World Journal of Pharmaceutical Science and Research
www.wjpsronline.com
Review Article
ISSN: 2583-6579
SJIF Impact Factor: 5.111
Year - 2025
Volume: 4; Issue: 6
Page: 71-74

DEFINITIONS AND EVOLUTION OF THE TERM OSTEOPOROSIS

Dr. Swapnil Jaiswal*[1], Dr. Brahamanand Sharma[2], Dr. Saurabh Agrawal[3], Dr. Avadhesh Shandily[4], Dr. Himani Rot[5], Dr. Pooja Rajpurohit[6]

[1,5,6]M.D. Scholar, Post Graduate Department of Swasthavritta & Yoga, Post Graduate Institute of Ayurveda, Jodhpur.
[2]H.O.D. Post Graduate Department of Swasthavritta & Yoga, Post Graduate Institute of Ayurveda, Jodhpur.
[3,4]Associate Professor, Post Graduate Department of Swasthavritta & Yoga, Post Graduate Institute of Ayurveda, Jodhpur.

Article Received: 5 October 2025 | | Article Revised: 26 October 2025 | | Article Accepted: 16 November 2025

***Corresponding Author: Dr. Swapnil Jaiswal**
M.D. Scholar, Post Graduate Department of Swasthavritta & Yoga, Post Graduate Institute of Ayurveda, Jodhpur.
DOI: https://doi.org/10.5281/zenodo.17777766

How to cite this Article: Dr. Swapnil Jaiswal, Dr. Brahamanand Sharma, Dr. Saurabh Agrawal, Dr. Avadhesh Shandily, Dr. Himani Rot, Dr. Pooja Rajpurohit (2025) DEFINITIONS AND EVOLUTION OF THE TERM OSTEOPOROSIS. World Journal of Pharmaceutical Science and Research, 4(6), pg: 71-74. https://doi.org/10.5281/zenodo.17777766

ETYMOLOGY

The word osteoporosis comes from the combination of two ancient Greek words: "osteon" (ὀστέον) which means "bone" and "poros" (πόρος), which means "passage" or "pore." The term first coined by French pathologist Jean Martin Lobstein in 1829. His work, which was published in French, was based on his studies of the human skeleton.[1]

KEYWORDS: Osteoporosis, Osteoporosis Definition, Evolution, WHO, NIH, Calcium, Bone, BMD, Bone Remodeling.

DEFINATION

Low bone density and microarchitectural degradation of bone tissue, which increases bone fragility, are the hallmarks of osteoporosis, a systemic skeletal disease. The process of bone loss damages bone strength more than might be apparent from the straightforward metric of bone "density" since it results in a decline in skeletal microarchitecture.[1,2] Low bone mass, bone tissue degradation, and disturbance of bone architecture are the hallmarks of osteoporosis, a skeletal condition that impairs bone strength and increases the risk of fracture. Bone strength is weakened more than may be indicated by a straightforward measurement of bone "density" due to the degradation of skeletal microarchitecture brought on by bone tissue loss. A bone mineral density (BMD) at the lumbar spine, femoral neck, or total hip that is less than or equal to 2.5 standard deviations as determined by DXA below the mean for young, healthy

adults of the same sex and race—also referred to as a T-score of -2.5—is considered osteoporosis, per the WHO diagnostic classification.[3,4,5]

BONE REMODELING.[6]

Bone loss brought on by age-related changes in bone remodeling, as well as extrinsic and intrinsic factors that exacerbate this process, is the cause of osteoporosis. Both linear growth and the apposition of new bone tissue on the cortex's outer surfaces cause the skeleton to enlarge during growth. The latter procedure, known as modeling, enables the long bones to change shape in response to external pressures. Skeletal maturation requires increased sex hormone production during puberty, and it reaches its maximal mass and density in early adulthood.

In adulthood, the primary metabolic skeletal mechanism is bone remodeling rather than modeling. Repairing microdamage within the skeleton to preserve skeletal strength and the skeleton's relative youth, as well as supplying calcium from the skeleton as required to maintain serum calcium, are the two main purposes of bone remodeling.

Microdamage to bone brought on by high or sustained stress might trigger remodeling. Both osteocyte-mediated calcium transport and osteoclast-mediated resorption are involved in acute calcium needs.

