

LIST OF FIGURES

Figure No.	Caption	Page No.
Introduction		
Figure 1.	General structures of monoglycerides.	4
Figure 2.	General structure of diglycerides.	5
Figure 3.	General structure of saturated and unsaturated phospholipids with different head groups:(A) 1,2-dipalmitoyl-sn-glycero-3-phospho (1'-rac-glycerol) (sodium salt) [DoTAP]; (B) 1,2-dipalmitoyl-sn-glycero-3-phospho-(1'-rac-glycerol) (sodium salt) [DPPC] and (C) 1,2-dipalmitoyl-sn-glycero-3-phospho-(1'-rac-glycerol) (sodium salt) [DPPG].	7
Figure 4.	General structure of glycolipid derivatives.	7
Figure 5.	General structure of cholesterol.	8
Figure 6.	Schematic diagram of a normal alveolus compared with an alveolus suffering from acute lung injury (ALI). During ALI, serum proteins become inserted and DPPC is replaced by less surface-active phospholipids.	10
Figure 7.	Pi diagram describing the composition of PS.	11
Figure 8.	Structure of lung surfactant proteins.	13
Figure 9.	Schematic diagram of a normal alveolus compared with an alveolus suffering from ALI acute lung injury (ALI). During ALI, serum proteins become inserted and DPPC is replaced by less surface-active phospholipids.	15
Figure 10.	Schematic of historical development of colloidal carrier system.	25
Figure 11.	Schematic diagram of three different types of drug delivery systems based on liposomes: (A) Charge and polymer stabilized liposome, (B) Targeted liposome, and (C) Theranostic liposome.	27

Figure No.	Caption	Page No.
Figure 12.	Schematic diagram of microemulsion and nanoemulsion.	28
Figure 13.	Schematic representation of a perfect lipid crystal in SLN (A) and a crystal lattice with many imperfections in NLC (B).	30
Figure 14.	Models of incorporation of active compounds into SLN (a) homogeneous matrix; (b) drug-enriched shell model; (c) drug-enriched core model.	32
Figure 15.	Drug incorporation models of nanostructured lipid carriers (A) the amorphous type, (B) imperfect type, and (C) multiple type.	34
Figure 16.	Particle size distributions of NLC (PEG – 25 – SA NLC).	39
Figure 17.	Zeta potential distributions of NLC (PEG – 25 – SA NLC).	40
Figure 18.	TEM (A) and SEM (B) images of PEG - 100 – SA coated SLNs.	41
Figure 19.	AFM image of γ -oryzanol loaded lipid nanoparticles (LN) at 0% liquid lipid (A), 5% liquid lipid (B) and 10% liquid lipid (C): 3D images (1), topographic images (2) and phase images (3). All images were scanned over a $1 \times 1 \mu\text{m}^2$ area.	42
Figure 20.	DSC image of (A) BCA–PEG–NLC and its ingredients, (B) physical mixture, (C) PEG–SA, (D) LEC, (E) BCA, and (F) GMS.	43
Figure 21.	<i>In vitro</i> drug release profile of different NLCs: (A) Tf _{10k} -PTX-DNA-NLC, (B) Tf _{5k} -PTX-DNA-NLC, (C) PTX-DNA-NLC, and (D) Taxol [®] .	47

