## Fig. S1 (Azami S. et.al.)

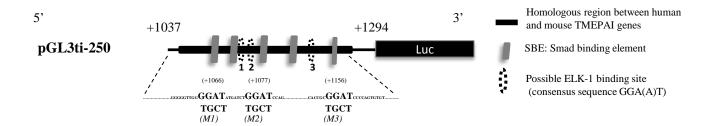
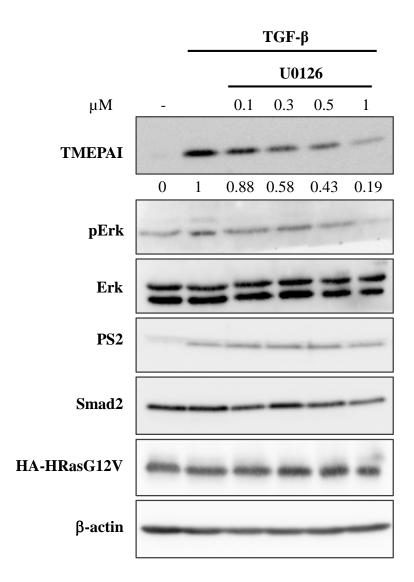
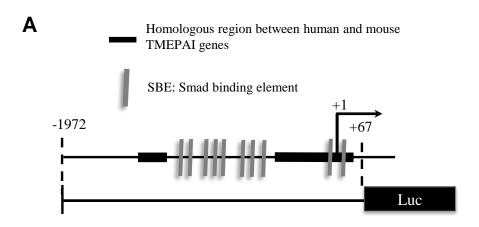


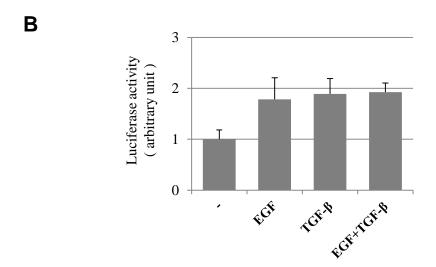
Fig. S1: Schematic representation of pGL3ti-250 reporter containing the ELK-1 binding sites. Three ELK-1 –binding sites possesses consensus sequence GG(A)T at position +1066, +1077, and +1156 within the first intron of the TMEPAI gene. The binding site mutants were made by changing GGAT to TGCT.



**Fig. S2:** EGF/Ras/MAPK pathway enhances TGF- $\beta$  induced-TMEPAI expression. HaCaT-RasG12V cells were treated with MEK inhibitor U0126, as indicated, 1 h before TGF- $\beta$  (0.5 ng/ml) stimulation for 8 h. Cell lysates were subjected to immunoblot analysis and detected with anti-TMEPAI antibody (9F10). The levels of phosphorylated Erk, total ERK, phosphorylated Smad2, Smad2 and HA-RasG12V were also detected to examine the effects of TGF- $\beta$  and U0126 treatments.  $\beta$ -actin was used as the loading control. Relative expression levels of TMEPAI/ $\beta$ -actin were detected by densitometry and indicated below the panels.

Fig. S3 (Azami S. et.al.)





**Fig. S3:** Transcriptional activity of TMEPAI promoter upon EGF and TGF- $\beta$  stimulation. (A) Schematic representation of the TMEPAI 5'-promoter luciferase reporter construct - 1972TMEPAI-luc. The nucleotide numbers of mouse TMEPAI gene was shown with the transcriptional initiation site as +1. (B) HepG2 cells were transfected with -1972TMEPAI-luc and stimulated with EGF (10 ng/ml), TGF- $\beta$  (0.1 ng/ml), or both EGF (10 ng/ml) and TGF- $\beta$  (0.1 ng/ml) for 18 h.

## Reporter: (CAGA)<sub>12</sub>-Luc

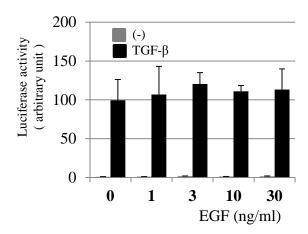


Fig. S4: The effect of EGF on Smad dependent transcription. HepG2 were transfected with  $(CAGA)_{12}$ -Luc and treated with EGF for 18 h with indicated concentrations in the presence or absence of TGF- $\beta$  (0.1 ng/ml) stimulation.

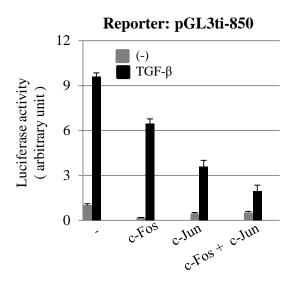


Fig. S5: The involvement of AP-1 transcriptional factor on TMEPAI expression. HepG2 cells were transfected with pGL3ti-850 together with c-Fos and/or c-Jun as indicated in the presence or absence of TGF- $\beta$  (0.1ng/ml) for 18h.

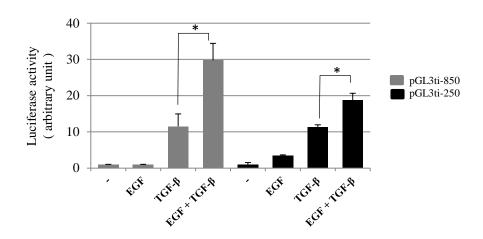


Fig. S6: Transcriptional activity of the first intron of TMEPAI gene upon EGF and TGF- $\beta$  stimulation. HepG2 cells were transfected with pGL3ti-850-luc or pGL3ti-250-luc, and stimulated with EGF (10 ng/ml), TGF- $\beta$  (0.1 ng/ml), or both EGF (10 ng/ml) and TGF- $\beta$  (0.1 ng/ml) for 18 h. (\*P < 0.05)