D2 DOPAMINE RECEPTOR AGONISTS INFLUENCE IN THE ANIMAL MODEL OF DIETARY OBESITY

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**Background**

Increased caloric intake in dietary obesity (DO) could be driven by central mechanisms regulating reward-seeking behavior. According to the current studies dopamine as a neurotransmitter turns out to regulate food intake [1] by modulating food reward by means of the mesolimbic circuitry of the brain [2]. Medications that block dopamine D2 receptors increase appetite and result in significant weight gain [3] whereas ones that increase brain dopamine concentration are anorexigenic [4]. However, the role of dopamine in pathological consumption and obesity is not clear understood.

Dopaminergic neurotransmission in the ventral basal ganglia plays an integral role in the response to salient rewarding stimuli including drugs of abuse, sex, social bonding, and food [5] and work in animal models suggests leptin modulates central dopamine function [6].

**Objectives**

We supposed D2 dopamine receptor agonists to influence weight gain and leptin level in genetically unmodified rats with high caloric diet (HCD) as dietary obesity model.

**Methods**

Young male rats (n=48), with body mass equals 183.0±14.0 g, were divided into HCD group (n=24) and control (C) (n=24, standard diet) depends on caloric intake. During 3 months of research, 8 rats from both groups were daily injected intraperitoneally with D2 dopamine receptor agonist Bromocriptin (Br) (1mg/kg), 8 rodents - dimethyl sulfoxide (DMSO) (1 ml/kg) used as diluent for Br. Length, weight and caloric intake were registered twice a week. Animals’ serum leptin levels were measured by immunoenzymatic analysis with standardization relatively to weight (leptin/weight ratio, LWR) at the 1st and 3rd months of experiment.

Rodents’ total mobility (TM, assessed as a complex of total distance and speed) was assessed by plus maze test at the same time. Experimental results were compared in subgroups, depends on pharmacological agent with noninjected animals (comparison groups). Nonparametric analysis was performed (SPSS 16.0, p<0.05).

**Results**

HCD rats showed weight gain in 1st and 3rd months irrespective to injected agent (p>0.05) (fig. 1).

Weight gain was similar in Br injected HCD rats and HCD group (p>0.05) (fig. 2).

Br injected HDC rats showed TM decrease (fig. 3, 4) and closed arm time increase in 1 month relative to HCD group (fig. 6). These changes were leveled in 3 months.

**Conclusions**

Leptin and LWR changes without weight gain and total mobility modifications in Bromocriptin injected HCD rat give a reason to suppose that long term Bromocriptin administration prevent obesity in genetically unmodified HCD rats.

**References**