Chapter I

Figure No.	Caption	Page No.
Figure 1.	MALDI-TOF mass spectrometry spectra of (a) BLES and (b) serum lipids (c) LDL and (d) table of serum lipids by Iatroscan. For the BLES sample, DPPC was the most abundant. As for the serum lipid extract, cholesterol was the most abundant. Several other phospholipid classes are detected in the 700+ m/z range.	88
Figure 2.	Adsorption isotherms (γ vs. time) of BLES dispersions in the presence of 10 and 20 wt% (a) LDL, (b) cholesterol, and (c) serum at 25 ± 1 °C. Each plot is an average of 3 independent experimental sets. Standard deviation is shown by the error bars of n=3 experiments.	65
Figure 3.	Surface tension - area isotherms of 5 cycles of dynamic compression - expansion at a rate of 2 mm ² /sec for (a) pure BLES, (b) 10 wt% serum, (c) 20 wt% serum, (d) 10 wt% cholesterol, (e) 20 wt% cholesterol, (f) 10 wt% LDL and (g) 20 wt% LDL adsorbed films. Each experiment was conducted in triplicate and the best representative graph from a single experiment is shown for clarity. All isotherms were plotted as percentage of film area change versus surface tension (γ).	68
Figure 4.	Effects of serum, cholesterol and LDL on compressibility of BLES films based on mean % pool area compression required for a surface tension drop of 15 mN/m (C_{15} values). Serum and LDL had to be compressed two fold more in area than the ones of pure BLES.	69
Figure 5.	Height differences (section analysis) between the gel and fluid phases of the AFM image of BLES + additive films at γ 52, 42, and 32 mN/m for (a) pure BLES and BLES with (b) 10wt% serum, (c) 20wt% serum, (d) 10wt% cholesterol, (e) 20wt% cholesterol, (f) 10wt% LDL and (g) 20wt% LDL. Both three dimensional (3D) and sectional analysis.	75

Figure No.	Caption	Page No.
Figure 6.	Representative contact mode AFM images of Langmuir - Blodgett film deposited (imaged in air in contact mode) at equivalent γ of 52 mN/m for (a) Pure BLES, and BLES in presence of (b) 10 wt% serum, (c) 20 wt% serum, (d) 10 wt% cholesterol, (e) 20 wt% cholesterol, (f) 10 wt% LDL, and (g) 20 wt% LDL. Image field sizes shown were taken at $10\ \mu\text{m}\times 10\ \mu\text{m}$ (X-Y plane) and height differences (Z plane) are shown by the 10.0 nm bar, indicated in (a). The condensed domains are about 1.2 nm above the surrounding fluid regions. The bright regions represent condensed gel domains and other structures which are 0.5 - 1 nm higher than the surrounding phase. Each frame represents a scan area of $10\ \mu\text{m}\times 10\ \mu\text{m}$.	76
Figure 7a.	Representative AFM images of films of BLES deposits taken at $\gamma = 52, 42, 32$ mN/m respectively in each vertical panel. Each frame represents a scan area of $10\ \mu\text{m} \times 10\ \mu\text{m}$.	77
Figure 8b-d.	Representative AFM images of films of BLES + 10% serum (b), 10% Cholesterol (c) 10 wt% LDL (d). Deposits were taken at $\gamma = 52, 42, 32$ mN/m respectively in each vertical panel. Upon addition of materials to BLES films, the gel domain (bright circular areas) formation is altered. Each frame represents a scan area of $10\ \mu\text{m}\times 10\ \mu\text{m}$.	77
Figure 9e-g.	Representative AFM images of films of BLES with 20 wt% serum (e), 20 wt% cholesterol (f) and 20 wt% LDL (g). Deposits were taken at $\gamma 52, 42, 32$ mN/m respectively in each vertical panels. Upon addition of materials to BLES films, the gel domain (bright circular areas) formation is altered.	78

