Scientists find linkages between serotonin reuptake inhibitors and bone mass

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Scientists at The Forsyth Institute have found that fluoxetine (Prozac), a drug used in the treatment of depression and obsessive-compulsive disorders, increases bone mass. The team of researchers analyzed the ability of fluoxetine to stimulate new bone formation under normal conditions and to block bone loss caused by inflammation or estrogen loss due to ovariectomy. They found that the antidepressant induced the formation of new bone under normal conditions and reversed total bone loss triggered by inflammation.

Bone destruction is characteristic of several chronic inflammatory diseases including rheumatoid arthritis and gum disease. Previous research has shown a correlation between the serotonin transporter -- serotonin is the chemical substance involved in transmitting signals between neurons and which plays a role in anxiety and mood disorders-- and bone destructive cells (osteoclasts). (Fluoxetine is a serotonin reuptake inhibitor (SSRIs).) However, it was not clear what role serotonin played in bone metabolism.

Trabecular bone, one of two main types of bone, is spongy, and makes up the bulk of the interior of most bones, including the vertebrae. After a six-week treatment with fluoxetine, laboratory mice showed increased trabecular bone volume and total volume in femurs and vertebrae as determined by micro-computed tomography. Fluoxetine-treated animals were not protected from bone loss after ovariectomy, suggesting that its anabolic effect requires the presence of estrogen. The effect on bone loss was also investigated following a bacterial-mediated inflammatory

challenge. Injections of lipopolysaccharide (LPS), a component of the membrane of certain strains of bacteria, resulted in an increased number of osteoclasts and net bone loss. However, LPS given with fluoxetine caused new bone formation and a net gain in bone mass. The study concluded that fluoxetine treatment in vivo leads to increased bone mass under normal physiological conditions or inflammatory conditions, but does not prevent bone loss associated with estrogen deficiency.

This research, which will be published in the next issue of the Journal of Cellular Biochemistry, currently available online, was led by Ricardo Battaglino, PhD, Assistant Member of the Staff in the Department of Cytokine Biology at The Forsyth Institute.

"As this class of medication is widely prescribed and used across all age groups, the consequences of the relationship between these drugs and bone metabolism may be very relevant to public health. This work will help us learn more about the underlying causes of osteoporosis and gain a new understanding of bone formation at a molecular level," said Dr. Battaglino. "Furthermore, this research provides exciting clues on how to prevent destructive bone loss and even improve bone mass in certain medical/dental conditions."

Source: Forsyth Institute