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Review Article

Follow-up bone mineral density testing: 2023 official positions of the International Society for Clinical Densitometry

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Abstract

Dual-energy X-ray absorptiometry (DXA) is the gold standard method for measuring bone mineral density (BMD) which is most strongly associated with fracture risk. BMD is therefore the basis for the World Health Organization's densitometric definition of osteoporosis. The International Society for Clinical Densitometry (ISCD) promotes best densitometry practices and its official positions reflect critical review of current evidence by domain experts. This document reports new official positions regarding follow-up DXA examinations based on a systematic review of literature published through December 2022. Adoption of official positions requires consensus agreement from an expert panel following a modified RAND protocol. Unless explicitly altered by the new position statements, prior ISCD official positions remain in force. This update reflects increased consideration of the clinical context prompting repeat examination. Follow-up DXA should be performed with pre-defined objectives when the results would have an impact on patient management. Testing intervals should be individualized according to the patient's age, sex, fracture risk and treatment history. Incident fractures and therapeutic approach are key considerations. Appropriately ordered and interpreted follow-up DXA examinations support diagnostic and therapeutic decision making, thereby contributing to excellent clinical care. Future research should address the complementary roles of clinical findings, imaging and laboratory testing to guide management.

Keywords: Osteoporosis; Bone mineral density; Dual-energy X-ray absorptiometry; Fracture risk; Aging; Monitoring.

Introduction

Bone mineral density (BMD) as measured by Dual-energy X-ray Absorptiometry (DXA) scan is strongly associated with fracture risk. A DXA-measured BMD T-score ≤ -2.5 is the WHO diagnostic criterion of osteoporosis. Follow-up BMD measurement is frequently used in managing patients at risk of fracture (with or without treatment), but practitioners are often uncertain about both the indications for repeat BMD measurement and how the findings should be interpreted. This uncertainty leads to inappropriate testing,[1] with both overuse and underuse of DXA.[2]

The International Society for Clinical Densitometry (ISCD) promotes best DXA practices, and to that end

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periodically convenes Position Development Conferences (PDC) to establish and explain its Official Positions. Topics pertinent to follow-up BMD measurement were last addressed in 2019.[3] The 2023 PDC reviewed and updated guidance on the indications for follow-up BMD measurement in clinical practice as supported by available evidence. This paper reports the process and the thinking by which the new positions were developed.

Methods

The ISCD follows a modified RAND/UCLA appropriateness method to establish its Official Positions. This has been set out in the previous ISCD executive summary.[4] The ISCD assembled a clinical task force to review the topic of Follow-up Bone Mineral Density Testing and created an expert panel to independently assess the position statements developed by the task force. Official Positions for 2023 are presented below with their respective grading which reflects quality of evidence, strength of recommendation and applicability. This grading system is also reviewed in the 2019 Executive Summary.

Key questions

The appropriate use of follow-up DXA examination was selected as a topic for review and the following list of key questions was developed:

1. Can follow-up BMD testing guide initiation of treatment in untreated patients?
2. Can follow-up BMD testing assess response to osteoporosis treatment?
3. Can follow-up BMD testing guide changes in osteoporosis treatment?
4. Can follow-up BMD testing be used to monitor bone health in patients during a planned interruption in therapy?

Literature review

A full systematic search of the English language literature was conducted in accordance with guidance document provided by the Cochrane Collaboration.[5] We incorporated search strategies and selected peer reviewed journals from previous PDC on "Repeating Measurement of Bone Mineral Density when Monitoring with Dual-energy X-ray Absorptiometry: 2019 ISCD Official Position" which included all journals from 1980 to December 2018.[6] In addition, peer-reviewed literature published between January 2019 and December 2022 was also identified from two major electronic databases: MEDLINE and EMBASE. The search strategy included following search terms: *fracture*, *fragility fracture*, *men*, *male, women, female,* search terms specific to BMD change *(including: change in BMD, decrease in BMD, increase in BMD, decrease in bone mass, increase in bone mass, decrease in bone density, increase in bone density, change in bone mass, bone density loss, and bone mass loss),* related to no treatment *(conservative management, conservative treatment, no treatment, untreated, and not treated),* and all known approved anti-osteoporotic drug interventions *(including: bisphosphonate treatment, zoledronate, zoledronic acid, pamidronic acid, pamidronate, risedronic acid, risedronate, fosamax, alendronic acid, alendronate, calcitonin, hormone replacement treatment, SERM, selective oestrogen receptor modulators, lasofoxifene, bazedoxifene, raloxifene, Prolia, denosumab, anabolic agents, anabolic therapy, teriparatide, Forteo, parathyroid hormone, abaloparatide, romosozumab, Evenity.* In addition, search strategies pertaining to treatment interruption studies were reviewed with all the drugs listed combined with following terms: *drug cessation, drug holiday, drug interruption.* The search strategy also included special circumstances, treatments, and diseases with the following terms: *glucocorticoids, aromatase inhibitors, breast cancer treatment, androgen deprivation therapy, cancer, immobilisation, rheumatoid arthritis, cystic fibrosis, chronic obstructive airways disease, short gut syndrome, bariatric surgery, diabetes, antiretroviral therapy.* BMD and behavioral outcome studies were reviewed with these terms: *behavioral change, patient education, physician understanding, patient knowledge, physician knowledge.* These terms were appropriately linked by Boolean operators such as "AND" and "OR." Relevant truncations and wildcards were also used as needed.

