

EDITORIAL

OSTEOPOROSIS AND LEPTIN

Osteoporosis is a metabolic disorder associated with aging. The prevalence of osteoporosis is alarmingly increasing in Pakistan with osteopenia present even at a higher rate in young and illiterate population^[1] There are multiple well-known conventional factors involved in the progression of osteoporosis; however, after the discovery of several adipocytokines, the researchers have focused on exploring their role in the mechanism of the osteoporosis. Several studies have demonstrated that these bioactive molecules modulate the function of osteoblasts and osteoclasts and are involved in bone metabolism. One such adipokine is leptin, which is a 16kDa protein and has been linked with bone metabolism. Few studies have confirmed that leptin is involved in the pathophysiology of osteoporosis at different levels while others reported contrasting results^[2] Despite the abundance of literature regarding the specific role of leptin in the process of osteoporosis; however, still their interplay with bone mass density and other bone resorption and bone ----- markers is uncertain.

Further investigations are needed to find out whether leptin plays synergistic, opponents, or agonists' impacts on bone shaping. A case-control follow-up study investigated effects of bisphosphonates treatment on serum leptin level in osteoporotic females and concluded that treatment with bisphosphonate reduces serum leptin levels and it might be one of the mechanisms of action of bisphosphonate to alleviate osteoporosis signs and symptoms^[3] However, several other studies concluded that body mass index and body weight had an impact on bone mineral density, but serum leptin is not associated with BMD in both premenopausal females and postmenopausal osteoporotic females.^[4-5] Although several basic and clinical studies explaining the possible mechanism of leptin in the wellbeing and disease process of osteoporosis, yet there is no consensus for their involvement. Leptin may affect the bone synthesis or resorption process by directly or indirectly interacting with other biochemical parameters. Our future research should be focused around these significant questions whether leptin could be used as an osteoporosis marker and changing levels of leptin have protective or counter protective role in developing osteoporosis, and administration of synthetic leptin

adjunct with other anti-resorptive treatment have some advantageous role in treating osteoporosis. It is quite possible that in future we can design such novel agents that directly stimulate or inhibit leptin secretion and directly or indirectly interfere with the bone resorption or synthesis. Despite of several types of research still, there is a gap in the knowledge, which hinders our understanding regarding the possible role of leptin in the process of osteoporosis. Therefore, further experimental and clinical studies are needed to elucidate leptin exact role in the process of osteoporosis.

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Dr. Saba Tariq

Associate Professor/ Head of Department
Pharmacology and Therapeutics
University Medical & Dental College,
The University of Faisalabad