The long-term consumption of ethanol by pregnant or nursing rat mothers results in extensive damage of the hippocampal area in their offspring. The histochemical methods combining bis-benzimide 33342 (Hoechst) and Fluoro-Jade B staining were used to detect ethanol effects on the structure of the rat hippocampus and gyrus dentatus during development and adult age (18, 35, 90 and 360 days old offspring).

In all experimental groups, in all analyzed areas, degenerative changes were observed, loss of pyramidal and granular cells and neural cells with segmented nuclei. In animals, whose mothers drunk 20% ethanol, structural changes were more intensive when compared with the group consuming 10% ethanol.

The highest density of the degenerating (FJ/B positive) cells was found in 18-days-old animals whose mothers were exposed to 20% ethanol. In the age of 90 and 360 days no degenerating cells were identified in the monitored areas.

With respect to the properties of the ethanol and mechanism of its effect we suppose that exposure to ethanol during the perinatal period induced the neural cell loss by apoptotic mechanism.

From the results of the work can be concluded:

1. The perinatal exposure to ethanol results in degeneration of neural cells in the hippocampus and gyrus dentatus (working hypothesis I was confirmed).
2. The rate of degenerative changes of the neural cells in the hippocampus and gyrus dentatus is directly proportional to the concentration of the ethanol consumed (working hypothesis II was confirmed).
3. Exposure to ethanol evoked probably apoptosis in the all observed areas (working hypothesis III was confirmed).
4. The qualitative result of ethanol effect was the same in different age groups (degeneration and loss of neural cells), difference was only quantitative (working hypothesis IV was not fully confirmed).