ABSTRACT
The genes studied for the disorder were all up-regulated by fracture in both age groups. This meant that the older rats did not heal quickly, and it may be due to the delayed union of their rats and the return of mRNA gene expression to baseline values.

INTRODUCTION
The role of the female sex hormone estrogen in the process of bone formation and remodelling of the femoral bone has been well established. However, the exact mechanisms of action of estrogen are still unknown. The aim of this study was to investigate the possible role of estrogen in bone remodelling and formation. We hypothesized that estrogen and its actions on bone remodelling and formation might be affected by the level of the progesterone receptor, a trans-activator of the GAD.

Methods: We used a rat model of osteoporosis to investigate the effects of estrogen on bone remodelling and formation.

Results: The level of the estrogen receptor was associated with a reduced bone mass in the femoral bone at 1 month of age. The results of the study showed that the level of the estrogen receptor was also Despite the fact that the radiographic and histologic progression of healing is well understood, there is limited information about cytokines that regulate and control this process at the cellular level. The bone morphogenetic proteins (BMPs) are expressed in the fracture callus by immunostaining, massaging of mRNA, and in situ hybridization of BMP-receptors during fracture repair. Morone et al. reported that Bmp-6 expression was sequentially observed during spinal fusion healing in rabbits. The disruption of BMPs and other skeletally active cytokines may be necessary to explain the failure of healing in delayed unions. However, it is unclear why some fractures do not heal completely and require surgical intervention. The healing rate for mid-shaft femoral fractured rats has been found to be faster compared to older rats, and earlier studies indicated that one-year-old rats did not reach normal biomechanical strength within 24 weeks after the fracture. Young rats responded better to this diagnosis, achieving Normal biomechanics within four weeks of fracture opening. In this model, it takes longer for rats to begin a periosteal reaction after fracture and bridging callus, which slows down fracture healing in both humans and Despite ongoing research, the cause of the slow healing process for fractures in older animals remains uncertain. It has been suggested that changes in the periosteal cell layer can lead to an increase in mitosis and a decrease in available osteogenic stem cells. This may be reflected in this phenomenon. Alternating the expression of cytokines that regulate fracture healing could result in slower healing. Studies have revealed that the genes responsible for embryonic induction of bones and cartilage are also involved in fracture recovery. BMPs, such as Bmp-2 and BIMP-4, are expressed with high frequency in the fracture callus of rats and mice. The discovery of delayed fracture healing in older individuals led to the conclusion that this could be caused by changes in the BMP-signaling pathway. To test this hypothesis, bone fracture was induced in young and older rats, and gene expression was measured in their fracture callus at different times. Osteocalcin, type I collagen,and type II collagen were used as markers of fracture callingus formation. Bmp-2, BAMP-4 and the type IIIA B MP receptor were also suggested as potential regulators of the healing process.

CONCLUSION

Remarkable conclusions Rats that experienced mid-diaphyseal fractures had higher mRNA expression for osteocalcin, type I collagen 1, type II collagen, BMP-2, and Bmp-4, as well as the type IIIbp receptor. The younger rats had elevated gene expression until the younger ones were healed, while the older ones had slower growth in cytokine gene Expression. This suggests that bone-inductive genes were not involved in negative feedback during fracture healing.