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**Title Page**

<AT>An intergenerational effect of neuroendocrine metabolic programming alteration induced by prenatal ethanol exposure in rats

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<ABS-HEAD>Highlights ► Prenatal ethanol exposure (PEE) attenuated the physical growth in F2 offspring. ► PEE induced a low basal activity and hyper-responsiveness of HPA axis in F2 offspring. ► PEE induced glucocorticoid-associated glucose and lipid metabolic phenotypes in F2. ► The neuroendocrine metabolic programming alteration induced by PEE was inheritable

**<ABS-HEAD>Abstract**

<ABS-P>Prenatal ethanol exposure (PEE) induces hypothalamic-pituitary-adrenal (HPA) axis-related neuroendocrine metabolic programming alteration in the first generation (F1) rats. In this study, the HPA hormones and glucose/lipid phenotypes under basal state and stressed condition induced by a fortnight ice-water swimming were examined in F2 to verify the intergenerational effect. Under the basal state, serum corticosterone (CORT) and glucose of some PEE groups were lowered while those of serum triglycerides (TG) were increased comparing with controls. Following chronic stress, the percentage increase in CORT from the basal state tended to be greater for some PEE groups compared with controls while the percentage reduction of glucose and percentage elevation of TG were smaller. These results revealed that the low basal activity and hyper-responsiveness of the HPA axis as well as glucocorticoid-associated glucose and lipid phenotypic alterations were partially retained in F2, which indicates PEE-induced neuroendocrine metabolic programming alteration may have an intergenerational effect.

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