Oxytocin

Oxytocin is a peptide of 9 amino acids (Cys-Tyr-Ile-Gln-Asn-Cys-Pro-Leu-Gly).

It acts on certain smooth muscles:

stimulating contractions of the uterus at the time of birth;

stimulating the release of milk when the baby begins to suckle.

Oxytocin is often given to prospective mothers to hasten birth. In rodents, oxytocin also acts on the nucleus accumbens and amygdala in the brain where it enhances:

bonding between males and females after they have mated;

bonding between a mother and her newborn.

In mice, oxytocin acts on striated muscle stem cells to promote repair after they have been injured.

In humans, oxytocin increases the level of one's trust in other people.

The pituitary stalk (also known as the infundibular stalk or simply the infundibulum), is the connection between the hypothalamus and the posterior pituitary.

The floor of the third ventricle is prolonged downward as a funnel-shaped recess, the infundibular recess, into the infundibulum, and to the apex of the latter the hypophysis or pituitary is attached.

It passes through the dura mater of the diaphragma sellae as it carries axons from the magnocellular neurosecretory cells of the hypothalamus down to the posterior pituitary where they release their neurohypophysial hormones, oxytocin and vasopressin, into the blood.

This connection is called the hypothalamo-hypophyseal tract or hypothalamo-neurohypophyseal tract.

OT and OTR can be expressed on bone marrow mesenchymal stem cells (BMSCs), osteoblasts (OB), osteoclasts (OC), osteocytes, chondrocytes, and adipocytes. OB can synthesize OT under the stimulation of estrogen as a paracrine-autocrine regulator for bone formation. OT/OTR, estrogen, and OB form a feed-forward loop through estrogen mediation. The osteoclastogenesis inhibitory factor (OPG)/receptor activator of the nuclear factor kappa-B ligand (RANKL) signaling pathway is crucially required for OT and OTR to exert an anti-osteoporosis effect. Downregulating the expression of bone resorption markers and upregulating the expression of the bone morphogenetic protein, OT could increase BMSC activity and promote OB differentiation instead of adipocytes. It could also stimulate the mineralization of OB by motivating OTR translocation into the OB nucleus. Moreover, by inducing intracytoplasmic Ca2+ release and nitric oxide synthesis, OT could regulate the OPG/RANKL ratio in OB and exert a bidirectional regulatory effect on OC. Furthermore, OT could increase the activity of osteocytes and chondrocytes, which helps increase bone mass and improve bone microstructure. This paper reviews recent studies on the role of OT and OTR in regulating cells in bone metabolism as a reference for their clinical use and research based on their reliable anti-osteoporosis effects [1)]

1)
Feixiang L, Yanchen F, Xiang L, Yunke Z, Jinxin M, Jianru W, Zixuan L. The mechanism of oxytocin and its receptors in regulating cells in bone metabolism. Front Pharmacol. 2023 May 9;14:1171732. doi: 10.3389/fphar.2023.1171732. PMID: 37229246; PMCID: PMC10203168.