DXA in vivo BMD methodology: An erroneous and misleading research and clinical gauge of bone mineral status, bone fragility, and bone remodelling

H.H. Bolotin *

School of Medical Sciences, RMIT University, Bundoora, Victoria 3083, Australia

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Abstract

The seemingly unqualified reliance and near-universal dependence upon in vivo dual-energy X-ray absorptiometric (DXA) methodology to provide accurate, quantitative, and meaningful in vivo (in situ cadaveric) bone mineral areal density (“BMD”) determinations are proven to be unwarranted and misplaced. The underlying systematics of sizable, inherently unavoidable and un-correctable inaccuracies in the DXA output values of in vivo “BMD” are shown to be quantitatively consistent with being the root cause of unreliable, misdirected, and misinterpreted aspects of consensual knowledge of bone fragility, osteoporotic diagnostics/prognostics, and remodelling therapies. The “BMD” label that DXA ascribes to the output values of in vivo (in situ cadaveric) bone densitometry scans is shown to be a misnomer and an erroneous and invalid measure of bone mineral material. The DXA-derived “BMD” value does not correctly represent the areal density of bone mineral material, as it is contaminated by sizable, unavoidable, inextricable, independent soft tissue contributions. Due to intra- and extra-osseous soft tissue X-ray absorptiometric effects, it is unknown (and unknowable) exactly what DXA in vivo “BMD” is a measure of in any given case, or what proportion of the “BMD” value represents the actual bone mineral material areal density present in the DXA scan region of interest (ROI) of any predominantly trabecular bone-site (e.g., lumbar vertebrae, proximal femora). This inherent fundamental defect in DXA in vivo bone mineral areal density methodology adversely compromises both the validity and reliability of patient-specific diagnostic/prognostic evaluations, cross sectional and prospective studies, and DXA-based interpretations of bone quality and bone fragility. It further undermines the WHO characterizations (and definitions) of ‘normal’, ‘osteopenic’, and ‘osteoporotic’ classifications. It is also seen to make equivocal the qualitative and quantitative epidemiological estimates of the proportion of the population that is, or is deemed to become, osteoporotic. The present quantitative exposition shows DXA-measured in vivo “BMD” methodology to be an intrinsically flawed and misleading indicator of bone mineral status and an erroneous gauge of relative fracture risk.

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Introduction

Over the last several years, various perplexing anomalies and unresolved inconsistencies in the body of consensual knowledge of bone fragility have served to make equivocal current understanding of the underlying causes, assessments (diagnostic/prognostic), and treatments of osteoporosis. A number of the most salient of these have been highlighted in recent research studies, mini-reviews, and perspective journal articles [1–21].

These and other concerns have led some investigators [6,8,10,14,15,18,19] to propose one or another plausible alternative paths of investigation which might lead to a more internally self-consistent development of the field.

While each of these inconsistencies appears to pertain to a different aspect of bone fragility and treatment, these may, nevertheless, be linked in some way. Careful scrutiny of these various inconsistent studies and the interpretations underpinning them indeed reveals a factor common to all: the seemingly unqualified reliance and near-universal dependence upon DXA in vivo measurements to provide accurate, quantitative bone mass/bone mineral areal density/bone fragility determinations.