Figure No.	Caption	Page No.
Figure 10.	Raman spectra of the 2800-3000 cm^{-1} range for (a) BLES, (b) BLES + 10wt% serum, (c) BLES +10wt% cholesterol, and (d) BLES + 10wt% LDL. Each graph is a representative of three different experimental trials. Similar trends were shown for BLES and BLES + 20wt% serum, 20wt% cholesterol, and 20wt% LDL samples. Temperature range indicated from 10 $^{\circ}\text{C}$ - 40 $^{\circ}\text{C}$.	82
Figure 11.	Vibrational wave number shift for BLES compared to (a) BLES + serum, (b) BLES + cholesterol and (c) BLES + LDL. The temperature range was from 10 – 40 $^{\circ}\text{C}$ for all graphs. Each graph is the mean of wave number shifts from three independent experiments and the error bars represent the standard deviation.	84
Chapter II		
Figure 1.	Hydrodynamic diameter (d_h) – time profile of NLCs (Span 65 + SLC + SA , 2:2:1 M/M/M) dispersed in Tweens in presence of varying concentration of drugs. Panel A: LIDO loaded NLC in Tween 40; panel B: PRO.HCl loaded NLC in Tween 40; panel C: LIDO loaded NLC in Tween 60 and panel D: PRO.HCl loaded NLC in Tween 60. 5 mM NLC was dispersed in 10 mM Tweens in each case. Drug concentration (mM) : O, 0; Δ , 0.5; \square , 1; \bullet , 2 and \blacktriangle , 2.5 . Temp. 25 $^{\circ}\text{C}$.	97
Figure 2.	Variation in the polydispersity index (PDI) of NLCs (Span 65 + SLC + SA, 2:2:1 M/M/M) with time in presence of varying concentration of drugs. Panel A: LIDO loaded NLC in Tween 40; panel B: PRO.HCl loaded NLC in Tween 40; panel C: LIDO loaded NLC in Tween 60 and panel D: PRO.HCl loaded NLC in Tween 60. 5 mM NLC was dispersed in 10 mM Tween in each case. Drug concentration (mM) : O, 0; Δ , 0.5; \square , 1; \bullet , 2 and \blacktriangle , 2.5 . Temp. 25 $^{\circ}\text{C}$.	98

Figure No.	Caption	Page No.
Figure 3.	Influence of LIDO and PRO.HCl on the (Z.P.) of NLCs (Span 65 + SLC + SA , 2:2:1 M/M/M) dispersed in Tweens. Systems: O, LIDO -Tween 40; Δ , LIDO -Tween 60; \square , PRO.HCl-Tween 40 and ∇ , PRO. HCl -Tween 60. Temp. 25 °C. 5 mM lipid in the absence and presence of the drug was dispersed in 10 mM aqueous Tween solution.	99
Figure 4.	Representative TEM images of LIDO loaded NLC dispersion in Tween 40 (A) and PRO.HCl loaded NLC dispersion in Tween 40 (B).	99
Figure 5.	DSC heating (---) and cooling (—) thermogram of 2.5 mM LIDO loaded NLC (5 mM, Span 65+SLC+SA, 2:2:1 M/M/M) dispersed in Tween 60 (10mM). Scan rate: 2 °C/min.	100
Figure 6.	DSC cooling curves of LIDO (panel A) and PRO.HCl (panel B) loaded NLC dispersed in Tween 40; LIDO (panel C) and PRO.HCl (panel D) loaded NLC dispersed in Tween 60. 5 mM NLC with Span 65 + SLC + SA , 2:2:1 M/M/M , was dispersed in 10 mM aqueous Tween solution. Concentration of drug (mM): 1, 0; 2,0.2; 3,0.5; 4,1.0; 5,1.5; 6,2.0 and 7,2.5. Scan rate 2 °C/min.	101
Figure 7.	Effects of drugs on the temperature of maximum heat flow (T_m , panel A), width of half peak height ($\Delta T_{1/2}$, panel B), change in enthalpy (ΔH , panel C) and heat capacity (ΔC_p , panel D) of NLCs (Span65 + SLC + SA , 2:2:1 M/M/M). 5 mM lipid was dispersed in 10 mM aqueous Tween solution. System O; LIDO loaded in Tween 40: Δ ; LIDO loaded Tween 60: \square ; PRO.HCl loaded Tween 40: \square and \bullet ; PRO.HCl loaded Tween 60.	101
Figure 8.	Effects of drug concentration on the degree of crystallinity of NLC (5 mM, Span 65+SLC+SA, 2:2:1 M/M/M). Systems: Δ , LIDO -Tween 40; ∇ , LIDO -Tween 60; O, PRO.HCl-Tween 40 and \square , PRO.HCl-Tween 60.	104

