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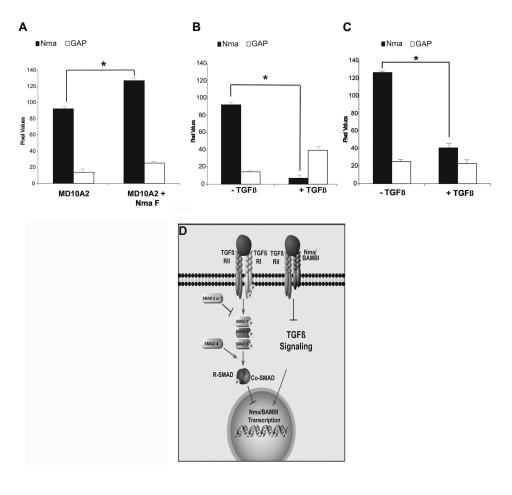
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Competing Roles of TGF β and Nma/BAMBI in Odontoblasts

APPENDIX

Semi-quantitative RT-PCR (SQ-RTPCR) amplification of Nma/BAMBI expression (500 bp) and GAPDH (407 bp) transcripts from MD10-A2 cells transiently co-transfected with Nma/BAMBI-V5-His expression construct and the DSPP-Luc reporter construct. (A) SQ-RTPCR of Nma/BAMBI in MD10-A2 cells

transfected with Nma/BAMBI-V5-His expression construct (Nma F) compared with non-transfected control cells. The asterisk (*) indicates that transfected cells expressed statistically significantly higher levels of Nma/BAMBI compared with non-transfected control cells (n = 4; p = 0.05; \pm SD). (B) SQ-RTPCR of Nma/BAMBI in MD10-A2 cells treated with TGF β compared with untreated cells. TGF β significantly decreased endogenous



Nma/BAMBI expression in non-transfected MD10-A2 cells, as indicated by the asterisk (*) (n = 4; p = 0.05; \pm SD). (C) SQ-RTPCR of Nma/BAMBI in MD10-A2 cells co-transfected with the Nma/BAMBI-V5-His expression construct (Nma F) and the DSPP-Luc reporter construct. TGF β significantly decreased Nma/BAMBI expression in transfected MD10-A2 cells, as indicated by the asterisk (*) (n = 4; p = 0.05; \pm SD). (D) Illustration summarizing the competing roles of Nma/BAMBI and TGF β .

Nma/BAMBI interacts with the TGF β /TGF β RII complex, thereby inhibiting its ability to interact with other TGF β type I receptors. Nma/BAMBI also lacks a serine/threonine kinase domain; thus, no downstream phosphorylation events take place, and TGF β action is halted. In this illustration, we demonstrate that TGF β signaling down-regulates Nma/BAMBI mRNA transcription. However, if Nma/BAMBI is present, it blocks TGF β signaling and allows Nma/BAMBI mRNA transcription to occur.