Bone morphogenetic protein-2 (BMP-2) and transforming growth factor- $\beta$ 1 (TGF- $\beta$ 1) alter connexin 43 phosphorylation in MC3T3-E1 Cells

# **ABSTRACT**

The mRNA or protein levels of Cx43 are not altered by BMP-2 and TGF-1 expression. These factors may reduce the phosphorylated form of this molecule in MC3T3-E1 cells and inhibit GJIC.

### INTRODUCTION

Introduction The purpose of this study was to gain insight into the role of BMP-2 and TGF-β1 in bone morphogenesis in MC3T3-E1 cells.

## Materials and Methods:

In vitro bone morphogenesis was induced in MC3T3-E1 cells by transfection with BMP-2 or TGF-β1. The growth factor of interest was used as a control.

## Results:

The BMP-2 and TGF-β1 mutant cells were significantly more sensitive to collagenase than the non-mutant cells. The expression of the β1 subunit of the BMP-2 Cell-cell communication through gap junctional intercellular communication (JIC) is a mechanism that has been implicated in the maintenance of interferon homeostasis and regulation of signals during embryogenesis, differentiation, growth and regeneration. Gap junctions have been shown to exist between osteoblasts and osteocytes in homologous and heterologOUS cell populations using electron microscopy. Earlier research has shown that the expression, phosphorylation, and subcellular localization of Cx43 in MC3T3-E1 cells was significantly inhibited by BMP-2 or TGF-1. We hypothesize that Ccx3, the primary gap junctional protein expressed in this cell type, also mediates intercellular communication. In contrast, we report on the effects of Bmp-2 and TNF--1 on CX43 specifically in these murine-derived cells.

### CONCLUSION

Remarkable conclusions The mRNA or protein levels of Cx43 are not altered by BMP-2 and TGF-1 expression. These factors may reduce the phosphorylated form of this molecule in MC3T3-E1 cells and inhibit GJIC.