The Role of Peripheral glycine receptors in cannabinoid-induced feeding

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Abstract

Background & Aim: Peripheral cannabinoid and glycine receptors are involved in food intake regulation. This study was conducted to investigate the possible interaction between these two receptors in regulating food intake.

Methods: This is an experimental study which was conducted on forty male Wistar rats. In the first phase of the experiment, the rats simultaneously received intraperitoneal injections of arachidonyl-2'-chloroethylamid (ACEA), CB1 cannabinoid receptor agonist (1 mg/kg) and glycine (100 mg/kg). In the second phase, the effect of intraperitoneal pretreatment with strychnine hydrochloride (post synaptic glycine receptor antagonist) with a dose of 0.01 mg/kg on ACEA, induced feeding was investigated. In the third phase, the impact of intraperitoneal pretreatment with AM281 with a dose of 0.5 mg/kg (CB1 receptor antagonist), glycine induced feeding behaviors was surveyed. Finally, in the fourth phase, the interaction effect of AM281 and strychnine hydrochloride was evaluated.

Results: In the first step, intraperitoneal injection of glycine significantly increased food intake. Also, following the administration, food intake significantly increased ($p<0.05$). Co-injection of glycine and ACEA had no significant effect on food intake compared to the sole injection of each agent ($p>0.05$). In the second step, intraperitoneal pretreatment with strychnine hydrochloride had no significant effect on ACEA-induced hyperphagia. Furthermore, intraperitoneal pretreatment with AM281 did not affect glycine induced feeding. In the fourth phase, co-administration of AM281 and strychnine hydrochloride significantly decreased cumulative food intake compared to the sole administration of each agent ($p<0.05$).

Conclusion: The interaction between peripheral CB1 cannabinoid receptors and glycine receptors has not been investigated so far. In the present study, despite some synergistic effects, it seems that glycine and cannabinoid receptors can cause hyperphagia through independent neuronal pathways.

Key words: CB1 cannabinoid receptors, rats, glycine receptors

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