

# العلاقة بين معامل كتلة الجسم و هشاشة العظام الأوليه في كبار السن المصريين

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Relationship Between Body Mass  
Index and Primary Osteoporosis  
in Elderly Egyptian.

Thesis

*Submitted for partial fulfillment of Master degree in Geriatric Medicine*

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# List of Abbreviations

<b>ACTH</b>	Adrenocorticotrophic hormone
<b>ADL</b>	Activities of daily life
<b>AIDS</b>	Autoimmune deficiency syndrome
<b>BMD</b>	Bone Mineral Density
<b>BMI</b>	Body Mass Index
<b>BSAP</b>	Bone specific alkaline phosphatase
<b>CA</b>	Calcium
<b>COPD</b>	Chronic Obstructive Pulmonary Disease
<b>CTx</b>	C -telopeptide of collagen cross-links
<b>CVD</b>	Cardiovascular disease
<b>DEXA</b>	Dual Energy X-ray Absorptiometry
<b>DPA</b>	Dual-photon Absorptiometry
<b>Dpd</b>	deoxypyridinolines
<b>DRA</b>	Dual energy Radiographic Absorptiometry
<b>FDA</b>	Food-Drug administration
<b>FRAX</b>	Fracture Risk Assessment Tool
<b>g/cm<sup>2</sup></b>	gram/square centimeter
<b>GnRH</b>	Gonadotropin-releasing hormone
<b>IADL</b>	Instrumental Activity of daily living
<b>ICTP</b>	Cross-linked C -telopeptide of type I collagen

## List of Abbreviations (Cont.)

<b>Kg/m<sup>2</sup></b> .....	Kilogram/square meter
<b>MRI</b> .....	Magnetic Resonance Imaging
<b>NOF</b> .....	National osteoporosis foundation
<b>NTx</b> .....	N –telopeptide of collagen cross-links
<b>OC</b> .....	Osteocalcin
<b>OPG</b> .....	Osteoprotegerin
<b>PICP</b> .....	Carboxyterminal propeptide of type I collagen
<b>PINP</b> .....	Aminoterminal propeptide of type I collagen
<b>PO<sub>4</sub></b> .....	Phosphorus
<b>PTH</b> .....	Parathyroid hormone
<b>Pyd</b> .....	Pyridinolines
<b>QCT</b> .....	Quantitative computed tomography
<b>QTLs</b> .....	Quantitative trait loci
<b>RANK</b> .....	Receptor activator of nuclear factor Kappa-B ligand
<b>RANKL</b> .....	Receptor activator of nuclear factor kappa-B ligand
<b>RI</b> .....	Recombinant inbred
<b>SD</b> .....	Standard Deviation
<b>SERMs</b> .....	Selective estrogen receptor modulators
<b>SPA</b> .....	Single-photon absorptiometry
<b>SPECT</b> .....	Single-photonemission computed tomography
<b>SPSS</b> .....	Statistical Package for Social Sciences
<b>T2DM</b> .....	Type2 diabetes mellitus



## List of Abbreviations (Cont.)

**T-FN BMD**.....T-score of Femur Neck Bone Mineral Density

**T-LS BMD** .....T-score of Lumbar Spine Bone Mineral  
Density

**US**.....United States

**WHO**.....The world health organization



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## Introduction

Osteoporosis is accelerated bone loss. Normally, there is loss of bone mass with aging, perhaps 0.7% per year in adults. However, bone loss is greater in women past menopause than in men of the same age. The process of bone remodeling from resorption to matrix synthesis to mineralization normally takes about 8 months; a slow but constant process. Bone in older persons is not as efficient as bone in younger persons at maintaining itself. There is decreased activity of osteoblasts and decreased production of growth factors and bone matrix (*Sambrook and Cooper, 2006*).

Osteoporosis is a systemic disease in which bone density is reduced leading to the weakening of the skeleton and increase vulnerability to fractures (*Wells et al., 2005*).

