ETHANOL-INDUCED AQP4 EXPRESSION IS INVOLVED IN SODIUM ION CHANNEL IN ASTROCYTE

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It is well known that acute EtOH consumption results in poor prognosis after traumatic brain injury (TBI). Survival rate was dropped about 50% by brain edema augmentation after TBI under EtOH consumption in rat. Water channel aquaporin-4 (AQP4) is related to brain edema. We previously reported that AQP4 is important role in brain swelling after TBI under EtOH. And TBI under EtOH affects sodium ion concentration. AQP4 is known to be regulated by Na-K ATPase, Na(+)-K(+)-2Cl(-) co-transporter. EtOH is involved in those channels. These findings indicate that sodium ion channel may affect AQP4 expression under EtOH. In this study, rat primary astrocyte was exposed to iso-, hypo- or hyper-sodium MEM up to 24 hour. EtOH (-100 mM) was added to each medium. Second, astrocyte was exposed in EtOH with tetrodotoxin (TTX, 400, 800 nM). Astrocyte AQP4 protein expression was checked by western blotting and its mRNA by real-time RT-PCR. Hypo-sodium condition increased AQP4 expression. Hypo-sodium and EtOH didn’t affect AQP4 expression. AQP4 expression was decreased by hyper-sodium and EtOH for short time exposure, and increased for long time. TTX had the effect on AQP4 expression under EtOH. From these findings, AQP4 expression is involved in sodium ion channel under EtOH.