LIST OF FIGURES

Chapter 2:

Figure 1. The wurtzite structure model of ZnO. The tetrahedral coordination of Zn-O is shown.

Figure 2. (a) Scanning electron microscopic image of ZNP(N); (b) Transmission electron micrograph of ZNP(N) and energy-dispersive X-ray analysis of ZNP(N) (inset).

Figure 3. (a) Scanning electron microscopic image of ZNP(C); (b) Transmission electron micrograph of ZNP(C) and energy-dispersive X-ray analysis of ZNP(C) (inset).

Figure 4. Measurements of hydrodynamic radius of ZNP(T) by using DLS.

Figure 5. (a) UV-VIS absorbance spectra of synthesized ZNP(T), (b) X-ray diffraction pattern of ZNP(T).

Figure 6. (a) FTIR spectra of ZNP(T), (b) Measurement of zeta potential as a function of pH.

Figure 7. (a) Morphological analysis of ZNP(T) by FESEM; inset indicating the EDX spectrum of ZNP(T), (b) HR-TEM image of ZNP(T).

Figure 8. Topological attributes of ZNPs as determined from AFM studies (a) 1D and (b) 3D view.

Figure 9. (a) Thermogravimetric weight loss of ZNP(T) as a function of temperature, (b) PL spectra of ZNP(T).

Figure 10. (a) Morphological analysis of ZNP(S) by FESEM; (b) HR-TEM image of ZNP(S).

Figure 11. (a) IR spectra and (b) XRD of ZNP(S).

Figure 12. (a) Morphological analysis of ZNP(P) by FESEM; (b) HR-TEM image of ZNP(P).

Figure 13. (a) IR spectra and (b) XRD of ZNP(P).

Chapter 3:
Figure 1. Comparative antifungal effects of ZNP(T) and MZnO against *A. niger* strains: MTCC 10180 & MTCC 2196 [(a) and (b)] and *F. oxysporum* strains: NCIM 1043 & NCIM 1072 [(c) and (d)] compared to control; Reduction in radial growth (mean of zone diameter ± standard deviation) of (e) *A. niger* strains: MTCC 10180 and MTCC 2196, and (f) *F. oxysporum* strains: NCIM 1043 and NCIM 1072 after treatment with control (water), MZnO and ZNPs. Where four fungal strains MTCC 10180, MTCC 2196, NCIM 1043 and NCIM 1072 were denoted by 1, 2, 3 and 4 respectively.

Figure 2. Comparative antifungal effects of MZnO, ZNP(N), ZNP(C), ZNP(S), ZNP(P) and ZNP(T) against *A. niger* (MTCC 2196) [(a) and (b)] compared to control; Reduction in radial growth (mean of zone diameter ± standard deviation) of *A. niger* (MTCC 2196) after treatment with control (water), MZnO and various uncapped and capped ZNPs [ZNP(N), ZNP(C), ZNP(S), ZNP(P) and ZNP(T)].

Figure 3. Comparative antifungal effects of MZnO, ZNP(N), ZNP(C), ZNP(S), ZNP(P) and ZNP(T) against *F. oxysporum* (NCIM 1043) [(a) and (b)] compared to control; Reduction in radial growth (mean of zone diameter ± standard deviation) of *F. oxysporum* (NCIM 1043) after treatment with control (water), MZnO and various uncapped and capped ZNPs [ZNP(N), ZNP(C), ZNP(S), ZNP(P) and ZNP(T)].

Chapter 4:

Figure 1. Comparative analysis of (a) Superoxide dismutase activity, (b) Ascorbate peroxidase activity, (c) Catalase activity, (d) Glutathione reductase (GR) activity, (e) Change in intracellular thiol content and (f) Extent of lipid peroxidation in fungal strains (*A. niger* and *F. oxysporum*) induced by two different concentrations (250 ppm and 500 ppm) of ZNPs respectively in comparison to control; where four fungal strains MTCC 10180 (*A. niger*), MTCC 2196 (*A. niger*), NCIM 1043 (*F. oxysporum*) and NCIM 1072 (*F. oxysporum*) were denoted by 1, 2, 3 and 4 respectively.

