List of Figures

Figure No.	Description	Page No.
2.1.	Types of diabetes based on pathophysiology	8
2.2.	Diagrammatic representation of ATP production from glucose	11
2.3.	Antioxidant defence mechanism in mitochondria and its involvement in type 2 diabetes	13
2.4.	<i>Ficus religiosa</i> : (A) Tree; (B) Leaves; (C) figs; (D) stem bark; (E) root	22
2.5.	Schematic representation of mitochondrial targeting of nanoparticles loaded with antioxidant	27
2.6.	Structure of SLN	36
2.7.	Structure of triphenylphosphonium	38
2.8.	Structure of glyceryl monostearate	40
2.9.	Structure of compritol ATO 888	42
2.10.	Structure of poloxamer 188	44
2.11.	Structure of polysorbate 80	46
2.12.	Structure of lecithin	49
2.13.	Structure of sodium deoxycholate	50
5.1.	TLC finger printing of lupeol standard and <i>Ficus religiosa</i> Linn. extract	64
5.2.	LCMS (positive mode) spectrum of ethanolic extract of <i>Ficus religiosa</i> Linn.	65
5.3.	FTIR spectrum of lupeol in Ficus religiosa Linn.	66
5.4.	¹ H NMR spectrum of lupeol in <i>Ficus religiosa</i> Linn.	67
5.5.	¹³ C NMR spectrum of marker compound of <i>Ficus religiosa</i> Linn.	68
5.6.	Structure of lupeol	69

5.7.	HPLC chromatograms of A – <i>Ficus religiosa</i> Linn. extract and B – lupeol standard	71
6.1.	In vitro release profile of lupeol from standard and SLN formulations A) in pH 1.2 for first two hours followed by pH 6.8 and C)) in pH 7.4.	94
6.2.	Average particle size of SLN formulations	97
6.3.	SEM images of SLN formulations prepared using single and binary surfactants	101
6.4.	Diagrammatic representation of mechanisms of increase in particle size of SLNs prepared using single surfactants and maintenance of particle size of SLNs with binary surfactants	104
6.5.	Diagrammatic representation of mechanisms of drug leaching from SLNs prepared using single surfactant and binary surfactants	106
6.6.	3D images of A - effect of homogenization speed and time on particle size and B - effect of sonication time and intensity on particle size	112
6.7.	3D images of A - effect of homogenization speed and time on PDI and B - effect of sonication time and intensity on PDI	114
6.8.	3D images of A - effect of homogenization speed and time on zeta potential and B - effect of sonication time and intensity on zeta potential	115
6.9.	<i>In-vitro</i> drug release studies of suspension of <i>Ficus religiosa</i> Linn. extract and optimized batch of SLN	116
6.10.	FTIR spectra: A - <i>Ficus religiosa</i> Linn. Extract, B – Glyceryl monostearate, C – Compritol ATO 888, D – SLN prepared using glyceryl monostearate and E – SLN prepared using compritol ATO 888	118
6.11.	DSC thermograms of A - <i>Ficus religiosa</i> Linn. extract, B – Glyceryl monostearate, C – Compritol ATO 888, D – SLN prepared using glyceryl monostearate and E – SLN prepared using compritol ATO 888	119

6.12.	PXRD spectra	120
6.13.	Characterization of core CdSe quantum dots; A) UV - Visible absorption spectrum, B) FTIR spectrum and C) PXRD pattern	121
6.14.	<i>In vitro</i> characterization of nanoparticles A) diagrammatic representation of ETNPs, B) particle size distribution curve, zeta potential curve and SEM image of ETNPs, C) particle size distribution, zeta potential curve and SEM image of EUNPs, and D) <i>In vitro</i> release profiles of <i>Ficus relgiosa</i> L. extract, EUNPs and ETNPs in pH 1.2 for first two hours followed by pH 6.8 and E) <i>In vitro</i> release profiles of <i>Ficus relgiosa</i> L. extract, EUNPs and ETNPs in pH 7.4	123
6.15.	<i>In vitro</i> cytotoxicity assessments A) different treatment groups and B) different concentrations of TPP	126
6.16.	Confocal images for colocalization of nanoparticles with mitochondria	127
6.17.	Mitochondrial structural changes	128
6.18.	Mitochondrial membrane potential of different treatment groups	129
6.19.	Complex-I, II, IV and V activity	131
6.20.	Quantitation of calcium ion concentration in different treatment groups	132
6.21.	Western blotting expressions of cytochrome C, caspase-9 and caspase-3	133
6.22.	ROS generation in different groups	134
6.23.	Antioxidant levels of different treatment groups on A) superoxide dismutase, B) catalase and C) glutathione peroxidase	136
6.24.	Estimation of A) nitrite levels and B) malondialdehyde formation following different treatments	137
6.25.	Effect of different treatments on A) blood glucose, B) plasma insulin, C) serum glycated haemoglobin	138

