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Comparative Effects Between Green Tea and Black Tea Polyphenols in Suppressing Adverse Effects of TNF-α Induced Inflammation in Osteoblasts

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Abstract: The aim of the study was to compare the osteoprotective Effects of Green Tea (GTE) and Black Tea (BTE) Extracts on Normal Human Osteoblast (NHOst) cells in non-inflammatory and inflammatory conditions. NHOst cells were treated with GTE and BTE 5, 10, 50 and 100 μg/mL for 2, 5 and 10 days. The experiments were performed in the absence and presence of Tumour Necrosis Factor-α (TNF-α) to emulate non-inflammatory and inflammatory conditions, respectively. All concentrations of GTE and BTE exhibited = 80% cell proliferation at all-time points. In the absence of TNF-α, 5 μg/mL of GTE and BTE significantly up-regulated Osteo Prote Gerin (OPG) level compared to control and 100 μg/mL of the extracts reduced Receptor Activator of Nuclear factor Kappa B Ligand (RANKL) level on day 5 and 10. In inflammation, 5 and 50 μg/mL BTE significantly elevated OPG level while with GTE only 5 μg/mL gave a similar effect. Higher concentrations 50 and 100 μg/mL of both extracts significantly suppressed RANKL expression. The 100 μg/mL GTE and all BTE concentrations tested except 100 μg/mL significantly increased Alkaline Phosphatase (ALP) activity by day 5 in non-inflammatory condition. About 5 μg/mL GTE increased the ALP activity in inflammatory condition. Likewise, BTE was also found to reverse the TNF-α effect by elevating the ALP activity. GTE and BTE increased formation of mineralized nodules in both conditions at each time points. BTE and GTE exert protective effects on osteoblast activities including reverting the TNF-α-induced adverse effects and these effects are more pronounced in BTE treatment.

Key words: Black tea, polyphenols, chronic inflammation, osteoblasts, OPG, RANKL

INTRODUCTION

Chronic inflammatory diseases such as Rheumatoid Arthritis (RA), psoriasis, ankylosing spondylitis, systemic lupus erythematous, multiple sclerosis inflammatory bowel diseases, pemphigus vulgaris, chronic periodontitis and others are frequently associated with bone loss and increased skeletal fragility (Dimitroulas *et al*., 2013; Straub *et al*., 2015). Inflammation-induced pathologic bone loss occurs as a result of disturbances in the normal bone remodelling process. Normal bone remodelling is a balance between bone-forming osteoblast and bone-resorbing osteoclast activities. Osteoblasts are responsible for mineralization of bone and modulation of osteoclast differentiation (Baum and Gravallese, 2014). Osteoblasts regulate osteoclast differentiation through its production of several factors including RANKL and OPG (Baum and Gravallese, 2014). RANKL interacts with the RANK receptor on the osteoclast precursors to stimulate osteoclastogenesis. OPG functions as a decoy receptor for RANKL to inhibit the binding of RANKL to RANK receptor, thus, limiting osteoclastogenesis and protecting against excessive bone resorption (Baum and Gravallese, 2014; Weitzmann, 2013). Chronic inflammation is associated with excessive production of pro-inflammatory cytokines including TNF-α and Inter Leukin-6 (IL-6) and their occurrence in the bone microenvironment inhibits the actions of osteoblasts, resulting in uncoupling of resorption and formation in favour of excess resorption.

Polyphenols, abundantly exist as constituents of fruits, vegetables, cereals, dry legumes, chocolate and

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