CLAIMS

1. Use of a compound selected from the group consisting of Nitazoxanide (NTZ), Tizoxanide (TZ) and Tizoxanide glucuronide (TZG), and a pharmaceutically acceptable salt of NTZ, TZ or TZG, in the manufacture of a medicament for the treatment of a cholestatic disorder selected from the group consisting of: primary biliary cholangitis (PBC), primary sclerosing cholangitis (PSC), Intrahepatic Cholestasis of Pregnancy, Progressive Familial Intrahepatic Cholestasis, Biliary atresia, Cholelithiasis, Cholangitis associated with Langerhans cell histiocytosis, Alagille syndrome, Nonsyndromic ductal paucity, Drug-induced cholestasis, and Total parenteral nutrition-associated cholestasis.

2. The use according to claim 1, wherein said compound is selected in the group consisting of NTZ and TZ, and a pharmaceutically acceptable salt of NTZ or TZ.

3. The use according to claim 1 or 2, wherein said compound is comprised in a pharmaceutical composition.

4. The use according to claim 1, wherein the cholestatic disorder is PBC.

5. The use according to any one of claims 1 to 4, wherein said compound is to be administered in combination with at least one therapeutically active agent with known antifibrotic activity selected from pirfenidone or receptor tyrosine kinase inhibitors (RTKIs) such as Nintedanib, Sorafenib and other RTKIs, or angiotensin II (AT1) receptor blockers, or CTGF inhibitor, or any antifibrotic compound susceptible to interfere with the TGFβ- and BMP-activated pathways including activators of the latent TGFβ complex such as MMP2, MMP9, THBS1 or cell-surface integrins, TGFβ receptors type I (TGFBRI) or type II (TGFBRII) and their ligands such as TGFβ, Activin, inhibin, Nodal, anti-Müllerian hormone, GDFs or BMPs, auxiliary co-receptors (also known as type III receptors), or components of the SMAD-dependent canonical pathway including regulatory or inhibitory SMAD proteins, or members of the SMAD-independent or non-canonical pathways including various branches of MAPK signaling, TAK1, Rho-like GTPase signaling pathways, phosphatidylinositol-3 kinase/AKT pathways, TGFβ-induced EMT process, or canonical and non-canonical Hedgehog signaling pathways including Hh ligands or target genes, or any members of the WNT, or Notch pathways which are susceptible to influence TGFβ signaling.

6. The use according to any one of claims 1 to 4, wherein the compound is to be administered in combination with at least one therapeutically active agent selected from JAK/STAT inhibitors, other anti-inflammatory and/or immunosuppressant agents.

7. The use according to claim 6, where the therapeutically active agent is selected from glucocorticoids, NSAIDS, cyclophosphamide, nitrosoureas, folic acid analogs, purine analogs, pyrimidine analogs, methotrexate, azathioprine, mercaptopurine, ciclosporin, myriocin, tacrolimus, sirolimus, mycophenolic acid derivatives, fingolimod and other sphingosine-1-phosphate receptor modulators, monoclonal and/or polyclonal antibodies against such targets as proinflammatory cytokines and proinflammatory cytokine receptors, T-cell receptor, integrins.