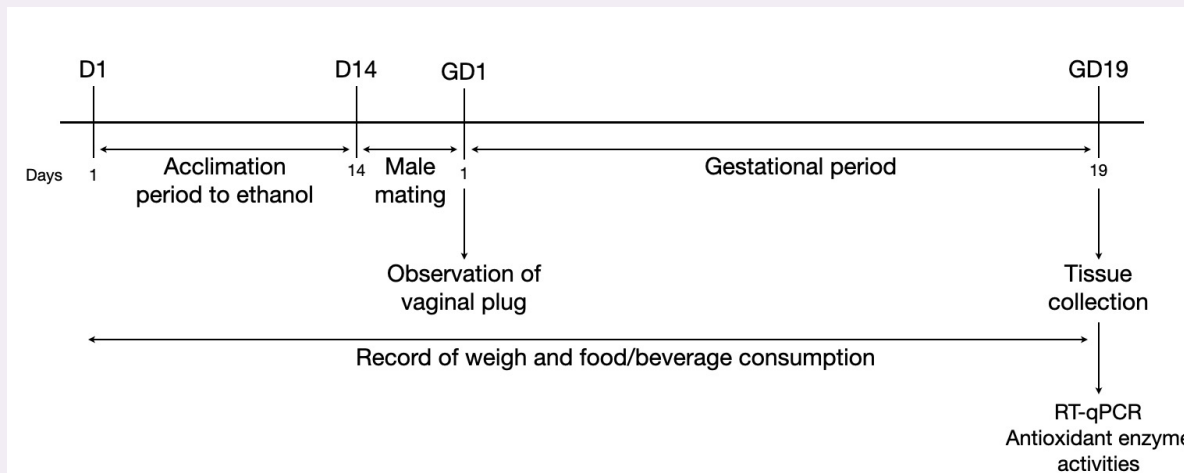


IGF-1 deficiency and ethanol consumption during gestation produce changes in CYP2E1 expression and antioxidant enzymatic system in maternal liver and placentas.

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Background: Ethanol use during pregnancy is a risk factor to develop adverse outcomes, e.g. fetal growth restriction (FGR), a condition of IGF-1 deficiency. Ethanol metabolism by CYP2E1 produces ROS that promote apoptosis and cellular injury. The placenta can be altered due to ethanol consumption. Its use during gestation impairs insulin and IGF-1 signaling pathways, altering cell proliferation, differentiation and placentation; however, to date no studies have been conducted to determine the teratogenic effects due to a synergic result of ethanol and IGF-1 deficiency in mice placentas. The aim of the present study is to determine the status of CYP2E1 and antioxidant system in placentas from IGF-1 partial deficient mice chronically exposed to ethanol during gestation.

Methodology:



Results: Pregnant HZ dams were smaller than controls, having less body weight gain through gestation due to ethanol consumption. Cyp2e1 gene was overexpressed in the liver of HZ pregnant dams exposed to ethanol (Fig. 1A); however, at protein level, CYP2E1 expression was slightly higher in WT-Ethanol. The antioxidant enzymatic system (catalase, glutathione peroxidase, superoxide dismutase) was altered by ethanol consumption in both maternal liver and placenta (Fig. 1B, 1C). Curiously, IGF-1 deficiency reduced the expression of CYP2E1 in the junctional zone of placentas from KO fetuses.

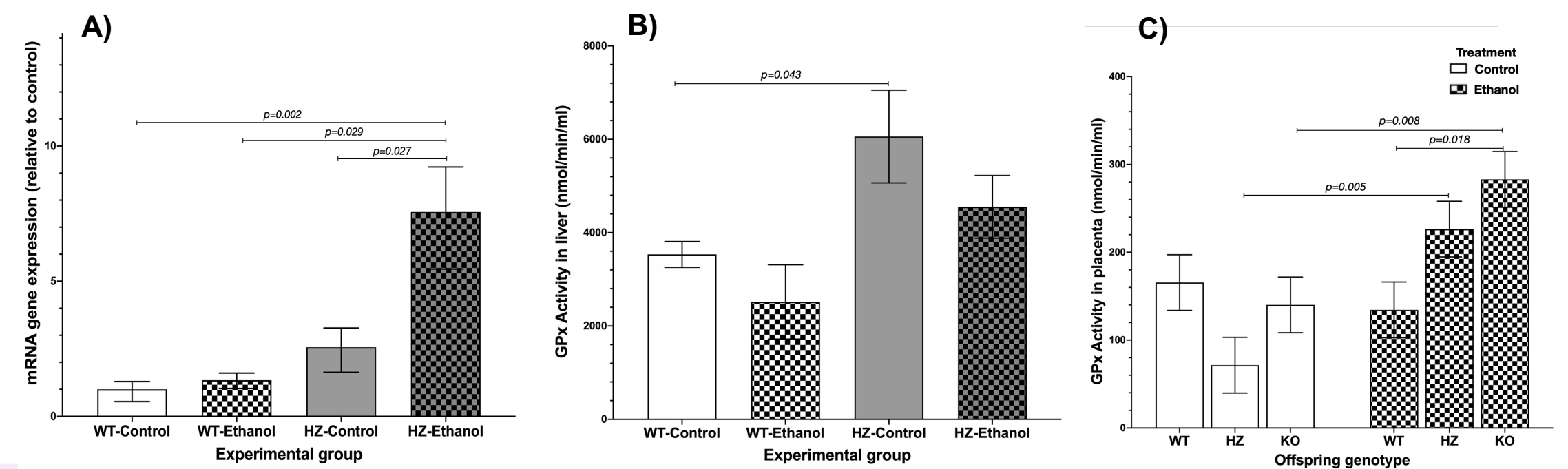


Figure 1. Effect of ethanol consumption during gestation in **A)** Cyp2e1 hepatic gene expression by RT-qPCR; and **(B)** glutathione peroxidase (GPx) activities in maternal liver and **(C)** placentas.

Conclusion: IGF-1 deficiency and ethanol consumption during gestation impair maternal and fetal parameters, including CYP2E1 expression and the antioxidant system, highlighting the crucial role of IGF-1 in intrauterine development.

References
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