Figure No.	Caption	Page No.
Figure 9.	UV-visible absorption spectra of 1 mM LIDO (panel A) and PRO.HCl (panel B) loaded in NLC as well as solvents of different polarity; at 25 °C. Systems : —, n-hexane; —, acetonitrile; —, chloroform; —, ethanol; —, methanol and —, drug loaded NLC respectively.	105
Figure 10.	Dependence of absorption maxima (λ_{max}) of LIDO (panel A) and PRO.HCl (panel B) on the E_T30 scale of medium at 25 °C.	105
Figure 11.	Dependence of entrapment efficiency (E.E.) (panel A) and loading content (L.C.) (panel B) of NLC (5mM, Span 65 + SLC + SA, 2:2:1 M/M/M) on the concentration of drugs. LIDO loaded NLCs dispersed in: O, Tween 40; Δ , Tween 60; and PRO.HCl dispersed in \square , Tween 40 and ∇ , Tween 60. Each value represents the mean \pm (S.D.) (n=3). Temp. 4 °C.	106
Figure 12.	<i>In vitro</i> cumulative LIDO release from NLC (5mM, Span 65 + SLC + SA, 2:2:1 M/M/M). System: LIDO loaded NLC dispersion in Tween 40 (O), Tween 60 (\square) and free LIDO in Tween 60(Δ). Each point represents the mean \pm (S.D.) (n=3). Drug concentration was kept constant at 0.5 mM in each case. Temp. 25 °C.	107
Figure 13.	<i>In vitro</i> cumulative PRO.HCl release from NLC (5mM, Span 65 + SLC + SA , 2:2:1 M/M/M). Systems: PRO.HCl loaded dispersion in Tween 40 (O), Tween 60 (\square) and free PRO.HCL in Tween 60(Δ). Each point represents the mean \pm (S.D.) (n=3). Drug concentration was kept constant at 0.5 mM in each case. Temp. 25 °C.	107

Chapter III

Figure No.	Caption	Page No.
Figure 1.	Surface pressure (π) – area (A) isotherm of (—), tribehenin; (—), trierucin; (—), HSPC; (—), behenic acid; (—), oleic acid and (—), ursolic acid (panel A); TB+HSPC+BA (panel B); and TE+HSPC+OA (panel C). Panel C describes the π -A isotherm of mixed monolayer in the absence and presence of ursolic acid respectively using water as subphase. Mole % of ursolic acid with respect to lipid mixture: (—), 0; (—), 2.5; (—), 5; (—), 10; (—), 30; (—), 50; and (—), 70. Temperature 25 °C.	117
Figure 2.	Surface pressure (π) – area (A) isotherm of mixed lipid and ursolic acid – lipid mixture: TB+HSPC+OA (panel A); and TE+HSPC+BA (panel B) describes the π -A isotherm of mixed monolayer in the absence and presence of ursolic acid respectively using water as subphase. Mole% of ursolic acid with respect to lipid mixture: (—), 0; (—), 2.5; (—), 5; (—), 10; (—), 30; (—), 50; and (—), 70. Temperature 25 °C.	118
Figure 3.	Variation in elasticity moduli (C_s^{-1}) with % of compressed area for mixed monolayer systems: TB+HSPC+BA (panel A); TB+HSPC+OA (panel B); TE+HSPC+BA (Panel C); and TE+HSPC+OA (panel D), lipid mixture, component 1, (2:2:1, M/M/M) and ursolic acid, component 2. Mole % of ursolic acid with respect to lipid mixture: (—) 0; (—), 2.5; (—), 5; (—), 10; (—), 30; (—), 50; and (—), 70. Temperature: 25 °C.	119
Figure 4.	Dependence of excess molecular area (A_{ex}) on the relative proportion of ursolic acid in the mixed monolayer systems: TB+HSPC+BA (panel A); TB+HSPC+OA (panel B); TE+HSPC+BA (Panel C); and TE+HSPC+OA (panel D), lipid mixture, component 1, (2:2:1, M/M/M) and ursolic acid, component 2, (Mole % of ursolic acid with respect to lipid mixture): 0, 2.5, 5, 10, 30, 50, 70 and 100), at different surface pressure (mNm^{-1}): \circ , 5; \triangle , 10; ∇ 15; \diamond 20; \triangleleft , 25 and \triangleright , 30. Temperature: 25 °C.	120