Figure 2. Change in proline content in fungal strains (*A. niger* and *F. oxysporum*) treated with different ZNPs concentrations (250 ppm and 500 ppm).
Figure 3. (a) Estimation of carbonyl content (as a marker of oxidative stress) of ZNPs treated fungal strains (*A. niger* and *F. oxysporum*) which resulted the protein damage, Where four fungal strains MTCC 10180, MTCC 2196, NCIM 1043 and NCIM 1072 were denoted by 1, 2, 3 and 4 respectively.; (b) FTIR analysis of fungal cell (*A. niger*, MTCC 10180) : (A) Control (B) 250 ppm ZNPs treated (C) 500 ppm ZNPs treated; (c) Comparative study of gene expression of ZNPs and MZnO treated *A. niger* (MTCC 10180).

Figure 4. SDS PAGE gel electrophoresis of control and ZNPs treated samples of *A. niger*. Increase of protein carbonylation (marker of oxidative stress) was shown in Lane 4 and 5 (250 ppm ZNPs treated *A. niger* sample) and Lane 7 and 8 (250 ppm ZNPs treated *A. niger* sample) with respect to control (Lane 1 and 2). Lane 3 and 6 denoted molecular marker (Protein marker, BLM003, 14-95 kDa, SRL).

Figure 5. Western blot analysis of control and ZNPs treated samples of *A. niger*. Immuno blot analysis of control and ZNPs treated (250 ppm and 500 ppm) *A. niger* samples with oxyblot detection kit (Millipore). Figure 3 (a) showed SDS PAGE of protein carbonylation (Lane 1,2 denoted control, Lane 3, 6 denoted molecular marker, Lane 4, 5 denoted 250 ppm ZNPs treated *A. niger* sample and Lane 7, 8 denoted 500 ppm ZNPs treated *A. niger* samples). Figure 3 (b) showed corresponding western blot analysis of corresponding protein where Lane 1,2 denoted control, Lane 3, 4 denoted 250 ppm ZNPs treated *A. niger* sample and Lane 5, 6 denoted 500 ppm ZNPs treated *A. niger* samples.

Figure 6. AFM study of Fungal hypha (*A. niger*, MTCC 10180): (a) Control (b) 250 ppm ZNPs treated (c) 500 ppm ZNPs treated.

Figure 7. Upper panel-FE SEM study of Fungal hypha (*A. niger*, MTCC 10180): (a) Control (b) 250 ppm ZNPs treated (c) 500 ppm ZNPs treated; Lower panel- Outer cell surface study of fungal hypha by HRTEM (*A. niger*, MTCC 10180): (d) Control (e) 250 ppm ZNPs treated and (f) 500 ppm ZNPs treated. Arrows in the lower panel demonstrated cell membrane damage.
Figure 8. Zn$^{2+}$ release from ZNPs by ICPMS analysis at three different interval of times 24h, 48h and 72h respectively. A small amount of Zn$^{2+}$ was released from ZNPs at the pH of fungal culture medium which could simultaneously contribute to the fungitoxic effect of ZNPs.

Figure 9. (a) EDX analysis of ZNPs uptake by A. niger (MTCC 10180), inset illustrated corresponding SEM image, (b) HR-TEM image of ultrathin section of A. niger (MTCC 10180) hypha, where arrows demonstrated distribution of ZNPs on the fungal hypha.

Figure 10. Selected area diffraction pattern (SAED) of (a) ZNPs and (b) ZNPs after internalization on fungal hyphae. The first one i.e. native ZNPs exhibited distinct crystalline structure but after internalization on to fungal hyphae its crystalline pattern was partially lost but still present.