Hormones such as estrogens (in both sexes), androgens, vitamin D, and parathyroid hormone (PTH) are also involved in bone remodeling. Additionally, locally produced growth factors like IGF-I, transforming growth factor β (TGF-β), PTH–related peptide (PTHrP), interleukins (ILs), prostaglandins, and members of the tumor necrosis factor (TNF) superfamily are involved. These variables mainly affect the rate at which new remodeling sites are triggered, which leads to bone resorption by osteoclasts at first, followed by a repair phase where osteoblasts produce new bone tissue.
In young adults, an equivalent amount of new bone tissue replaces resorbed bone. Therefore, after peak bone mass is reached by the age of about 20 years, the mass of the skeleton stays constant. However, the processes of resorption and formation become unbalanced at the ages of 30 to 45, with resorption outpacing formation. Women may experience an exaggeration of this imbalance following menopause or any other cause of estrogen deprivation. It may also start at different ages and vary at different bone sites. An increase in osteoclastic activity or a reduction in osteoblastic activity may be the cause of excessive bone loss. Furthermore, the slight imbalance observed at each remodeling unit may be exacerbated by an increase in the frequency of remodeling activation and, consequently, the number of remodeling sites.

In trabecular bone, fast bone loss and decreased cancellous connection result from osteoclasts penetrating trabeculae, which leaves no template for future bone production. This incident is more likely to occur when there are more remodeling sites. More remodeling activity in cortical bone results in more porous bone.

In contrast to the peripheral cortical bone, which has a lower turnover rate, the core trabecular bone in the human skeleton has a higher turnover rate.[7]

EVOLUTION OF DEFINATION OF OSTEOPORORSIS

Bone disease was not well understood thirty years ago. Even many medical professionals thought that fractured and weak bones were an inevitable aspect of aging. We now understand that this is untrue. Since the Guide's first publication by the National Osteoporosis Foundation (NOF) in 1999, it has become evident that many patients are not getting the proper information, testing, or care.[8]

The current founding of the definition followed following steps:

- **Early 20th Century:** The idea that menopause is the cause of vertebral fractures was first proposed by American endocrinologist Fuller Albright in 1941. Albright also played a significant role in popularizing the term in the medical community by pointing out the connection between ovarian dysfunction and vertebral fractures and the subsequent improvement of spinal issues with estrogen administration. He concentrated on "Too-Little-Calcified-Bone" and "Too-Much-Calcified-Bone" in relation to adult bone-related metabolic diseases. He further divided the former into two categories:

a) “Bone-Formation-Too-Little"

b) "Bone-Resorption-Too-Much".

Osteoporosis, in which the defect is in the laying down of the matrix, and osteomalacia, in which the defect is in the calcification of the matrix, are two subtypes of "bone-formation-too-little" because bone formation is comprised of two steps: the laying down of the matrix by the osteoblasts and the deposition of calcium salts in this matrix. Although "Osteogenesis Imperfecta" and "Osteoporosis" are closely similar, the former is distinguished by a lack of extracellular substance produced by osteoblasts rather than a lack of osteoblasts.[9]

- **1980s:** The two patterns of osteoporosis that Riggs et al. defined in the early 1980s were used for a very long time. Low levels of estrogen have been linked to type 1 osteoporosis, while low levels of calcium, vitamin D, and parathyroid hormone have been linked to type 2 osteoporosis, which is senile.[10,11]
- **1990s:** According to the 1990 Consensus Conference on Osteoporosis in Copenhagen, osteoporosis is characterized by an increased risk of fracture due to increased bone fragility as a result of decreasing bone mass.[12]
- **1994 WHO Definition:** Based on the recommendations of the 1990 Consensus Conference of Osteoporosis in Geneva, which indicated that bone mineral density ≤ 2.5 standard deviation of the T-score at the hip as osteoporosis.[13] the 1994 World Health Organization definition of osteoporosis was created. The T-score was computed using a reference population that is young and healthy and is balanced for gender and ethnicity. The *T*-score is calculated using the DEXA scan, and the patient’s score is then compared to nomograms.[14]
- **2001 NIH Update:** The NIH updated its definition again, as "a skeletal disorder characterized by compromised bone strength predisposing a person to an increased risk of fracture." This characterized osteoporosis and broadened the focus from just bone mass to encompass overall bone quality and architecture.[15]

  One of the most popular prediction tools is the FRAX tool. Models for predicting fracture risk have been created by evaluating a number of clinical factors. Nevertheless, the FRAX instrument is not endorsed by the World Health Organization.[16]

The way the concept of osteoporosis has evolved throughout time reflects our understanding of the pathophysiological mechanisms and risks associated with the condition. There is not much information available about the phrase's history. The NIH's 2001 definition, which was the most recent, still seems to be the most suitable.[17,18] Future developments in the understanding of other fracture-related factors, bone mineral density, and sophisticated diagnostic techniques may open the door for additional definitional advancements.

