transcription factor induced by hypoxic condition, which regulate expression of specific target genes including angiogenic factors, erythropoietin, glucose transporters, and glycolytic enzymes. Recently, many studies connected to intracellular calcium levels and regulation of HIF-1α protein level. Calbindin-D_{9k} is a cytosolic calcium-binding protein and compensates with other calcium transporter protein for maintenance of cellular calcium level. It is expressed in primarily duodenum for absorption of calcium and in kidney for resorption of calcium. The objective of this study is to investigate interaction between HIF-1α and calbindin-D_{9k}

Materials and Methods: 8-10 weeks old C57BL/6mice and calbindin-D_{9K} knockout mice were exposed to hypoxia for 3weeks in closed polycarbonate chamber with nitrogen supply to remove the oxygenvs the normoxic groups. Oxygen concentration were measured and maintained thoroughly about 12±2% O2. ACHNcells were transfected with siRNA targeting calbindin-D_{9k}. ACHN cells and transfected ACHN cells were cultured in normobaric hypoxia (1% O2), with match control in normoxic conditions. Expression of HIF-1α were analyzed by Western blotting and real-time PCR. Also, expression of glucose transporter1 (GLUT1), down regulated gene of HIF-1α, was measured by real-time PCR.

Results: Protein level of HIF-1α was increased in calbindin-D_{9K} knockout mice compared with that of control in normoxic condition. However, increased protein level of HIF-1α of calbindin-D_{9k} knockout mice was reduced in hypoxic condition. mRNA levels of GLUT1 showed similar pattern with HIF-1α expression. In comparison to in vivo experiment, expression of HIF-1α and GLUT1 in mRNA and protein levels corresponded to that of calbindin-D_{9k} knockout mice.

Conclusions: These results suggest that calbindin-D_{9k} can regulate expression of HIF-1α in protein level.

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Effects of sex steroid hormone on expression of the Calbindin-D_{9k} in rat brainstem

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Introduction: Calbindin-D_{9k}(CaBP-9k) is a 9kDa polypeptide that is expressed in the mammalian intestine, uterus, and pituitary gland. The protein increase Ca^{2+} absorption by buffering Ca^{2+} in the cytoplasm. The factors that the role and regulation of the expression of the estrogen receptor and CaBP-9k in the brainstem are currently unknown. In this study, we investigate the expression of CaBP-9k in brainstem and whether the CaBP-9k was regulated by steroid hormones which known as the regulator of CaBP-9k in other tissues.

Materials and Methods: To investigate the effect of steroid hormone to the brainstem CaBP-9k expression, we performed gonadectomy. mRNA and protein expression were analyzed by real-time qPCR, western blot and immunohistochemistry.

Results: The uterus and thymus weight after treatment with surgery was regulated, suggesting that the rats responded properly to hormone. By eliminating the steroid producing organ, male and female CaBP-9k expression in brainstem were decrease and recovered by administrating steroid hormone. Immunohistochemistry shows that CaBP-9k is localized in the spinal trigeminal nucleus and Purkinje neurons. Western blot and immunofluorescence show that CaBP-9k protein expression was decreased by orchiectomy group in male.

Conclusions: Our findings provide the foundation for in-depth studies to improve the understanding of the effects of obesity on the trigeminal system and may have implications for the pathophysiology of headache disorders.

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Expressions of cation transporter channel are altered by OP and BPA in the mouse placenta

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Introduction: Cation (copper, iron, calcium), oxygen, carbon dioxide, and glucose are essential factor to fetal growth. These molecules are transferred by specific receptors located on cell membrane or cytoplasm in placenta. During pregnancy, expression of these channels is controlled by maternal and fetal nutrition state. These substances disturb action of reproduction-related hormones (ex>estrogen, progesterone) by interacting with their receptors, or affecting the expression of transporting genes for cations. To examine the effects of EDCs exposure during pregnancy, we conducted the in vivo model study using Pregnancy mouse.

Materials and Methods: Female ICR mice 8 weeks old were mated with adult males ICR mice 10 weeks old overnight and then examined for the presence of vaginal plug at the following morning. The day vaginal plugs were observed as GD (gestational date) 0.5. We used octyl-phenol (OP; 50 mg/kg/day), and bisphenol A (BPA; 50 mg/kg/day) in pregnancy mice for GD 11.5~16.5. Ethinyl estradiol (EE; 0.2 mg/kg/day), which activates estrogen receptors, was used as a positive control. Transcriptional level of calcium (PMCA1, TRPV6), copper (CTR1, ATP7A), and iron (IREG1, HEPH) transporting genes was quantified by qRT-PCR.

Results: TRPV6 mRNA expression were significantly decreased by EE and OP. mRNA levels of PMCA1 were significantly decreased by EE, OP, and BPA. CTR1 mRNA expression were significantly reduced by EE, OP and BPA. ATP7A mRNA expression were significantly decreased by EE, OP, and BPA. The expression of IREG1 mRNA was significantly decreased