**Supplementary File 1**

**Supplementary Methods and Materials**

***Real-time qRT-PCR***

RNA was isolated by TRIzol reagent (Thermo Fischer Scientific, Shanghai, China) and a total of 1 μg RNA was reverse transcribed according to the manufacturer's instructions (Takara, Dalian, China). The mRNA levels and circRNA were monitored using a 7500 Real-time PCR System (Thermo Fischer Scientific). miRNA-specific stem-loop primer (RiboBio, Guangzhou, China) was used to synthesize the first-strand cDNA. A bulge-loop miRNA qRT-PCR starter kit (RiboBio) was used to detect miRNA levels. Total RNA from serum was isolated with a miRNeasy Serum/Plasma Kit (Qiagen, Hilden, Germany). cel-miR-39-3p was added as an external control for the miRNAs. The primer sequences are shown in Supplementary Table 1.

***Western blot assay***

Briefly, cells were lysed using RIPA(Qiagen). lysis buffer with a protease inhibitor cocktail. Proteins were separated using 8–15% sodium dodecyl sulfate-polyacrylamide gel electrophoresis and were transferred to polyvinylidene fluoride membranes. After blocking with 5% nonfat milk(Qiagen), membranes was incubated with primary antibodies at 4°C overnight. Membranes were then washed extensively with tris-buffered saline-Tween 20 and incubated with a horseradish peroxidase-conjugated secondary antibody for 1 h at room temperature. Proteins were visualized using an ECL Plus detection system (GE Healthcare, Waukesha, WI, USA). The band densities of each sample were normalized to β-actin. The primary antibodies used were α-SMA (cat. no. 19245; Cell Signaling Technology [CST]), COL1A1 (ab270993; Abcam, Cambridge, UK), COL3A1 (ab184993; Abcam), CTGF (86641; CST), MMP-2 (40994; CST), MMP-9 (ab76003; Abcam), TIMP-1 (ab211926; Abcam), p-SMAD2 (ab188334; Abcam), p-SMAD3 (ab52903; Abcam), SMAD2 (ab40855; Abcam), SMAD3 (ab40854; Abcam), TGF-β1 (3711; CST), TUBD1 (ab246956; Abcam), and β-ACTIN (A1978; Sigma-Aldrich, St Louis, MO, USA).

***Cell transfection***

To knock down circTUBD1, siRNA targeting circTUBD1 and its negative control were cloned into pLVX-IRES-Puro vectors, and lentiviruses were constructed by Genechem Co. Ltd. (Shanghai, China). Transfection was performed following the manufacturer’s instructions. Hsa-miR-203a-3p inhibitor and its negative control purchased from RiboBio Corporation (RiboBio) were transfected into LX-2 cells using Lipofectamine 3000 reagent (Invitrogen, Carlsbad, CA, USA). The siRNAs targeting SMAD3 and its negative control were transfected into LX-2 cells using RNAiMAX transfection reagent (Life Technologies) following the manufacturer’s instructions.

***Dual luciferase assay***

Potential target genes of circTUBD1 were predicted using online programs with different bioinformatic algorithms, including Circinteractome, miRanda v-2.0, and RNAhybrid-2.1. The target genes and binding sites for the seed region of miR-203a-3p were predicted using Targetscan, PicTar, and miRcode. Different fragment sequences (wild-type, mutant, and corresponding negative control) were synthesized and inserted into the pmirGLO vectors (Promega, Madison, WI, USA). All constructs were verified by sequencing. Based on the conserved motif of SMAD3, the binding sites of SMAD3 on the TUBD1 promoter sequence were predicted using the JASPAR database (http://jaspa r.gener eg.net/). Three putative sites in the promoter sequence of TUBD1 were identified (+533 to +542, +1220 to +1229 of the sense strand, and +1220 to +1229 of the antisense strand). To determine functionally relevant SMAD3 binding sites, the putative binding sites for the seed region of SMAD3 were mutated using site-specific sequence mutagenesis kits (TransGen, Beijing, China). GV141-SMAD3, which contained the coding region of SMAD3, and a luciferase reporter plasmid GV238-TUBD1 that contained the three putative binding sites in the promoter sequence of TUBD1 were constructed by Genechem. LX-2 cells were seeded into 24-well plates. LX-2 cells at 60–70% confluence per well were transfected with 1 μg GV238-TUBD1 plasmid, 1 μg GV141-SMAD3 plasmid, and 20 ng Renilla luciferase plasmid was an internal control by usig Lipofectamine 3000 reagent (Promega). All dual luciferase assays were performed following the manufacturer’s instructions using a dual luciferase assay kit (Promega). Each assay was independently performed at least three times.