Figure 11. Confocal microscopic image of (a), (b), (c) Control A. niger (MTCC 10180) sample; (d), (e), (f) RITC tagged ZNPs treated A. niger (MTCC 10180) sample, where the later one clearly demonstrated bright red fluorescence at 540 nm excitation wavelength of RITC tagged ZNPs distributed on fungal sample.

Figure 12. (a) Cell membrane integrity assay by DNA release study; (b) Outer membrane permeability assay of control and ZNPs treated E.coli (MTCC 443).

Figure 13. Outer membrane permeability assay of ZNPs treated E.coli (MTCC 443) compared to control.

Figure 14. FESEM image of control (a) and ZNPs treated E.coli (MTCC 443) (b).

Figure 15: Band gap of ZNPs

Chapter 5:

Figure 1. Upper and lower panel showed H&E stained control, 0.5g/kg ZNP(T) treated, 1.0 g/kg treated, 2.0 g/kg treated mice liver and kidney respectively (magnification = 20x).
Figure 2. Diagrammatic representation of relative hematology indicators such as (a) Haemoglobin, (b) Platelets count, (c) Red blood cell count (RBC), (d) White blood cell count (WBC), (e) Lymphocytes, (f) Monocytes, (g) Eosinophils and (h) Neutrophils of Control samples, MZnO treated mice at maximum concentration after 2 months (intravenous injection) and ZNPs treated mice at maximum concentration after 2 months (intravenous injection).

Figure 3. Diagrammatic representation of relative serum biochemical indicators such as (a) Alkaline phosphatase (ALP), (b) Lactate dehydrogenase (LDH), (c) Creatinine, (d) Blood urea nitrogen (BUN), (e) Uric acid, (f) Total protein (TP), (g) Serum glutamic pyruvic transaminase (SGPT) and (h) Serum glutamic oxaloacetic acid (SGOT) of Control samples, MZnO treated mice at maximum concentration after 2 months (intravenous injection) and ZNPs treated mice at maximum concentration after 2 months (intravenous injection).

Figure 4. Diagrammatic representation of other biochemical parameters such as (a) Triglyceride (TG), (b) Cholesterol and (c) Phosphorus of Control samples, MZnO treated mice at maximum concentration after 2 months (intravenous injection) and ZNPs treated mice at maximum concentration after 2 months (intravenous injection).

Figure 5. Major organs such as brain, lung, heart, liver, kidney, spleen and stomach of Control mice (left panel), MZnO (500 μg/mL) intravenously injected mice after 2 months (middle panel) and ZNPs (500 μg/mL) intravenously injected mice after 2 months (right panel).

Figure 6. Histopathological analysis of major organs such as brain, heart, kidney, liver, lung, spleen, testis and uterus of Control mice (left panel), MZnO (500 μg/mL) intravenously injected mice after 2 months (middle panel) and ZNPs (500 μg/mL) intravenously injected mice after 2 months (right panel).

Figure 7. *In vivo* haemolysis experiment on mice model. Maximum concentration used for long term toxicity determination was used for this haemolysis experiment.
Figure 8. Comparative acetylcholine esterase activity study of mice brain [control and ZNPs treated (2g/kg)].

Figure 9. Estimation of leaf area, root length, shoot length of control and ZNP treated plants.

Figure 10. Estimation of dry weight of control and ZNPs treated plants.

Figure 11. Estimation of total protein of control and ZNPs treated plants.

Figure 12. Estimation of total amino acid of control and ZNPs treated plants.

Figure 13. Estimation of total lipid of control and ZNPs treated plants.

Figure 14. Estimation of total chlorophyll content of control and ZNPs treated plants.

Figure 15. Estimation of total carotene content of control and ZNPs treated plants.

Figure 16. Estimation of total xanthophyll content of control and ZNPs treated plants.

Figure 17. (a) Survivability of MRC-5 cells after 72 h incubation with ZNP(T) and MZnO using WST assay; (b) Percentage of cytotoxicity of MRC-5 cells after 72 h incubation with ZNP(T) and MZnO using LDH assay.