postpartum vaginal or endocervical administration suppresses lactation is not known, but it should probably not be used postpartum in mothers who wish to breastfeed. By one month postpartum, the drug appears not to suppress lactation.

Drug Levels

Maternal Levels. Milk levels of dinoprostone have not been measured after exogenous administration to humans. However, it is a normal component of breastmilk, where it may play a role in protecting the infant's gastrointestinal tract.[1][2][3][4][5][6][7] Normal concentrations in milk vary widely over a range up to about 500 ng/L, but appear to be similar to the maternal plasma concentrations.[5]

Vaginal or endocervical administration of dinoprostone for induction of labor produces maternal serum concentrations about double the normal levels,[8][9] so milk concentrations are likely to be comparably higher following exogenous administration.

Infant Levels. Relevant published information was not found as of the revision date.

Effects in Breastfed Infants

Relevant published information was not found as of the revision date.

Effects on Lactation and Breastmilk

A retrospective cohort study of birth records in Cardiff, Wales, UK found that the use of vaginal prostaglandins for the induction of labor resulted in an 11% decrease in the likelihood that mothers would be breastfeeding at 48 hours postpartum. The subgroup of first-time mothers had a 15% decrease.[10]

A nonrandomized prospective study compared women who had spontaneous deliveries with those who had elective induction using dinoprostone vaginal gel. At hospital discharge, exclusive breastfeeding rates were similar between the two groups (88% and 89%). However, at 1 and 3 months postpartum, exclusive breastfeeding rates were significantly lower in mothers who had dinoprostone induction than in those who delivered spontaneously. Exclusive breastfeeding rates were 54% and 85% at 1 month and 46% and 59% at 3 months postpartum, respectively. Rates of supplemental and exclusive formula feeding were higher in the induced mothers at both time points also.[11]

Dinoprostone has been used investigationally to inhibit postpartum lactation and engorgement by reducing serum prolactin concentrations.[12][13][14][15][16] The effect on prolactin levels, engorgement and lactation appears to be dose and duration related. Oral dosages of 3 mg daily for 4 days[17] or 0.5 mg three times daily were ineffective,[16] whereas oral dosages of 8 to 12 mg over 24 to 30 hours were effective.[12][14] These effects seem to be limited to the first few days postpartum; dinoprostone had no effect on serum prolactin or milk production when given to women 30 days postpartum.[12] Compared to oral bromocriptine 2.5 mg every 12 hours for 14 days, dinoprostone 12 mg orally in divided doses over 30 hours was as effective as bromocriptine, but resulted in less rebound breast tenderness.[14]

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Substance Identification

Substance Name

Dinoprostone

CAS Registry Number

363-24-6

Drug Class

Breast Feeding

Lactation

Oxytocics

Prostaglandins