Figure No.	Caption	Page No.
Figure 5.	Dependence of change in excess free energy (ΔG°_{ex}) on the relative proportion of ursolic acid in the mixed monolayer systems: TB+HSPC+BA (panel A); TB+HSPC+OA (panel B); TE+HSPC+BA (Panel C); and TE+HSPC+OA (panel D), lipid mixture: component 1, (2:2:1, M/M/M) and ursolic acid: component 2. Mole % of ursolic acid with respect to lipid mixture: 0, 2.5, 5, 10, 30, 50, 70 and 100) at different surface pressure (mNm^{-1}): \circ , 5; \triangle , 10; ∇ 15; \diamond 20; \triangleleft , 25 and \triangleright , 30. Temperature: 25 °C.	121
Figure 6.	Changes in the free energy of mixing (ΔG°_{mix}) on the relative proportion of ursolic acid in the mixed monolayer systems: TB+HSPC+BA (panel A); TB+HSPC+OA (panel B); TE+HSPC+BA (Panel C); and TE+HSPC+OA (panel D), lipid mixture, component 1, (2:2:1, M/M/M) and ursolic acid, component 2. Mole% of ursolic acid with respect to lipid mixture: 0, 2.5, 5, 10, 30, 50, 70 and 100 at different surface pressure (mNm^{-1}): \circ , 5; \triangle , 10; ∇ 15; \diamond 20; \triangleleft , 25 and \triangleright , 30. Temp. 25 °C.	122
Figure 7.	Surface potential of ursolic acid and pure lipid components; systems mentioned inside the figure. Pure water was used as subphase. Temp. 25 °C.	122
Figure 8.	Variation in the surface potential of mixed lipid and ursolic acid – lipid mixture: TB+HSPC+BA (panel A); TB+HSPC+OA (panel B); TE+HSPC+BA (Panel C); and TE+HSPC+OA (panel D) describes the surface potential of mixed monolayer in the absence and presence of ursolic acid respectively using water as subphase. Mole% of ursolic acid with respect to lipid mixture: (—) 0; (—), 2.5; (—), 5; (—), 10; (—), 30; (—), 50; and (—), 70. Temp. 25 °C.	123

Figure No.	Caption	Page No.
Figure 9.	Variation in the hydrodynamic diameter (d_h)–time profile of NLCs Panel A: TB+HSPC+BA; panel B: TB+HSPC+OA; panel C: TE+HSPC+BA and panel D: TE+HSPC+OA. 5 mM NLC was dispersed in 10 mM Tween 80 in each case. UA concentration (mM): (\circ), 0; (\square), 0.125; (\triangle), 0.25 and (∇), 0.5. Temp. 25 °C.	125
Figure 10.	Variation in the polydispersity index (PDI)–time profile of NLCs Panel A: TB+HSPC+BA; panel B: TB+HSPC+OA; panel C: TE+HSPC+BA and panel D: TE+HSPC+OA. 5 mM NLC was dispersed in 10 mM Tween 80 in each case. UA concentration (mM): (\circ), 0; (\square), 0.125; (\triangle), 0.25 and (∇), 0.5. Temp. 25 °C.	125
Figure 11.	Variation in the zeta potential (Z.P.) – time profile of NLCs Panel A: TB+HSPC+BA; panel B: TB+HSPC+OA; panel C: TE+HSPC+BA and panel D: TE+HSPC+OA. 5 mM NLC was dispersed in 10 mM Tween 80 in each case. UA concentration (mM): (\circ), 0; (\square), 0.125; (\triangle), 0.25 and (∇), 0.5. Temp. 25 °C.	126
Figure 12.	TEM images of NLC formulations. Composition : (A) TB+HSPC+BA; (B): TB+HSPC+OA; (C): TE+HSPC+BA and (D): TE+HSPC+OA. I and II represents the blank NLCs as well as ursolic acid loaded NLCs respectively.	126
Figure 13.	Representative AFM images of NLC formulation. Composition : (I) TB+HSPC+BA; and (II) TE+HSPC+OA. 2D image (A, B); 3D image (C) and section analysis (D).	127
Figure 14.	DSC heating thermograms of UA and the pure lipid components; systems mentioned inside the figure. Scan rate: 2.5 °C min ⁻¹ .	128
Figure 15.	DSC heating thermograms of physical mixture of lipids and UA+ lipids; systems mentioned inside the figure. Scan rate: 2.5 °C min ⁻¹ .	128

