

# Alterations in osteoclast morphology following long-term 17beta-estradiol administration in the mouse

## ABSTRACT

The results provide further insight into the structure and function of osteoclasts in the mouse model undergoing in vivo treatment with high levels of estrogen.

## INTRODUCTION

The osteoclast (O1) is a cell-like structure in the bone, which forms the core of the bone marrow. Osteoclast-like structures are produced by osteocytes, which are cells that develop in bone marrow precursor cells. In the case of the O1, the O1 is produced by osteocytes, and it is a primary site for bone marrow differentiation. Osteoclast-like structures can be divided into three types. Type I osteoclast-like structures are associated with bone marrow precursor cells. Type II osteoclast-like structures are associated with bone marrow precursor cells. Type III osteoclast-like structures are associated with bone marrow precursor cells that have not yet differentiated. The O1 is a type III osteoclast-like. While the osteoclast's role in bone resorption is being better understood, there is still not a lot of information about osteoclastogenesis and the specific mechanism of action of anti-resorbing agents on functional osteoclasts. 17-estradiol is particularly important because its decline at menopause is linked to the development of postmenopausal osteoporosis. The present report describes alterations in osteoclast morphology following long-term administration of 17-estradiol to B6D2F1 mice. This provides an interesting and useful model as marrow stromal cells, which not only encode the precursors for osteogenic cell lineages but also exert significant effects on osteoclastic and systemic factors. In the current study, osteoclasts were evaluated in mice treated with estrogen to determine if they maintained tartrate-resistant acid phosphatase activity (TRAP) and their normal ultrastructural characteristics.

## CONCLUSION

Remarkable conclusions The results provide further insight into the structure and function of osteoclasts in the mouse model undergoing in vivo treatment with high levels of estrogen.