

## **PRESS RELEASE**

### **OSTA BIOTECHNOLOGIES INC. ANNOUNCES A BREAKTHROUGH DISCOVERY IN AGING AND CANCER RESEARCH**

#### ***A PLENARY TALK WAS GIVEN AT THE 27<sup>th</sup> ANNUAL MEETING OF THE AMERICAN SOCIETY OF BONE AND MINERAL RESEARCH***

MONTREAL, QC – October 4, 2005 - Osta Biotechnologies Inc. today announced that its researchers Dr. David Goltzman, Dr. Andrew Karaplis and Dr. Dengshun Miao have discovered a key role of specific segments of the parathyroid hormone related peptide (PTHrP) on growth retardation and early aging in mice. The researchers found that the expression of tumor suppressing genes p16<sup>INK4a</sup> and p21 was significantly increased, but the expression of oncogene, Bmi-1 was reduced in the skeleton of the animal model compared to the normal control.

Dr. Andrew Karaplis, Osta's President & Chief Scientific Officer commented "We are very excited about these major findings and their potential applications. We have demonstrated that mid- and carboxyl regions of PTHrP play a critical role on growth retardation and early aging in mice. These findings open up novel approaches for the treatment of cancer and aging in humans."

Dr. Ajay Gupta, Osta's Chairman and CEO added: "This breakthrough was achieved by our researchers at McGill University and we have an exclusive option to license this technology from McGill University. Although much work is needed before a product can be offered to prolong life in people or to treat cancer, this research has led to a very significant discovery and opens new and very promising avenues for developing therapeutics to treat cancer and to prolong life in humans."

Dr. Dengshun Miao, the Company's Director of Research & Development, was invited to give a plenary talk entitled "Deletion of the mid and carboxyl regions of PTHrP produces growth retardation and early senescence in mice" at the 27<sup>th</sup> annual meeting of the American Society of Bone and Mineral Research (ASBMR) on September 25, 2005."

Osta's researchers discovered a key role of mid- and carboxyl regions of PTHrP on growth retardation and early senescence in mice created by inserting a premature termination codon TGA in the PTHrP gene corresponding to the codon encoding amino acid residue 85 of the mature protein and creating a "knock-in"(KI) mouse expressing truncated segment containing 1-84 residues of the mature form of the PTHrP protein.

Postnatal life in these mice was characterized by an early onset of phenotypic changes characteristic of aging, including unstable gait, cachexia, reduced fat deposition, osteoporosis and hyperkeratosis.

Although morphologically normal, newborn PTHrP KI mice were slightly smaller than their wild-type littermates. By 2 weeks of age, PTHrP KI mice were much smaller than wild-type littermates and died at 1-3 weeks postnatally with growth retardation, although having normal serum calcium and PTH levels similar to their wild type littermates. The mutant mice exhibited severely decreased accumulation of subcutaneous fat and radiographs of the long bones confirmed the presence of severe osteopenia. These mice appeared to have defects in skeletal development and premature osteoporosis.

Histological analysis revealed that skeletal growth retardation resulted from a reduction of chondrocyte proliferation and differentiation in PTHrP KI mice. Premature osteoporosis was caused by reduced osteoblastic bone formation in both trabecular and cortical bone associated with the down-regulation of osteoblastic gene expression levels of Cbfa1, ALP, type I collagen and osteocalcin in skeletal tissue and immunostaining for PTH/PTHrP receptor (PTHR) was dramatically decreased in bone sections from KI mice.

By postnatal day 14, the skin of KI mice was thinner with hyperkeratosis of the epidermis.

In view of the fact that senescence is the final phenotypic state of decreased cell proliferation, which is often mediated by increased expression and activation of tumor suppressor genes such as p16<sup>INK4a</sup> and p21, Osta's researchers examined the expression of p16, p21 and the oncogene, Bmi-1 in the tissues of the knock-in mice and in mouse embryonic fibroblasts (MEFs) from these animals. Osta's researchers found that the expression of p16<sup>INK4a</sup> and p21 was significantly increased, but the expression of Bmi-1 was reduced in the skeleton and MEFs from PTHrP KI mice compared to wild-type mice.

***Osta Biotechnologies Inc.***

Osta is a biopharmaceutical company listed on the TSX Venture Exchange (TSXV: OBI) dedicated to developing novel diagnostics and therapeutics for the aging population particularly in the areas of Osteoporosis, Osteoarthritis and Alzheimer's disease.

The TSX Venture Exchange does not accept responsibility for the adequacy or accuracy of this release.

*Certain information in this press release is forward-looking and is subject to numerous risks and uncertainties. By their nature, such forward-looking statements involve risks and uncertainties that could cause actual results to differ materially from those contemplated by the forward-looking statements. These risks include actions of Osta's competitors, and those inherent in scientific research and development.*

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