FIGURE 1 (A) Uninjured MHS carotid

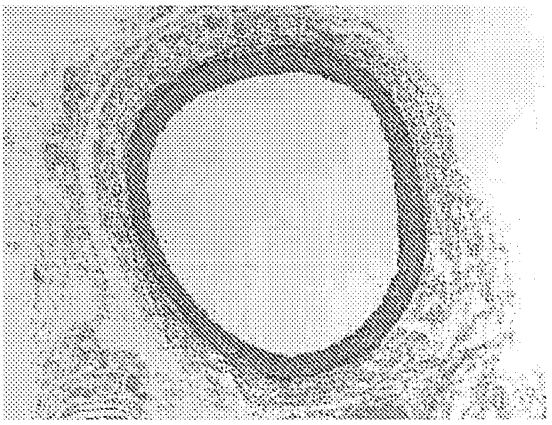


FIGURE 1 (B) Injured MHS carotid harvested 30 days after surgery: vehicle (Methocel 0.5%) oral treatment

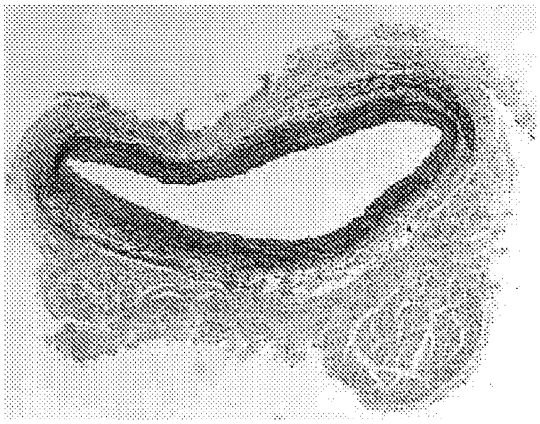


FIGURE 1 (C) Injured MHS carotid harvested 30 days after surgery: Rostafuroxin oral treatment (100 µg/kg)



FIGURE 2 (A) Uninjured MNS carotid

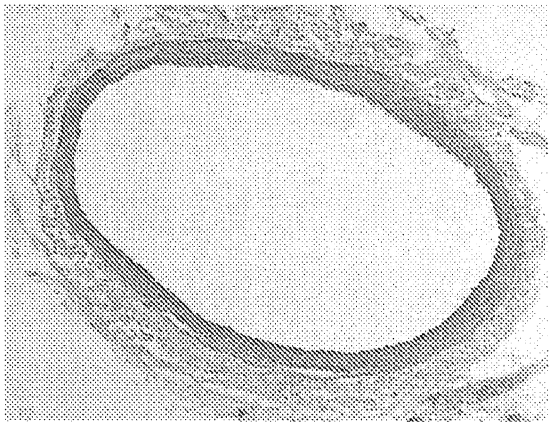


FIGURE 2 (B) Injured MNS carotid harvested 30 days after surgery: vehicle (Methocel 0.5%) oral treatment

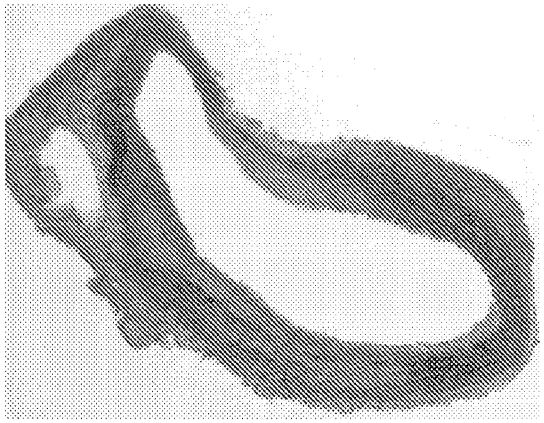
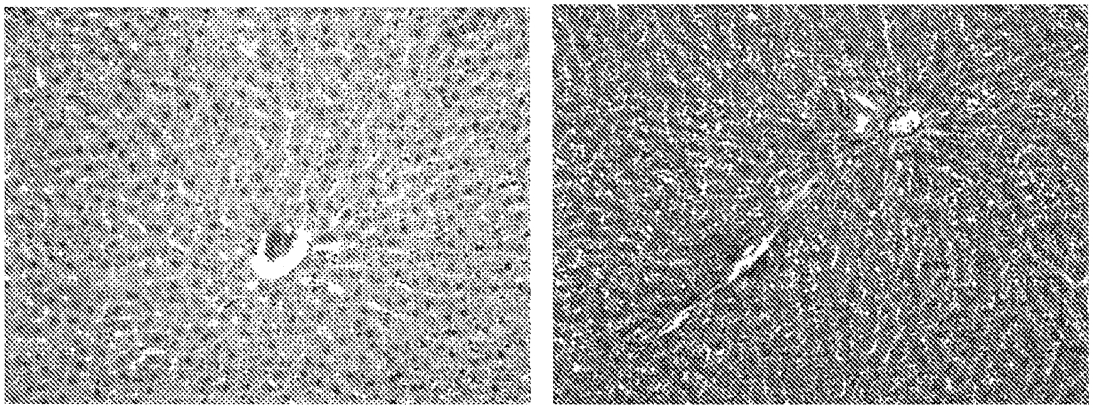