It is called silent disease since there are few associated symptoms; osteoporotic fracture is chief clinical feature with an enormous burden on the health related quality of life and mortality (*Bagnato et al., 2007*).

Osteoporosis can be classified as primary or secondary. Primary osteoporosis is simply the form seen in older persons and women past menopause in which bone loss is accelerated over that predicted for age and sex. Secondary osteoporosis results from a variety of identifiable conditions (*Sweet et al., 2009*).

Several studies discussed the relation between Body Mass Index and osteoporosis, *Nguyen, 2000* found that body weight or body mass index (BMI) is positively associated with BMD.

Some epidemiological data show that higher body weight or BMI is positively correlated with bone mass, and weight loss may cause bone loss (*Guney et al., 2003; Radak, 2004 and Gnudi et al., 2007*). If so, increasing BMI in postmenopausal women is assumed to protect against osteoporosis (*Reid et al., 1992; Felson et al., 1993*). Furthermore, weight loss may increase significantly bone resorption markers, suggesting that body weight directly influences osteoclastic activity (*Hyldstrup et al., 1993*).

*Ricci et al. (2001)* support this hypothesis, in a cross-sectional study with 100 healthy postmenopausal women; it was observed that BMI is inversely related to serum tartrate-resistant acid phosphatase activity.

Still the relation between BMI and Osteoporosis is considered a research question in several studies and also how obesity can affect osteoporosis (*Greco et al., 2010*).

## Aim of the Work

To detect the relationship between body mass index and primary osteoporosis in elderly Egyptian.

# Osteoporosis

## **Introduction to Osteoporosis**

Osteoporosis is accelerated bone loss. Normally, there is loss of bone mass with aging, perhaps 0.7% per year in adults. However, bone loss is greater in women past menopause than in men of the same age. The process of bone remodeling from resorption to matrix synthesis to mineralization normally takes about 8 months, a slow but constant process. Bone in older persons just isn't as efficient as bone in younger persons at maintaining itself. There is decreased activity of osteoblasts and decreased production of growth factors and bone matrix (*Sambrook and Cooper, 2006*).

The World Health Organization (WHO) has defined osteoporosis as a spinal or hip bone mineral density (BMD) that is 2.5 standard deviations or more below the mean BMD for healthy, young women, measured by Dual Energy X-ray Absorptionmetry (DEXA). The WHO defines osteopenia as a spinal or hip BMD between 1 and 2.5 standard deviations below the mean for healthy, young women (*WHO, 2009*).

Osteoporosis affects 10 million women and men in the United States, with direct costs of \$17 billion in 2005 (*Burge et al., 2007*).

The prevalence of osteoporosis in men over the age of 50 years is 3 times less frequent than in women (*Kanis et al., 2008*).

## **Pathophysiology of Osteoporosis**

The overall architecture of bone is divided into cancellous bone (also referred to as trabecular bone) and cortical bone; because the surface area of cancellous bone far exceeds that of cortical bone, and is more metabolically active, cancellous bone is more severely affected if bone remodeling becomes uncoupled. During the accelerated period of bone loss immediately after menopause, cancellous bone loss increases 3-fold, while rates of cortical bone loss are slower, because the vertebrae are rich in cancellous bone, vertebral fractures are relatively common in the early postmenopausal years, with hip fractures tending to occur in later years (*American Medical Association, 1998*).

Approximately half of the bone mass is accumulated during pubertal development (*Bonjour et al., 1991*). This is associated with the increase in sex hormone levels and is almost completed with closure of the end plates. There is only minimal additional accumulation of the bone minerals during the next 5 to 15 years (skeletal consolidation).

Genetic factors have long been recognized as playing an important role in osteoporosis (*Zintzaras et al., 2006*).

The main factor influencing peak bone mass is genotype. The genes implicated in osteoporosis include those for the estrogen receptor, transforming growth factor- $\beta$ , and Apolipoprotein E and collagen (*Skugor, 2010*).