Thiazolidinediones induce osteocyte apoptosis and increase sclerostin expression

Titre	Thiazolidinediones induce osteocyte apoptosis and increase sclerostin expression
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Mots-clés	Apoptosis [4], osteocyte [5], RANKL [6], sclerostin [7], thiazolidinedione [8]
Résumé en anglais	Aims Thiazolidinediones (TZDs) are associated with a higher risk of bone fracture in women compared with men. The aim of the present study was to investigate whether TZDs could influence osteocyte behaviour and contribute to the skeletal phenotype observed in TZD-treated patients.Methods The murine MLO-Y4 cell line was used as a source of osteocytes. These cells were cultured for 24 h with 0, 10−8 m, 10−7 m, 10−6 m, 10−5 m or 10−4 m of pioglitazone, rosiglitazone or troglitazone in the presence or absence of 17β-oestradiol. The extent of osteocyte apoptosis was assessed, as was the expression of the bone formation inhibitor sclerostin and receptor activator for nuclear factor κB ligand (RANKL) also.Results In the absence of 17β-oestradiol, pioglitazone, rosiglitazone and troglitazone induced osteocyte apoptosis dose-dependently even at the lowest concentration of 10−8 m. Furthermore, the expression of sclerostin but not RANKL was significantly increased in TZD-treated cultures compared with untreated cultures. The presence of 17β-oestradiol significantly reduced TZD-induced osteocyte apoptosis and also sclerostin up-regulation.Conclusions These findings therefore raise the potential concern of using TZDs in post-menopausal women where the lack of oestrogen would not prevent osteocyte apoptosis and sclerostin up-regulation and may aggravate the reduction in bone mass in these patients.
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