FIGURE 2 (C) Injured MNS carotid harvested 30 days after surgery: Rostafuroxin oral treatment (100 µg/kg)



Figure 3 shows normal staining patterns of the liver in control rat (Ctr. group)



H&E; final magnification 200X; Trichrome stain

Figure 4 shows that CCl4 liver injury induces remarkable hepatocellular damage which in turn stimulates the onset of a complex wound healing process, leading to hepatic fibrosis (Ctr. LF group)

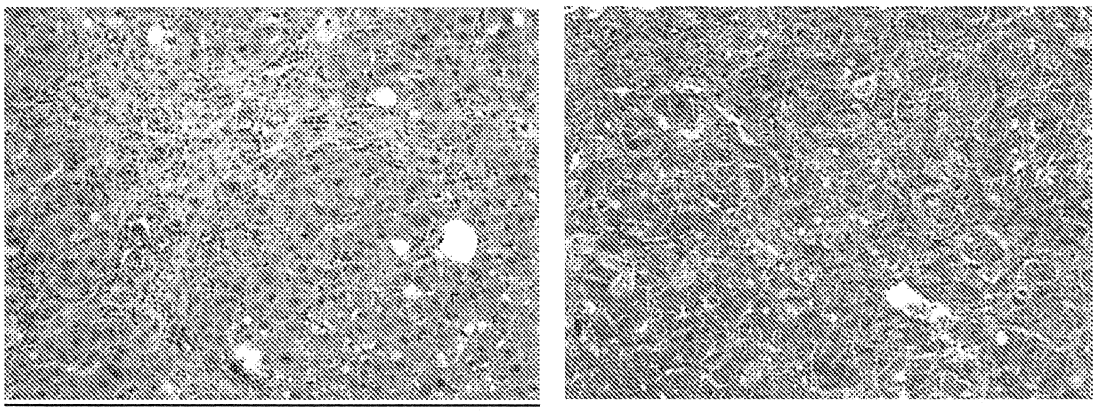


Figure 5 shows that in Rostafuroxin treated animals (RLF group), less extended tissue damage is associated to a minor degree of liver fibrosis.

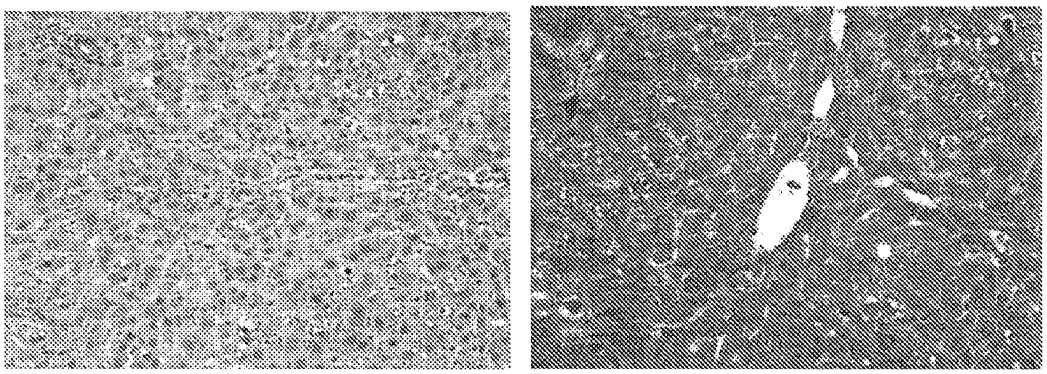


Figure 6 shows normal staining pattern of the liver in control rat (Ctr. group): score 0