The retrieved articles were initially screened by title and abstract, then relevant full text articles were assessed for eligibility. Inclusion criteria were: (1) studies addressing BMD change in any female or male population, (2) published in English language, (3) patient age $\geq$ 20 year (4) studies that adequately reported BMD data regardless of the statistical method used to report BMD change (e.g., bone density in g/cm^2, percentages of mean BMD change, means $\pm$ SD change, mean $\pm$ SEM change, etc.), and (5) observational, randomized controlled trials, and post hoc analyzes. Exclusion criteria were: (1) Studies with initial sample size less than 100 (this does not exclude studies with numbers less than 100 during follow-up or after randomization), (2) Editorials, case reports, reviews, and letters, (3) Studies with unclear "BMD change" data, and (4) For duplicate reports, or duplicate original BMD data with different published study aims and results, we chose the most informative and detailed report and exclude any other report with similar data. Fig. 1 depicts the search framework and the process of selection of various studies, performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[7]

Accepted official positions and rationales

Key Question #1: Can follow-up BMD testing guide initiation of treatment in untreated patients?

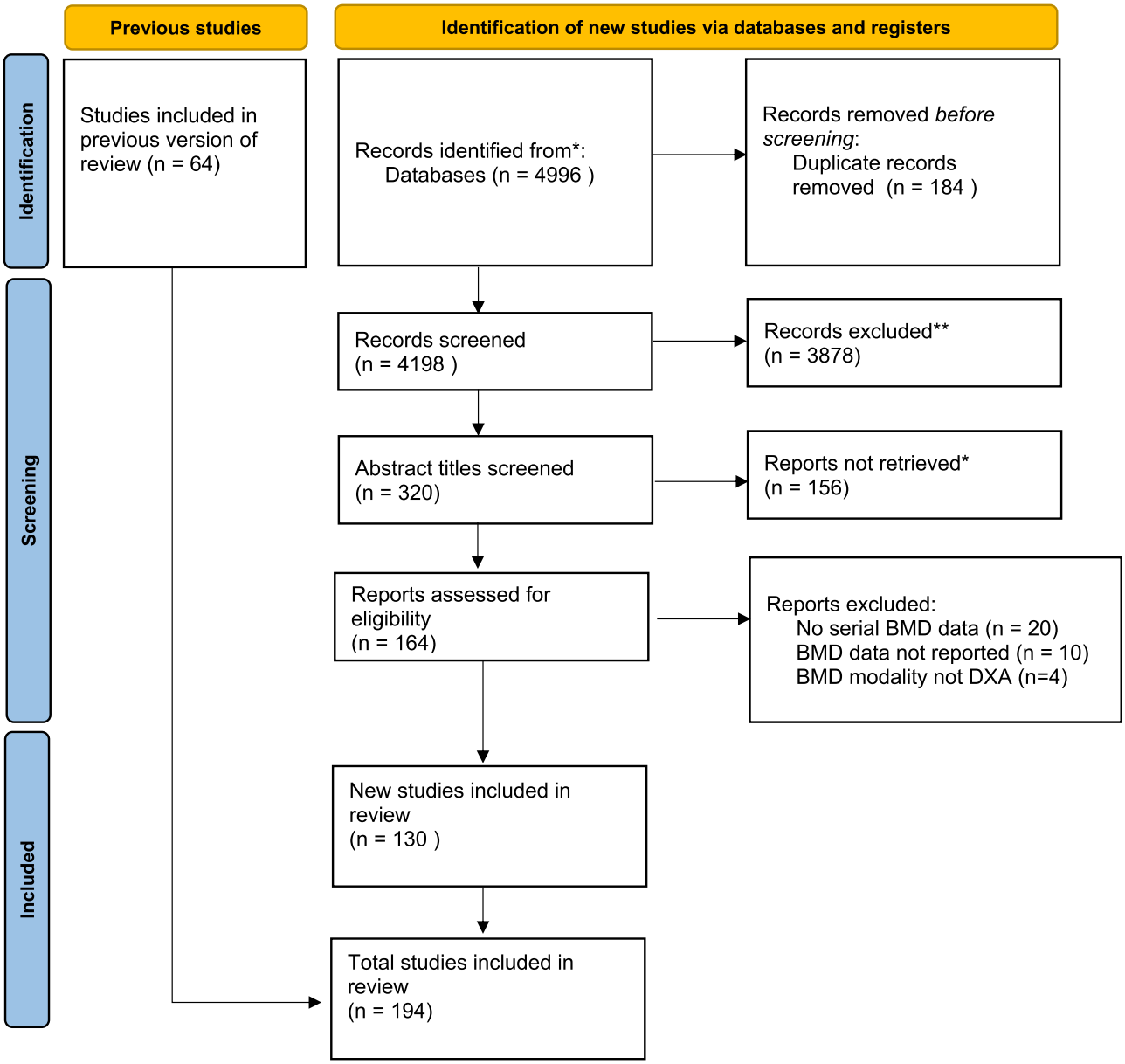

Fig. 1. PRISMA flow diagram of the systematic review.
** Studies not relevant to the current review *Does not meet inclusion criteria for study selection as described in methods.

ISCD official position

Follow-up BMD testing should be undertaken with clearly defined objectives and when the results are likely to influence patient management.
Grade: good-A-W

ISCD official position