Figure No.	Caption	Page No.
Figure 16.	Representative DSC thermogram of (TB+HSPC+OA, 2:2:1 M/M/M) comprising UA-NLC and corresponded UA/lipid mixtures presented inset in the figure. Scan rate: 2.5 °C min ⁻¹ .	129
Figure 17.	DSC heating curves of ursolic acid loaded NLCs. Composition of the NLCs: A: TB+ HSPC+BA; B: TB+HSPC+OA; C: TE+HSPC+BA and D: TE+HSPC+OA; [Ursolic acid]/mM: 1, 0; 2, 0.125; 3, 0.25; and 4, 0.5. Scan rate 2.5 °C/min.	130
Figure 18.	<i>In vitro</i> cumulative release of ursolic acid from NLCs. Composition of NLCs: TB+HSPC+BA (∇); TB+HSPC+OA (□); TE+HSPC+BA (Δ); TE+HSPC+OA (○) and free UA (◇) in PBS pH 7.4 / Tween 80 1% v/v, at 37±0.1 °C. Total release from the NLCs: (A) TE+HSPC+OA; (B) TE+HSPC+BA; (C) TB+HSPC+OA and (D) TB+HSPC+ BA. Error bars represent standard deviation (SD) of 3 different release experiments (n = 3).	132
Figure 19.	<i>In vitro</i> cytotoxicity activity of free ursolic acid (—) and ursolic acid loaded with different NLCs: TB+HSPC+BA (—); TB+HSPC+OA (—); TE+HSPC+BA (—) and TE+HSPC+OA (—) on the viability of B16 and K562 cell. Cell was grown and treated for 24h, 48h and 72h. Experiments were performed in triplicate, with the results showing the mean and standard deviation of the triplicate of each group. The experiments were repeated three times with similar results.	133

Chapter IV

Figure No.	Caption	Page No.
Figure 1.	Variation in the hydrodynamic diameter (d_h)–time profile of NLCs (black): TS+OA; (red): TS+PEG - 25 - SA+OA; (blue): TS+PEG - 55 - SA+OA; (dark yellow): TS+OA+OG; (wine): TS+PEG - 25 - SA+OA+OG and (olive): TS+PEG - 55 - SA+OA+OG. 100mg NLC was dispersed in 200mg Tween 80 in each case. OG concentration was (5mg). Temp. 25 °C.	143
Figure 2.	Variation in the polydispersity index (PDI)–time profile of NLCs (black): TS+OA; (red): TS+PEG - 25 - SA+OA; (blue): TS+PEG - 55 - SA+OA; (dark yellow): TS+OA+OG; (wine): TS+PEG - 25 - SA+OA+OG and (olive): TS+PEG - 55 - SA+OA+OG. 100mg NLC was dispersed in 200mg Tween 80 in each case. OG concentration was (5mg). Temp. 25 °C.	143
Figure 3.	Variation in the zeta potential (Z.P.)–time profile of NLCs (black): TS+OA; (red): TS+PEG - 25 - SA+OA; (blue): TS+PEG - 55 - SA+OA; (dark yellow): TS+OA+OG; (wine): TS+PEG - 25 - SA+OA+OG and (olive): TS+PEG - 55 - SA+OA+OG. 100mg NLC was dispersed in 200mg Tween 80 in each case. OG concentration was (5mg). Temp. 25 °C.	144
Figure 4.	TEM and SEM images of (A and G) TS+OA, (B and H) TS+PEG-25-SA+OA and (C, I) TS+PEG-55-SA+OA NLC respectively; (D, E and F) are the corresponded orcinol glucoside loaded formulations.	145
Figure 5.	AFM images of (I) TS + OA (II) TS + PEG – 25 – SA + OA and (III) TS + PEG – 55 – SA + OA NLC formulations. (A) Two-dimensional images, (B) Three-dimensional images and (C and D) section analysis.	146
Figure 6.	AFM images of (I) TS + OA + OG (II) TS + PEG – 25 – SA + OA + OG and (III) TS + PEG – 55 – SA + OA + OG NLC formulations. (A) Two-dimensional images, (B) Three-dimensional images and (C and D) section analysis.	147