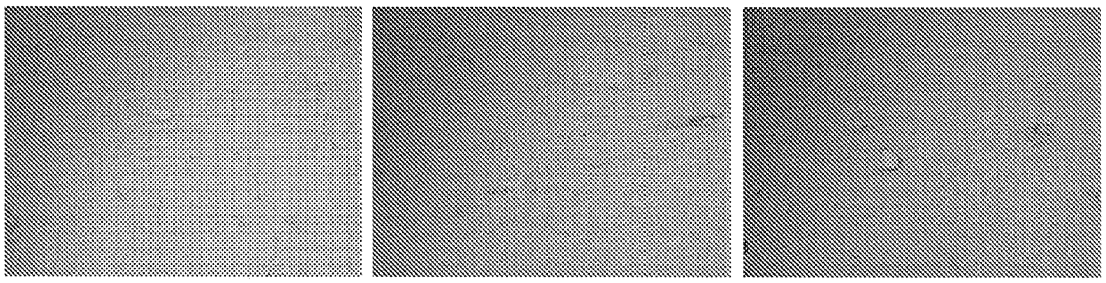


Figure 7 shows the histological appearance of liver from two CCl4 treated rats (Crt. LF group) with fibrotic lesions of moderate degree: score 2

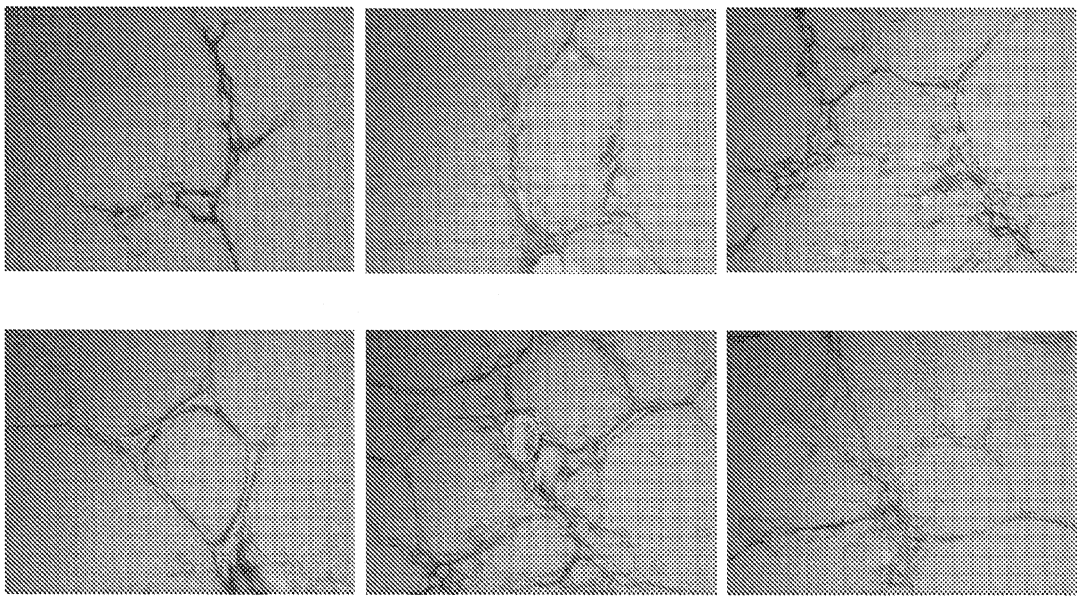


Figure 8 shows the histological appearance of liver from two CCl4 rats treated with Rostafuroxin at 1mg/kg os (RLF group) with fibrotic lesions of mild degree: score 1

