Figure Legends

Figure 70

Confocal image of NMDAR1 receptors in the cerebral cortex of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent NMDAR1 receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of NMDAR1 receptors in the cerebral cortex of experimental rats when compared to control rats. ➔ in pink shows NMDAR1 receptors.

Figure 71

Confocal image of NMDA2B receptors in the cerebral cortex of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent NMDA2B receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of NMDA2B receptors in the cerebral cortex of experimental rats when compared to control rats. ➔ in pink shows NMDA2B receptors.

Figure 72

Confocal image of mGluR5 receptors in the cerebral cortex of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent mGluR5 receptor specific primary antibody and Rhodamine as secondary antibody. There was an up regulation of mGluR5 receptors in the cerebral cortex of experimental rats when compared to control rats. ➔ in pink shows mGluR5 receptors.

Figure 73

Confocal image of IP3 receptors in the cerebral cortex of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent IP3 receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of IP3 receptors in the cerebral cortex of experimental rats when compared to control rats. The ➔ in pink shows IP3 receptors.
Figure 74

Confocal image of NMDAR1 receptors in the cerebellum of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent NMDAR1 receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of NMDAR1 receptors in the cerebellum of experimental rats when compared to control rats. The → in pink shows NMDAR1 receptors.

Figure 75

Confocal image of NMDA2B receptors in the cerebellum of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent NMDA2B receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of NMDA2B receptors in the cerebellum of experimental rats when compared to control rats. The → in pink shows NMDA2B receptors.

Figure 76

Confocal image of mGluR5 receptors in the cerebellum of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent mGluR5 receptor specific primary antibody and Rhodamine as secondary antibody. There was an up regulation of mGluR5 receptors in the cerebellum of experimental rats when compared to control rats. → in pink shows mGluR5 receptors.

Figure 77

Confocal image of IP3 receptors in the cerebellum of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent IP3 receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of IP3 receptors in the cerebellum of experimental rats when compared to control rats. → in pink shows IP3 receptors.

Figure 78

Confocal image of NMDAR1 receptors in the pancreas of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent NMDAR1 receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of NMDAR1 receptors in the pancreas of experimental rats when compared to control rats. The → in pink shows NMDAR1 receptors.
antibody and FITC as secondary antibody. There was an up regulation of NMDAR1 receptors in the pancreas of experimental rats when compared to control rats. → in pink shows NMDAR1 receptors.

Figure 79

Confocal image of NMDA2B receptors in the pancreas of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent NMDA2B receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of NMDA2B receptors in the pancreas of experimental rats when compared to control rats. → in pink shows NMDA2B receptors.

Figure 80

Confocal image of mGluR5 receptors in the pancreas of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent mGluR5 receptor specific primary antibody and Rhodamine as secondary antibody. There was an up regulation of mGluR5 receptors in the pancreas of experimental rats when compared to control rats. The → in pink shows mGluR5 receptors.

Figure 81

Confocal image of IP3 receptors in the pancreas of control, Diabetic, D+IIH and C+IIH rats using immunofluorescent IP3 receptor specific primary antibody and FITC as secondary antibody. There was an up regulation of IP3 receptors in the pancreas of experimental rats when compared to control rats. → in pink shows IP3 receptors.

Figure 82

Confocal image of calcium release from the pancreas in the presence of 1 mM Glucose, 10^{-5} M Dopamine, 10^{-5} M Dopamine + 10^{-5} M Sulpiride. Dopamine inhibited the calcium release from pancreatic islets. The presence of 10^{-5} M Dopamine and Sulpiride reversed the inhibition.
Figure 83

Confocal image of calcium release from the pancreas in the presence of 4 mM Glucose, $10^{-5}$ M Dopamine, $10^{-5}$ M Dopamine + $10^{-5}$ M Sulpiride. Dopamine increased the calcium release from pancreatic islets. The presence of $10^{-5}$ M Dopamine and Sulpiride reversed the calcium release.

Figure 84

Confocal image of calcium release from the pancreas in the presence of 20 mM Glucose, $10^{-5}$ M Dopamine, $10^{-5}$ M Dopamine + $10^{-5}$ M Sulpiride. Dopamine increased the calcium release from pancreatic islets. The presence of $10^{-5}$ M Dopamine and Sulpiride reversed the calcium release.

Figure 85

Confocal image of calcium release from the pancreas in the presence of 1 mM Glucose, $10^{-5}$ M Glutamate, $10^{-5}$M Glutamate + MK801. Glutamate increased the calcium release from pancreatic islets. The presence of $10^{-5}$ M Glutamate and MK801 reversed the calcium release.

Figure 86

Confocal image of calcium release from the pancreas in the presence of 4 mM Glucose, $10^{-5}$M Glutamate, $10^{-5}$M Glutamate + MK801. Glutamate increased the calcium release from pancreatic islets. The presence of $10^{-5}$ M Glutamate and MK801 reversed the calcium release.

Figure 87

Confocal image of calcium release from the pancreas in the presence of 20 mM Glucose, $10^{-5}$M Glutamate, $10^{-5}$M Glutamate + MK801. Glutamate increased the calcium release from pancreatic islets. The presence of $10^{-5}$ M Glutamate and MK801 reversed the calcium release.