List of Figures

Figure 1. 1. Types of nanoparticles
Figure 1. 2. (a) Structure of heart (b) Blockage of coronary artery
Figure 1. 3. Factors contributing to I/R
Figure 1. 4. (a) Guar beans (b) Guar gum powder (c) Structure of guar gum
Figure 2. 1. Particle size distribution of (a) GGN and (b) SGG
Figure 2. 2. TEM images of (a) GGN (b) SGG (c) EDX spectrum of SGG
Figure 2. 3. XRD images of (a) GG (b) Sodium selenite and (c) SGG
Figure 2. 4a. Particle size distribution for (a) SGG in serum containing DMEM (b) SGG in serum free DMEM
Figure 2. 4b. Particle size distribution for (a) GGN in serum free DMEM (b) GGN in serum containing DMEM
Figure 2. 5. Morphological examination of cells with nanoparticles
Figure 2. 6A. Uptake of nanoparticle by H9c2 cardiac myoblasts for 1h incubation with various concentrations of SGG
Figure 2. 6B. Uptake of nanoparticle by H9c2 cardiac myoblasts for 6 h incubation with various concentrations of SGG
Figure 2. 6C. Uptake of nanoparticle by H9c2 cardiac myoblasts for 24 h incubation with various concentrations of SGG
Figure 2. 7. In vitro cellular uptake of Se from sodium selenite and SGG
Figure 2. 8. Alteration in DNA integrity with Se and SGG
Figure 2. 9A. DNA damage protection assay with Fenton’s reagent
Figure 2. 9B. The effect of nanoparticle on plasmid DNA
Figure 2. 10A. Evaluation of ROS with various doses of Se and SGG after 1 h
Figure 2. 10B. Evaluation of ROS with various doses of Se and SGG after 6 h
Figure 2. 10C. Evaluation of ROS with various doses of Se and SGG after 24 h
Figure 2. 11A. Mitochondrial transmembrane potential with Se and SGG for 1 h
Figure 2. 11B. Mitochondrial transmembrane potential with Se and SGG for 6 h
Figure 2. 11C. Mitochondrial transmembrane potential with Se and SGG for 24 h
Figure 2. 12. Effect of Se and SGG on cytoskeleton of H9c2 cells
Figure 3. 1. Effect of Se, GGN and SGG on the morphology of H9c2 after I/R
Figure 3. 2. Fluorescent microscopic images of H9c2 cells stained for glutathione evaluation
Figure 3.3. Activity of XO in control and treated H9c2 cells after I/R
Figure 3.4. Activity of Nrf2 in control and treated H9c2 cells after I/R
Figure 3.5A. Effect of Se, GGN and SGG on ROS generation in I/R induced H9c2 cells
Figure 3.5B. Flow cytometric analysis of intracellular ROS generation in different groups
Figure 3.6. Intracellular calcium overload in H9c2 with Se, GGN and SGG treatment
Figure 4.1A. Mitochondrial transmembrane potential determined by JC-1 staining
Figure 4.1B. Integrity of permeability transition visualized by calcein and cobalt chloride staining
Figure 4.2. Fluorescent microscopic images of H9c2 cells stained with MitoSOX™ Red indicator
Figure 4.3. Oxygen consumption rate in different groups
Figure 4.4. ATP content in control and treated H9c2 cells after I/R
Figure 4.5. Aconitase enzyme activities in different treated groups
Figure 4.6. HIF-1 α transcription factor in nuclear extract of control, ischemia, I/R and treated cells
Figure 4.7. Changes in ANP level in control, ischemia, I/R and treatment groups
Figure 5.1. Estimation of inflammatory cytokines release by ELISA in control, ischemia, I/R and treatment groups. (A) IL-2 (B) IL-6 (C) MCP-1 (D) IFN-γ (E) TNF-α.
Figure 5.2. Estimation of NF-κB (p65) level in control and treated cells after I/R.
Figure 5.3. Estimation of TNNI3K level in control and treated cells after I/R.
Figure 5.4. Activity of caspase 3 in the control and treated cells after I/R.
Figure 5.5. Effects of Se, GGN and SGG on rate of apoptosis
Figure 5.6. Effect of SGG on ischemia and I/R induced cytoskeleton disorganization.
Figure 5.7. SGG upregulates mRNA levels of IGF-1, Raf-1, ERK-1 and ERK-2.
Figure 5.8. Expression of (a) Bax and (b) Bcl-2 protein ischemia and I/R subjected to SDS-PAGE.
Figure 5.9. Expression of (a) Raf-1 and (b) ERK1/2 and p-ERK1/2 protein in ischemia and I/R subjected to SDS-PAGE.