List of Figures

Figure 1.1: The data showing, worldwide prevalence of diabetes 4
Figure 1.2: Anatomy of Pancreas 6
Figure 1.3: Pancreas showing pancreatic & bile duct opening in duodenum 7
Figure 1.4: Islets of Langerhan 7
Figure 1.5 (a) Proinsulin and matured insulin, (b) Release of insulin in plasma 8
Figure 1.6: Metabolic effects of Insulin 9
Figure 1.7: Insulin signaling cascade 12
Figure 1.8: G-protein coupled signaling 13
Figure 1.9: Signal transduction of glucagon 14
Figure 1.10: Epinephrine, nor epinephrine, share the same β adrenergic receptor 15

Figure 1.11: Main symptoms of Diabetes 15
Figure 1.12: Islets of Langerhan [Florescent micrograph] 19
Figure 1.13: Type 1 Diabetes flow chart of events 19
Figure 1.14: Blood vessel carrying glucose in normal and diabetic patient 21
Figure 1.15: Type 2 Diabetes flow chart of events 22
Figure 1.16: Life style, the major cause of Type 2 Diabetes 23
Figure 1.17: Fluctuating blood sugar levels in correlation to insulin levels 26
Figure 1.18: Common complications associated with Diabetes 28
Figure 1.19: Clinical Manifestations of Autonomic Neuropathy (AN) 31
Figure 1.20: Normal and glaucoma vision 34
Figure 1.21: Normal and cataract vision
Figure 1.22: Manifestation of Diabetic retinopathy
Figure 1.23: Anatomy of the eye
Figure 1.24: Normal and retinopathy vision
Figure 1.25: The poly Pathway and Diabetic retinopathy
Figure 1.26: a. Methylglyoxal as a marker for AGEs. b. AGEs in old and young tissues
Figure 1.27: Schematic diagram of formation of AGE
Figure 1.28: Wolff pathway showing formation of Amadori products
Figure 1.29: Activation of protein kinase C (PKC), initiating a cascade of intracellular stress responses
Figure 1.30: Enzymes that mediate hexosamine pathway
Figure 1.31: Mechanism of ROS production and glucose metabolism
Figure 1.32: Role of ROS and RNS in Diabetic retinopathy
Figure 3.1: Reaction between MDA and thiobarbituric acid TBA
Figure 3.2: DNA damage as indicated by SCGE or Comet assay
Figure 3.3: Flow chart of SCGE or Comet assay
Figure 3.4: Normal angiogenesis as against decreased angiogenesis
Figure 3.5: Microscopic observation a. 40x b. oil immersion
Figure 3.6: Opacitometer
Figure 3.7 A. Cornea holder and its dimensions, B. Ray diagram showing the internal arrangement of cornea
Figure 3.8: Ray diagram of Muir opacitometer
Figure 3.9: Opacitometer, Assembly
Figure 4.1: Polyphenol content of medicinal plants
Figure 4.2: Proanthocyanidins content of medicinal plants
Figure 4.3: Flavonoid content of medicinal plants

Figure 4.4: HPTLC plate scanned at 254nm

Figure 4.5: HPTLC plate scanned in visible light

Figure 4.6: HPTLC plate scanned at 366nm

Figure 4.7: Conc. of flavonoids (µg) as detected in M. charantia

Figure 4.8: Conc. of flavonoids (µg) as detected in T. cordifolia

Figure 4.9: Conc. of flavonoids (µg) detected in W. somnifera

Figure 4.10: Conc. of flavonoids (µg) detected in S. cumini

Figure 4.11: Conc. of flavonoids (µg) detected in A. catechu

Figure 4.12: Conc. of flavonoids (µg) detected in F. benghalensis

Figure 4.13: Conc. of flavonoids (µg) detected in F. glomerata

Figure 4.14: Conc. of flavonoids (µg) detected E. officinalis

Figure 4.15: Conc. of flavonoids (µg) detected T. foenum

Figure 4.16: Conc. of flavonoids (µg) detected T. arjun

Figure 4.17: TPTZ – Fe^{3+} reducing ability of antioxidants

Figure 4.18: Radical scavenging by ABTS and DPPH

Figure 4.19: Thiobarbituric acid reactive substances (MDA) in RBC ghosts

Figure 4.20: Cytotoxicity expressed in terms of cell viability.

Figure 4.21: Absorption of Proanthocyanidins, Phenolics and flavonoids by goat intestinal model

Figure 4.22: a – f Methylene blue stained comets

Figure 4.23: Angiogenic effect of medicinal plants

Figure 4.24: Pericytes as seen under high power 40 X and oil immersion
Figure 4.25: GCOP assay, *invitro* score

Figure 5.1: Comparing of secondary metabolites and their antioxidant power and radical Scavenging ability