

# **InductionofcJunexpression**

**Mark Pierce, David Munoz, Cory Nichols, Timothy Hart,  
Casey Campbell, Tracy Reyes**

**Universiti Sains Malaysia**

by TGF- $\beta$  Induction of c-Jun ex- the same way. The RPMI cells were  
pression by TGF- $\beta$  by TGF- $\beta$  1All strains then treated with TGF- $\beta$ , and tumors  
were grown in Dulbecco's medium and were detected in the blood of WT and  
cultured in RPMI 1640 medium for the CD133- expressing cells at the indicated  
indicated time periods. 2. Results 2.1. time intervals. These results indicated  
TGF- $\beta$  induced c-Jun expression in wild- that TGF- $\beta$  modulates p38 MAPKs in  
type WT and CD133- and p38-expressing the same manner as the effect of TGF-  
plasmids WT and CD133- and p38-expressing CD11c expression. 3.2. TGF-  
plasmids were established by incuba- b induces c-Jun expression in CD133-  
tion with lentivirus GFP and cDNA, and p38-expressing plasmids CD133 is  
respectively, as described previously 2.2. a positive regulator of the p38 MAPKs  
TGF- $\beta$  induced c-Jun expression in Taq- [6–8]. CD133 is a positive regulator of  
Man wild-type plasmid expressing Taq- the p38 MAPKs, but is also involved in  
Man cDNA (WT and CD133) and p38- Toll-like receptor-like (TLR)-like neu-  
expressing plasmid (p38-expressing plas- rotoxic effects [9–11]. CD133 is a neg-  
mid) (upper panel) 2.3. TGF- $\beta$  induced ative regulator of TLR-like, and TLR-  
c-Jun expression in WT and CD133- like pathways [12–17]. The mechanisms  
expressing plasmids WT and CD133- by which TGF- $\beta$  modulates CD11c ex-  
and p38-expressing plasmid 2.4. WT pression are currently unknown. In this  
and CD133-expressing plasmids showed study, we investigated the c-Jun and  
increased c-Jun expression, which was c-Jun binding sites in the CD133- and  
consistently increased in WT (Fig. 3A p38-expressing plasmid, and showed that  
and B) and CD133- expressing plas- CD133 was bound to p38 MAPKs, but  
mids (Fig. 3C and D) (upper panel) not to the p38 MAPKs in WT and  
2.5. TGF- $\beta$  induced c-Jun expression CD133- and p38-expressing plasmids,  
in CD133- and p38- expressing plas- suggesting that TGF- $\beta$  modulates p38  
mids (Fig. 3E and F) 2.6. WT and MAPKs in the same way. 3.3. TGF-  
CD133-expressing plasmids showed in- b induces c-Jun expression in CD133-  
creased c-Jun expression, which was and p38-expressing plasmids CD133 is  
consistently increased in WT (Fig. 3G a positive regulator of the p38 MAPKs,  
and H) 2.7. TGF- $\beta$  induced c-Jun ex- but is also involved in Toll-like receptor-  
pression in CD133- and p38- express- like (TLR- like) neurotoxic effects [18–20].  
ing plasmids (Fig. 3I and J) (upper In this study, we investigated the c-Jun  
panel) 2.8. WT and CD133-expressing and c-Jun binding sites in the CD133-  
plasmids showed increased c-Jun ex- and p38-expressing plasmid, and found  
pression, which was consistently increased that CD133 was bound to p38 MAPKs  
in WT (Fig. 3K and L) 2.9. TGF- $\beta$  in WT but not in CD133- and  
induced c-Jun expression in CD133- and  
p38- expressing plasmids (Fig. 3M and  
M) (upper panel) 3. Discussion 3.1.  
TGF- $\beta$  is known to modulate CD11c  
expression, which is associated with a  
decrease in the p38 MAPKs, but other  
studies have shown that TGF- $\beta$  mod-  
ulates CD11c expression [1–5]. TGF-  
 $\beta$  may also modulate p38 MAPKs in