CLAIMS

1. Use of a combination product, comprising:

(i) cenicriviroc (CVC) or a pharmaceutically acceptable salt or solvate thereof; and

(ii) Elafibranor or a pharmaceutically acceptable salt thereof;

in the preparation of a medicament for the treatment of a diseases selected from liver fibrosis and cholestatic diseases.

2. Use according to claim 1, wherein the combination product is a composition comprising components (i) and (ii) and a pharmaceutically acceptable carrier.

3. Useaccording to claim 1 , wherein the combination product is a kit of parts comprising components (i) and (ii), for sequential, separate or simultaneous use.

4. Use according to any one of claims 1 to 3, wherein components (i) and (ii) are formulated in an injectable suspension, a gel, an oil, a pill, a tablet, a suppository, a powder, a capsule, an aerosol, an ointment, a cream, a patch, or means of galenic forms for a prolonged and/or slow release.

5. Use according to any one of claims 1 to 4, further comprising at least one therapeutically active agent with known antifibrotic activity selected from :

. pirfenidone;

. receptor tyrosine kinase inhibitors (RTKIs) such as Nintedanib or Sorafenib;

. angiotensin II (AT1) receptor blockers;

. CTGF inhibitor;

. 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); and

. 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.

6. Use according to any one of claims 1 to 5, further comprising at least one therapeutically active agent selected from JAK/STAT inhibitors, anti-inflammatory and/or immunosuppressant agents.

7. Use according to claim 6, wherein 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, sphingosine-1-phosphate receptor modulators, monoclonal and/or polyclonal antibodies against such targets as proinflammatory cytokines and proinflammatory cytokine receptors, T-cell receptor and integrins.

8. Use according to any one of claims 1 to 7, wherein the disease is selected in the group consisting of Primary Sclerosing Cholangitis (PSC), Primary Biliary Cholangitis (PBC), biliary atresia, familial intrahepatic cholestasis type 3 (PFIC3), liver fibrosis or cirrhosis of any origin, metabolic disease-induced liver fibrosis or cirrhosis, NAFLD-induced fibrosis or cirrhosis, NASH-induced fibrosis or cirrhosis, alcohol-induced liver fibrosis or cirrhosis, drug-induced liver fibrosis or cirrhosis, infectious agent-induced liver fibrosis or cirrhosis, parasite infection-induced liver fibrosis or cirrhosis, bacterial infection-induced liver fibrosis or cirrhosis, viral infection-induced fibrosis or cirrhosis, HBV-infection induced liver fibrosis or cirrhosis, HCV-infection induced liver fibrosis or cirrhosis, HIV-infection induced liver fibrosis or cirrhosis, dual HCV and HIV-infection induced liver fibrosis or cirrhosis, radiation- or chemotherapy-induced fibrosis or cirrhosis, biliary tract fibrosis, and liver fibrosis or cirrhosis due to any chronic cholestatic disease.

9. The use according to any one of claims 1 to 8, wherein the disease is PBC.