# Effects of dexamethasone on antigen expressions and proliferation of amoeboid microglial cells in fetal rat brain

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## 摘要

## Abstract

The present study examined the effect of maternal administration of dexamethasone (DEX) on amoeboid microglial cells (AMC) in fetal rats extending from 16 to 20 days postconception (E16 to E20). After an intraperitoneal injection of DEX into pregnant rats at E10, the external morphology and distribution of immunolabelled AMC as detected with OX-42 and ED1 monoclonal antibodies remained unaltered when compared with those of the controls. The major effect of dexamethasone was on microglial cell population. Thus, with OX-42 and ED1, the numbers of immunolabelled AMC in the intermediate zone lateral to the striatum (IZS) of DEX-treated fetuses which remained relatively unchanged at E16 were significantly reduced at E18. However, OX-42 labelled cells showed an unexpected increase in number at E20 following DEX treatment. Microglial response to DEX was also analyzed in sections stained with the isolectin, GSA I-B4, which specifically binds alpha-D-galactosyl glycoproteins on microglia. The number of GSA I-B4 labelled AMC was significantly increased at E16, declined at E18 and remained constant thereafter in DEX-treated rats when compared with that of the controls. A major finding after DEX treatment was the wider occurrence of AMC double labelled with anti-BrdU antibody and GSA I-B4 or OX-42 at E16 compared with those in the controls suggesting that the initial increase of GSA I-B4 labelled AMC may be attributed to their proliferation. The drastic reduction of OX-42 and ED1 positive microglial cells notably at E18 may be due to the downregulation of surface antigens as a result of possible suppressive action of dexamethasone. On the basis of present findings, it is concluded that the antigenic expressions of fetal AMC may be modulated by DEX administrated maternally. Such however appeared to be extremely selective as reflected by the varied expression for certain immune molecules at different stages of brain development. This information

would be useful in potential use of glucocorticoids in prenatal therapy of brain pathology via maternal circulation