At the end of the 20th century it was known that cannabinoid drugs interact with two receptors, CB_1 and CB_2. Subsequent pharmacological studies have confirmed that there are other receptors interacting with cannabinoids. GPR55 is a transmembrane G protein coupled receptor, which together with the receptor GPR18 and GPR119 belong to a group of new cannabinoid receptors and is involved in the function of the endocannabinoid system. In addition to some of cannabinoid substances, it is stimulated primarily phospholipid lysofosfatidylinositolem. LPI-dependent signaling GPR55 plays an important role in the regulation of many physiological and pathological processes, such as pain, inflammation, cell proliferation, or endothelial function. It was found that LPI confers tolerance to ischemic brain damage and has a cytoprotective effect on the pyramidal cells. The aim of the study was to determine whether the application of five ligands induce phosphorylation of protein kinase ERK 1/2, Akt and activate the GTPase RhoA and whether activation of the receptor GPR55 has cytoprotective effect in model cell line PC12, in which hypoxic conditions were simulated by adding $CoCl_2$. For working methods were used SDS-PAGE, Western bloting and colorimetric measurement. Pharmacological studies in recent years have shown inconsistency in the classification ligand for GPR55. Given that some research lies between the specific ligand agonists, antagonists or different between them does not assign any effect, it was important to determine how the concentration, respectively, the time series of action of ligands affected the GPR55 signaling pathway. LPI was determined in all scientific work as the GPR55 agonist. Activation of GPR55 may play a crucial role in the regulation of apoptosis. Determination of expression of regulatory proteins Bax, Bcl-2 helped to clarify whether the action of $CoCl_2$ induced their expression and whether the GPR55 stimulation had anti-apoptotic effects. We failed to determine the dependence of the intensity of phosphorylation by protein kinases to the increasing concentration of the ligand and failed to acknowledge that the application of the inverse agonist GPR55 CID-16020046 decreased phosphorylation induced by another ligand. By Western blotting overexpression of Bax was detected after application of $CoCl_2$.These results show the ambiguous classification of ligands for GPR55 and demonstrate that $CoCl_2$ mimics hypoxic conditions and the formation of ROS and induces apoptosis by increasing the expression of proapoptotic protein Bax.