SUPPLEMENTARY MATERIALS I. M. STUDNEVA, ET AL. "GALANIN REDUCES MYOCARDIAL ISCHEMIA/REPERFUSION INJURY IN RATS WITH STREPTOZOTOCIN DIABETES"

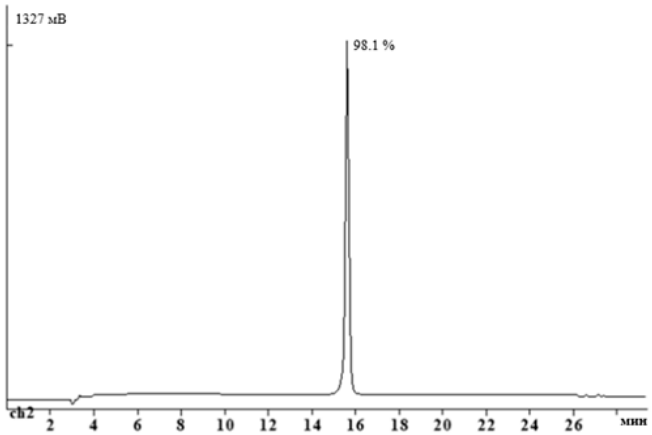

Fig. S1. Analytical HPLC profile of peptide G after purification. HPLC conditions: Kromasil-C18-100 column (4.6 × 250 mm), 5 μm particle size; elution rate – 1 ml/min; detection at λ = 220 nm. Mobile phase: buffer A – 0.1% trifluoroacetic acid (TFA); buffer B – 80% acetonitrile in buffer A; elution with a 20–80% linear gradient of buffer B in buffer A for 30 min

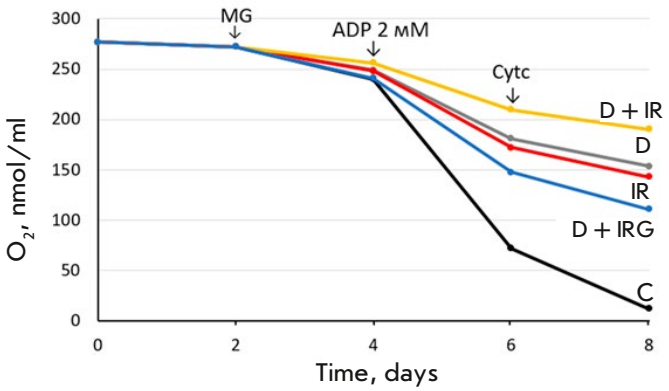

Fig. S3. Representative protocols of experiments in state 3 mitochondrial respiration in saponin-skinned LV cardiac fibers. C – control; D – rats receiving STZ; D – rats receiving STZ (60 mg/kg, i.v.); IR – rats subjected to regional myocardial IRI; D + IR – DM rats (STZ 60 mg/kg, i.v.) subjected to regional myocardial IRI; D + IRG – DM rats (STZ 60 mg/kg, i.v.) subjected to regional myocardial IRI, receiving G (i.v. at a dose of 1 mg/kg, bolus at the onset of reperfusion). GM – 10 mM glutamate and 5 mM malate, Cytc – 10 μM cytochrome *c*

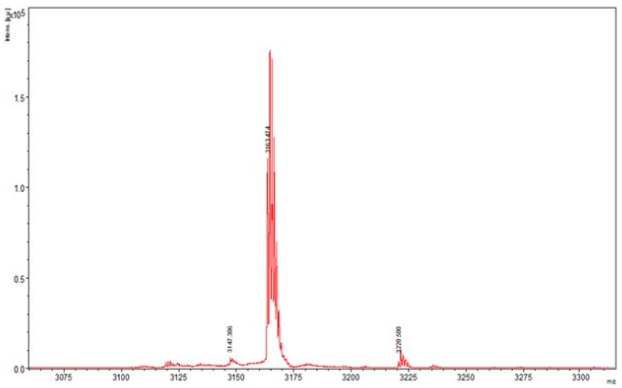

Fig. S2. Mass spectrum of peptide G. $M_{calculated}$ = 3,164.45; found m/z – 3,163.47 $[M + H]^+$

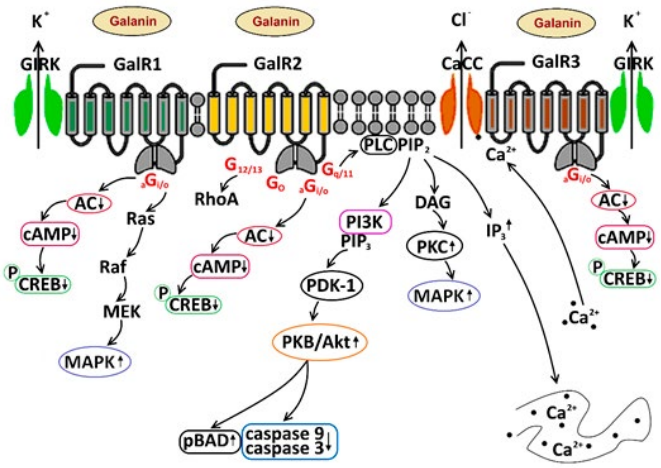

Fig. S4. Intracellular signaling pathways activated by galanin. AC – adenylate cyclase; (p)BAD – (phosphorylated) BCl-2-regulator of apoptosis; CaCC – Ca^{2+}-dependent chloride channel; cAMP – cyclic AMP; (p)CREB – phosphorylated cAMP-dependent element; DAG – diacylglycerol; GIRK – G-protein-coupled potassium influx channel; IP_3 – inositol triphosphate; MAPK – mitogen-activated protein kinase; PDK-1 – phosphoinositol-dependent protein kinase-1; PIP_2 – phosphatidylinositol 4,5-diphosphate; PIP_3 – phosphatidylinositol 3,4,5-triphosphate; PI3K – phosphatidylinositol 3-kinase; PKB – protein kinase B (Akt); PLC – phospholipase C; RhoA – Ras homolog family member A