CONFLICT OF INTREST

On behalf of all authors, the corresponding author states that there is no conflict of interest.

REFERANCES

1. Prevention And Management Of Osteoporosis, Report of a WHO Scientific Group. ISBN 92 4 120921 6 ISSN 0512-3054
2. Loscalzo J, Fauci A.S., Kasper D.L., Hauser S.L., Longo D.L., Jameson J.L., Harrison's Principals of Internal Medicine, 2022; 21st edition: volume 1.
3. F. Cosman, S. J. de Beur, M. S. LeBoff, E. M. Lewiecki, B. Tanner, S. Randall & R. Lindsay, Clinician's Guide to Prevention and Treatment of Osteoporosis. Osteoporosis International With other metabolic bone diseases ISSN 0937-941X Osteoporos Int DOI 10.1007/s00198-014-2794-2.
4. Loscalzo J, Fauci A.S., Kasper D.L., Hauser S.L., Longo D.L., Jameson J.L., Harrison's Principals of Internal Medicine, 2022; 21st edition: Volume 1.
5. Prevention And Management Of Osteoporosis, Report of a WHO Scientific Group. ISBN 92 4 120921 6 ISSN 0512-3054
6. Loscalzo J, Fauci A.S., Kasper D.L., Hauser S.L., Longo D.L., Jameson J.L., Harrison's Principals of Internal Medicine, 2022; 21st edition: Volume 1.
7. Poduval M, Vishwanathan K, Definition and Evolution of the Term Osteoporosis. Springer, Indian J Orthop, 2023 Oct 31; 57(Suppl 1): 42–44. doi: 10.1007/s43465-023-01013-2
8. F. Cosman, S. J. de Beur, M. S. LeBoff, E. M. Lewiecki, B. Tanner, S. Randall & R. Lindsay, Clinician's Guide to Prevention and Treatment of Osteoporosis. Osteoporosis International With other metabolic bone diseases ISSN 0937-941X Osteoporos Int DOI 10.1007/s00198-014-2794-2
9. Albright, F., and Reifenstein, E. C. The Parathyroid Glands and Metabolic Bone Disease. Baltimore: The Williams & Wilkins Company; 1948.
10. Riggs BL, Wahner HW, Dunn WL, Mazess RB, Offord KP, Melton JL., III Differential changes in bone mineral density of the appendicular and axial skeleton with aging. Relationship to spinal osteoporosis. Journal of Clinical Investigation, 1981; 67: 328–333. doi: 10.1172/JCI110039.
11. Riggs BL, Melton LJ., 3rd Evidence for two distinct syndromes of involutional osteoporosis. American Journal of Medicine, 1983; 75: 899–901. doi: 10.1016/0002-9343(83)90860-4.
12. Christiansen C. Consensus development conference: prophylaxis and treatment of osteoporosis. The American Journal of Medicine, 1991; 90: 107–110. doi: 10.1016/0002-9343(91)90512-V
13. Ford N, Norris SL, Hill SR. Clarifying WHO's position on the FRAX® tool for fracture prediction. Bulletin of the World Health Organization, 2016; 94(12): 862. doi: 10.2471/BLT.16.188532.
14. Poduval M, Vishwanathan K, Definition and Evolution of the Term Osteoporosis. Springer, Indian J Orthop, 2023 Oct 31; 57(Suppl 1): 42–44. doi: 10.1007/s43465-023-01013-2
15. NIH Consensus Development Panel on Osteoporosis Prevention, Diagnosis, and Therapy. Journal of the American Medical Association (JAMA), 285(6)P: 785-795; 2001.
16. Ford N, Norris SL, Hill SR. Clarifying WHO's position on the FRAX® tool for fracture prediction. Bulletin of the World Health Organization, 2016; 94(12): 862. doi: 10.2471/BLT.16.188532.
17. Schapira D, Schapira C. Osteoporosis: The evolution of a scientific term. Osteoporosis International, 1992; 2(4): 164–167. doi: 10.1007/BF01623921.
18. Lorentzon M, Cummings SR. Osteoporosis: The evolution of a diagnosis. Journal of Internal Medicine, 2015; 277(6): 650–661. doi: 10.1111/joim.12369.