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شماره پروژه ترجمه

نمونه ترجمه

ترجمه مقاله ☒

ترجمه کتاب ☐

نقش کانابینوئید علامت‌ده در ضدِدرد القا شده با ارکسین و گرلین مغزی در موش‌های بهوش

چکیده

ما این فرضیه را مطرح کردیم که سیستم کانابینوئید (CB) ممکن است در درمان انسدادی احشایی ناشی از اورکسین یا گرلین نتیجه دهد. تزریق داخل صفاقی آگونیست‌های CB1 / 2، WIN 55212 یا اتانول آمین O-Arachidonoyl (آراکیدونوئیل اتانول‌آمین) باعث افزایش حجم آستانه رفلکس برداشت شکمی ناشی از انسداد روده بزرگ در موش‌های صحرایی می‌شود، که نشان می دهد CB (سیستم کانابینوئید) می‌تواند ضد دردی احشایی را القا کند. پیشگیری با آنتاگونیست CB1 یا CB2 به طور بالقوه عمل ضد دردی ناشی از orexin-A ناشی از انسداد روده بزرگ را مسدود کرد در حالی که آنتاگونیست CB2 اما نه CB1 ضد دردی احشایی ناشی از گرلین را مسدود می کند. این نتایج نشان می‌دهد که سیگنالینگ کانابینوئید ممکن است در عمل ضد دردی ناشی از اورکسین یا گرلین به روش مکانیسمی متفاتی دخالت داشته باشد.

متن اصلی (انگلیسی) در صفحه بعدی آمده است ...

Short Communication

Role of the cannabinoid signaling in the brain orexin- and ghrelin-induced visceral antinociception in conscious rats

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ABSTRACT

We hypothesized that the cannabinoid (CB) system may mediate the brain orexin- or ghrelin-induced visceral antinociception. Intraperitoneal injection of either $CB_{1/2}$ agonist, WIN 55212 or O-Arachidonoyl ethanolamine increased the threshold volume of colonic distension-induced abdominal withdrawal reflex in rats, suggesting CB could induce visceral antinociception. Pretreatment with either the CB_1 or CB_2 antagonist potently blocked the centrally injected orexin-A-induced antinociceptive action against colonic distension while CB_2 but not CB_1 antagonist blocked the brain ghrelin-induced visceral antinociception. These results suggest that the cannabinoid signaling may be involved in the central orexin- or ghrelin-induced antinociceptive action in a different mechanistic manner.

Visceral pain sensation is an important physiological function in the gastrointestinal tract. For example, visceral hypersensitivity reflected by enhanced perception of physiological signals from the gut is commonly considered to play a major role in the pathophysiology of functional gastrointestinal disorders such as irritable bowel syndrome (IBS).[1] However, the mechanisms of regulation of visceral sensation in the brain have not been fully understood. We have recently demonstrated that orexin or ghrelin acts in the brain to induce visceral hyposensitivity,[2,3] suggesting that neuropeptides may play a role in the pathophysiology of IBS.

Biological effects of cannabinoids (CB) are mediated primarily through specific cannabinoid receptors (CB_1 and CB_2).[4] Among the roles of the cannabinoid system, the CB system may be implicated in the visceral antinociception through CB_1 and CB_2 receptors.[5]

Since either orexin or ghrelin acts centrally to induce a visceral antinociception[2,3] and cannabinoid could induce visceral hyposensitivity,[5] we made a hypothesis that the CB signaling may mediate the orexin- or ghrelin-induced visceral antinociception. In the present study, we tried to clarify the above speculation.

Visceral sensation was assessed by abdominal withdrawal reflex (AWR) by colonic distention using electromyogram (EMG) in conscious rats, which was validated as quantitative measure of visceral nociception as described previously.[2,3]

Male Sprague–Dawley rats (Charles River Laboratory, Atsugi, Japan) weighing about 200 g were housed under controlled light/dark conditions. Rats were allowed free access to standard rat chow and tap water. All of the experiments were performed in 24 h-fasted rats. Approval was obtained from the Research and Development and Animal Care committees at Asahikawa Medical University for all studies.

The specific $CB_{1/2}$ agonists, WIN 55,212 and O- Arachidonoyl Ethanolamine (Cayman Chemical, Ann Arbor, Michigan, USA), a CB_1 receptor antagonist, AM251 and a CB_2 receptor antagonist, AM630 (Wako Chemical, Osaka, Japan) were dissolved in 100% dimethyl sulfoxide (DMSO). Synthetic orexin-A and ghrelin were purchased from Peptide Institute, Osaka, Japan, and it was dissolved in normal saline.

Initially, we examined the dose-dependent effects of intraperitoneal injection of $CB_{1/2}$ agonists on the colonic distension-induced AWR threshold volume. Rats received intraperitoneal injections of several doses of WIN 55,212 or O-Arachidonoyl Ethanolamine.

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