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Effects of bombesin, vasoactive intestinal peptide and neurotensin on TRH-induced body shaking in rats[1]

G. Katsuura, K. Yoshikawa and S. Itoh

Shionogi Research Laboratories, Fukushima-ku, Osaka 553 (Japan), 26 May 1983

Summary. Bombesin, vasoactive intestinal peptide (VIP) and neurotensin were found to suppress body shaking behavior caused by intracerebroventricular injection of TRH.

Thyrotropin releasing hormone (TRH) induces a marked body shaking behavior[2-5] which can be suppressed by opioid agonists[6], particularly β-endorphin[4], and neurotensin (NT)[7]. The action mode of body shaking behavior in unknown. The aim of the present report was to study the interactions of TRH with other neuropeptides, bombesin (BBS), vasoactive intestinal peptide (VIP) and NT, using the body shakes as an index of the behavioral effect.

Materials and methods. Male Wistar rats, weighing 250–300 g, were housed at a constant temperature of 25°C under controlled illumination of a 12-h light:dark cycle (lights turned on at 07.00 h), with free access to standard rat biscuits and water. The method of intracerebroventricular (i.c.v.) injection has been described elsewhere[8]. Briefly, with the rat under pentobarbital anesthesia, a stainless steel guide cannula was fixed stereotaxically with dental cement on the skull at an appropriate position for insertion of an injection cannula into the left lateral ventricle. After a 5-day recovery period another 5 days were allowed for the animals to become accustomed to the insertion of the injection cannula in the morning. In the experiment, 5 µl of a peptide solution or physiological saline solution was injected with a microsyringe without anesthesia. After every experiment, the placement of the cannula was determined by injecting 5 µl of 1% Evans blue solution. The frequency of body shakes was counted visually for 30 min after the injection of neuropeptides into the lateral ventricle of the rat. The room temperature was maintained at 25°C.

The compounds used were TRH (Sigma), BBS (Osaka Protein Res. Foundation), VIP (Sigma) and NT (Osaka Protein Res. Foundation). Comparison of the data were performed by the method of Dunnett.

Results. As shown in table 1, 1.6 µg TRH produced vigorous body shake responses during the 30-min period. When 1 or 2 µg BBS, 5 or 10 µg VIP, or 10–40 µg of NT was injected, no body shaking response was observed (table 1).

The body shake scores when BBS, VIP and NT were injected together with 1.6 µg TRH are given in table 2. BBS in doses of more than 0.2 µg, VIP in doses of more than 2 µg and NT in doses of more than 5 µg significantly prevented the body shake responses induced by TRH. The antagonistic effect of BBS was particularly marked, while that of NT was slight.

Discussion. At present the mode and sites of action of neuropeptides on the incidence of body shaking response are obscure. Central actions of TRH have been shown to be related to enhanced turnover of catecholamines[9-12] and TRH-induced body shakes were suggested to be dependent on the brain dopamine[5]. The present results, that TRH-induced body shakes

Table 1. Body-shaking responses to i.c.v. injection of TRH, bombesin (BBS), vasoactive intestinal peptide (VIP) and neurotensin

Peptide	Dose (µg)	No. of rats	No. of shakes during 30 min
Saline		15	2 ± 0.7
TRH	1.6	14	90 ± 5.8**
BBS	1	9	2 ± 0.4
	2	10	2 ± 0.5
VIP	5	8	1 ± 0.9
	10	8	0 ± 0.0
NT	10	8	1 ± 0.3
	20	8	0 ± 0.0
	40	7	9 ± 0.2

** $p < 0.01$ vs saline (Dunnett's test). Means ± SEM.

Table 2. Effects of bombesin (BBS), vasoactive intestinal peptide (VIP) and neurotensin (NT) on TRH-induced body-shaking response

Peptide	Dose (μg)	No. of rats	No. of shakes during 30 min
TRH	1.6	14	90 ± 5.8
TRH (1.6 μg) plus			
BBS	0.1	8	85 ± 6.8
	0.2	9	53 ± 4.4**
	0.5	9	34 ± 5.2**
	1	11	27 ± 3.3**
	2	10	24 ± 4.3**
VIP	1	9	82 ± 6.1
	2	15	64 ± 6.7**
	5	15	54 ± 1.5**
	10	10	17 ± 4.5**
NT	2	8	78 ± 4.8
	5	8	63 ± 3.6**
	10	9	68 ± 3.4**
	20	8	55 ± 5.5**
	40	10	59 ± 4.5**

** $p < 0.01$ vs TRH alone (Dunnett's test). Means ± SEM.

were suppressed by BBS, VIP and NT may indicate that these neuropeptides affect the dopamine turnover induced by TRH in the brain. In this regard, NT has been reported to inhibit the dopaminergic system[13,14], but this is controversial in view of other observations[15,16]. The effects of BBS and VIP on catecholamine metabolism have not yet been clarified. Different mechanisms have been proposed for the shaking behavior, since activation of central serotonergic system[5,17] and i.c.v. administration of carbachol chloride[18] can also produce a body-shaking response in the rat. In this regard, we showed that VIP did not affect the dopaminergic system, but reduced serotonin-induced symptoms and that NT caused a pronounced decrease in serotonergic activities (unpublished data). It is possible that the stimulatory effects of TRH could be modified by these neuropeptides through their effects on the serotonergic system and probably other neurotransmitter systems in the brain are involved. It should be noted that even neuropeptides which are not effective alone could modify the effects of others in a strong positive or negative manner.

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Night shift paralysis[1]

S. Folkard, R. Condon and M. Herbert

MRC Perceptual and Cognitive Performance Unit, Laboratory of Experimental Psychology, University of Sussex, Falmer, Brighton, Sussex BN1 9QG (England) and Behavioural Science Section, University Hospital and Medical School, Clifton Boulevard, Nottingham NC7 2UH, (England), 15 August 1983

Summary. 12% of night nurses surveyed claimed to have suffered from a totally incapacitating paralysis that may be related to sleep paralysis, and contribute to impaired levels of safety on the night shift. The incidence of this paralysis is shown to be age-related, largely confined to the early hours of the morning, and to increase over consecutive night shifts.

Reduced levels of safety on the night shift[2-5] have usually been attributed to the nightworkers' low level of performance efficiency due to the combined effects of disrupted circadian rhythms[6-10] and partial sleep deprivation[11,12]. However, during the course of our investigations into the disruption of night nurses' circadian rhythms[5], we learned of a previously uninvestigated paralysis (known in the profession as 'night nurse paralysis') that may prevent nightworkers from performing their job for several minutes and might thus contribute to this problem of safety. We were told that this paralysis normally occurs when the nurse is performing a sedentary task in the early hours of the morning, and is then required to make a gross motor movement. This suggested that it may be a form of sleep paralysis which usually occurs during the transition between wakefulness and sleep[13] and is more common in young adults and adolescents[14].

Method and sample. We conducted a questionnaire survey of 434 nurses with experience of night work to determine the nature of this paralysis, its extent, and its dependence on age, time of day, and the number of consecutive nights the nurse had worked. This sample was drawn from 9 General Hospitals located in 4 different areas of England. The age range of the sample was 19–61 years, but was highly skewed with a median of only 31 years. Similarly, the nurses' experience of night duty ranged from less than 15 months to about 12 years, with a median of between 45 and 60 months. Only 24 (5.5%) of the