Figure No.	Caption	Page No.
Figure 7.	DSC heating thermograms of orcinol glucoside and the pure lipid components; systems mentioned inside the figure. Scan rate: 2.5 °C min ⁻¹ .	148
Figure 8.	Representative DSC thermograms of (red) TS+OA+OG and (black) TS+PEG-25-SA+OA+OG, 80: 20:5 and 40:40:20:5 mg/mg/mg/mg comprising physical mixture. Scan rate: 2.5 °C min ⁻¹ .	148
Figure 9.	DSC endothermic (panel A) and exothermic curves (panel B) of bare as well as orcinol glucoside-loaded non-PEGylated and PEGylated NLCs: (red) TS+OA, (blue) TS+OA+OG, (pink) TS+PEG-25-SA+OA, (dark yellow) TS+PEG-25-SA+OA+OG, (brown) TS+PEG-55-SA+OA and (green) TS+PEG-55-SA+OA+OG. Panel C and panel D are the physical mixture of corresponded NLC. An orcinol glucoside concentration was 5 mg. The scan rate was 2.5 °C/min.	149
Figure 10.	<i>In vitro</i> cumulative release of orcinol glucoside from NLCs. Composition of NLCs: (black squares) free OG, (red circles) TS + OA + OG, (blue up triangles) TS + PEG – 25 – SA + OA + OG and (green down triangles) TS + PEG – 55 – SA + OA + OG in PBS panel A (pH 7.4), panel B (pH 6.8) and panel C (pH 1.2) at 37 ± 0.1 °C. Error bars represent the standard deviation (SD) of three different release experiments.	153
Figure 11.	<i>In vitro</i> cytotoxicity activity of free orcinol glucoside on the viability of Hepatocellular carcinoma cell line (HepG2), hepatocyte-derived carcinoma cell line (Huh-7), colorectal carcinoma cell line (HCT-116) and gastric adenocarcinoma (AGS) cells. Cell was grown and treated for 24h. Experiments were performed in triplicate, with the results showing the mean and standard deviation of the triplicate of each group.	155

Figure No.	Caption	Page No.
Figure 12.	<i>In vitro</i> cytotoxicity activity of representative PEG coated bare NLC (—) and orcinol glucoside loaded with different NLCs: TS+OA (—); TS+PEG-25-SA+OA (—) and TS+PEG-55-SA+OA (—) on the viability of HepG2 and HUH7 cell. Cell was grown and treated for 24h, 48h and 72h. Experiments were performed in triplicate, with the results showing the mean and standard deviation of the triplicate of each group.	156
Figure 13.	<i>In vitro</i> cytotoxicity activity of representative PEG coated bare NLC (—) and orcinol glucoside loaded with different NLCs: TS+OA (—); TS+PEG-25-SA+OA (—) and TS+PEG-55-SA+OA (—) on the viability of HCT116 and AGS cell. Cell was grown and treated for 24h, 48h and 72h. Experiments were performed in triplicate, with the results showing the mean and standard deviation of the triplicate of each group.	157
