MORPHOMETRIC EVIDENCE OF ACTIVATION OF AXO-SOMATIC SYNAPSES BY DELTA-SLEEP INDUCING PEPTIDE

A. M. Mendzheritskii, G. A. Kuraev, I. I. Mikhaleva, and P. É. Povilaitite

UDC 612.822.014.46:[615.357:577.175.82

KEY WORDS: delta-sleep inducing peptide; synapses; morphometry.

Delta-sleep inducing peptide (DSIP), which belongs to the class of exogenous neuroregulators, causes an increase in the duration of the slow-wave component of sleep and possesses marked chronobiological and antistressor actions. As yet no receptors of DSIP have been discovered, and the prevailing view is that it has a modulating effect which is realized through traditional neurotransmitter systems. Data on its effect on both adrenergic and serotoninergic neurotransmitter systems have been obtained [1, 8]. Our previous investigations showed that activation of the GABA-ergic system takes place after both intraventricular and intraperitoneal administration of DSIP, and also against the background of stress [5, 6]. In the modern view, axo-somatic synapses on the bodies of pyramidal neurons are formed by inhibitory terminals of GABA-ergic interneurons [7].

It was therefore decided to study the state of ultrastructures of axo-somatic synapses in layers III-V of the neocortex 3 and 24 h after a single injection of DSIP in a dose of $12 \mu g/100 g$ body weight [6].

EXPERIMENTAL METHOD

Negatives with magnification of 3000× and 15,000× were prepared for quantitative study of characteristics of the synapses. Morphometric analysis was carried out on an IBAS automatic image analyzer (Opton, Germany), using prepared programs and having a programming facility [2].

EXPERIMENTAL RESULTS

The study of the ultrastructure of the synapses under magnification of $15,000 \times$ showed that a single intraperitoneal injection of DSIP in a dose of $12 \mu g/100$ g activates presynaptic endings on bodies of large pyramidal neurons in layers III-V of the rat sensomotor cortex. The formation of chains of synaptic vesicles and an increase in electron density of the active zone of contact were recorded, and small mitochondria with a condensed matrix were discovered in the nerve ending. To obtain additional factual evidence, the negatives were analyzed quantitatively.

Morphometric analysis of the plasmalemma of the pyramidal neurons in layers III-V, under magnification of 3000× revealed a significant increase in its mean length by 11.2% 3 h after injection of DSIP. By 24 h this parameter was back to normal. Changes in total length of the active zones of the synapses on neuron bodies were similar in character: a significant increase after exposure for 3 h by 12.4%, followed by a return to normal after 24 h. There was a tendency for the number of synapses per micron of the neuronal plasmalemma to increase. After 24 h this tendency became more marked (11.5%), but did not reach the level of significance. Changes in the width of the synaptic cleft, which is the principal index of informativeness of the synaptic junction, are particularly interesting [4]. Measurements of width were made in 10 randomly

Research Institute of Neurocybernetics, Rostov University. (Presented by Academician of the Academy of Medical Sciences of the USSR I. P. Ashmarin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 113, No. 2, pp. 202-203, February, 1992. Original article submitted July 16, 1991.

TABLE 1. Changes in Width of Synaptic Space 3 and 24 h after Injection of DSIP $(M \pm m)$

Conditions	Width of space, mm
Normal (230 synapses) 3 h of DSIP (200 synapses) 24 h of DSIP (290 synapses)	$22,867 \pm 0,2596$ $24,5534 \pm 0,2464*$ $23,9688 \pm 0,2829*$

Legend. *p < 0.05: Differences statistically significant.

chosen points on synapses cut through the center, where pre- and postsynaptic membranes were clearly visible. The results in Table 1 show that 3 h after injection of DSIP the width of the synaptic cleft was increased by 10.7%, which is highly significant (p < 0.0001). After 24 h this parameter fell to 10.4%, but still remained significantly greater than in the control (p < 0.0022).

It has to be pointed out that the synaptic cleft is a special form of intermembrane contact, and the chemical components of the synaptic cleft are responsible for the firm adhesive properties of this junction [3]. Even slight variations in the distance from the presynaptic to the postsynaptic terminal are possible only in the case of a considerable change in structure of the cytoskeleton of the pre- and post-synapse, It thus becomes clear that even a very small increase in width of the synaptic cleft will be evidence of activation of synapses of this type on the bodies of pyramidal neurons under the influence of DSIP. Activation of axo-somatic synapses thus revealed correlates with an increase in the GABA content and activation of the GABA-forming enzyme glutamate decarboxylase, and with the slow-wave activity recorded in the cerebral cortex [6].

LITERATURE CITED

(Missing in Russian Original — Publisher.)

NUCLEAR BODIES IN RAT HEPATOCYTES: DYNAMICS OF APPEARANCE AND FORMATION DURING AGING AND FUNCTIONAL LOADS

N. V. Berezhkov

UDC 612.35.014:576.315].06:612.66/.67

KEY WORDS: chromatin; ultrastructure; aging; blood loss; enterosorption.

The morphological equivalent of the genome is chromatin, and the state of expression of genetic information can be judged from changes in the fine structure of chromatin. For instance, the change from heterochromatin to euchromatin indicates activation of nucleic acid synthesis, as may be clearly demonstrated by electron-autoradiographic studies [4]. Besides the periodic condensation and decondensation of chromatin, and changes in conformation of the nucleoli and interchromatin granules, which have been adequately studied, the so-called nuclear bodies (NB), or foci with altered

Department of Pathomorphology, A. V. Vishnevskii Institute of Surgery, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR D. S. Sarkisov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 113, No. 2, pp. 203-208, February, 1992. Original article submitted February 21, 1991.