6.26.	Histology examination of pancreas followed by different treatments A) control rat, B) diabetic, C) extract, D) EUNPs and E) ETNPs	139
6.27.	Histology examination of liver followed by different treatments A) control rat, B) diabetic, C) extract, D) EUNPs and E) ETNPs	140
6.28.	Histology examination of skeletal muscle followed by different treatments A) control rat, B) diabetic, C) extract, D) EUNPs and E) ETNPs	141
6.29.	Histology examination of adipose tissue followed by different treatments A) control rat, B) diabetic, C) extract, D) EUNPs and E) ETNPs	142
6.30.	Histology examination of kidney followed by different treatments A) control rat, B) diabetic, C) extract, D) EUNPs and E) ETNPs	143
6.31.	HPLC chromatograms of A – Blank plasma, B – <i>Ficus religiosa</i> Linn. extract, and C – Lupeol standard	144
6.32.	Plasma concentration vs. time curve of lupeol in <i>Ficus religiosa</i> Linn. extract suspension and SLN	146
6.33.	Proposed mechanism of action of ETNPs	147
7.1.	A) SEM image of LTNPs and B) in <i>vitro</i> release profiles of lupeol, LUNPs and LTNPs in pH 1.2 for first two hours followed by pH 6.8 and C)) in <i>vitro</i> release profiles of lupeol, LUNPs and LTNPs in pH 7.4.	151
7.2.	Interaction studies of lupeol, glycerylmonostearate and in SLN form A) FTIR, B) DSC and C) PXRD	153
7.3.	In vitro cytotoxicity assessments of different treatment groups	154
7.4.	Mitochondrial structural changes in A) normal, B) diabetic, and following the treatment with C) lupeol, D) LUNPs and E) LTNPs.	155
7.5.	Mitochondrial membrane potential of different treatment groups	156

7.6.	Complex-I, II, IV and V activity	157
7.7.	Quantitation of calcium ion concentration in different treatment groups	158
7.8.	Western blotting expressions of cytochrome C, caspase-9 and caspase-3	159
7.9.	ROS generation in different groups	160
7.10.	Antioxidant levels of different treatment groups on A) superoxide dismutase, B) catalase and C) glutathione peroxidase	161
7.11.	Estimation of A) nitrite levels and B) maldialdehyde formation following different treatments	162
7.12.	Effect of different treatments on A) blood glucose, B) plasma insulin, C) serum glycated haemoglobin	163
7.13.	Histology examination of different organs followed by different treatments A) control rat, B) diabetic, C) lupeol, D) LUNPs and E) LTNPs	164
7.14.	Plasma concentration vs. time curve of lupeol in <i>Ficus religiosa</i> Linn. extract suspension and SLN	165
8.1.	<i>In vitro</i> characterization of ETNPs and LTNPs A) SEM image of ETNPs, B) SEM image of LTNPs and C) <i>in vitro</i> drug release	169
8.2.	In vitro cytotoxicity assessments of all treatment groups	170
8.3.	Mitochondrial structural changes in A) normal, B) diabetic and different treatment groups C) extract, D) EUNPs, E) ETNPs, F) lupeol, G) LUNPs and H) LTNPs	172
8.4.	Effect of different treatments on mitochondrial membrane potential	173
8.5.	Effect of different treatments on complex-I, II, IV and V activities	175
8.6.	Effect of different treatments on calcium ion levels	176

8.7.	Western blotting expressions of cytochrome C, caspase-9 and caspase-3	177
8.8.	Effect of different treatments on ROS level	178
8.9.	Effect of different treatments on A: Superoxide dismutase levels, B: Catalase levels, C: Glutathione peroxidase levels	180
8.10.	Effect of different treatments on A: Nitrite levels and B: malondialdehyde levels	181
8.11.	Effect of different treatments on A) blood glucose, B) plasma insulin, C) serum glycated haemoglobin	183
8.12.	Histology examination of different organs followed by different treatments A) control rat, B) diabetic, C) ETNPs and D) LTNPs	184
8.13.	Plasma concentration vs. time curve of lupeol in different forms, plain lupeol, plain extract and their nano-forms; LSLN and ESLN	186