Follow-up BMD testing should be performed if a fracture has occurred or new risk factors have developed but should not delay treatment for secondary fracture prevention.

Grade: fair-B-W

Rationale

The role of follow-up BMD testing in assessing the need for treatment remains unclear with a lack of consensus among clinicians. Since the publication of the 2019 Official Positions on monitoring BMD with DXA,[6] new studies have reported that in older community-dwelling patients, BMD remains stable over time.[8-11] Table 1 summarizes the studies evaluating BMD changes and fracture risk in untreated patients. In these populations, a baseline BMD was sufficient for predicting future hip fractures

Table 1
Summary of studies evaluating BMD changes and fractures in untreated patients

Studies	N	Participants	Age (Mean ± SD, y)	M (%)	Demographic information	Follow-up (y)	Bone site(s)	DXA (y)	Annual BMD change	Comments
Moilanen et al 2022[8]	3222[b] selected, 2695 consented, 686 completed 25 y	Population, W 100 % (OSTPRE)	53 ± 2.8	0	BMI 26.4 kg/m^2, smoking 11 %	25	FN	0, 5	FN: −0.4 % (−10.1 % over 25 y; −9.7 % in untreated, healthy subgroup)	Drop out 26.1 % (16.6 % dead; long-term healthcare institution 9.5 %)
Ensrud et al 2022[118]	3651[a]	Population (MrOS), W 89.9 %	72.3 ± 5.1	100	BMI 27.1 kg/m^2, multimorbidity score 1, fall in the last year 29.8 %	8.2	FN, TH	0, 7	FN: −0.38 ± 0.90 % TH: 0.38 ± 0.75 %; For the subgroup with fractures, FN: −0.52 ± 1.00 % TH: −0.52 ± 0.84 %	2.5 % transition to < −2.5 at Y7. Repeat BMD did not change fracture prediction.
Crandall et al 2020[119]	7419[a]	Population (WHI), W 77 %	66.1 ± 7.2	0	Height 161.2 ± 5.2 cm, weight 68.4 ± 11.5 kg	9	LS, FN, TH	0, 3	LS: 0.62 ± 1.70 % FN: −0.03 ± 1.60 % TH 0.20 ± 1.33 %	
Gourlay et al 2017[19]	6096[a]	Population (WHI), W 76.3 %	61.6 ± 6.7	0	BMI 28.3 ± 6.0 kg/m^2	13.2 ± 4.1	LS, FN, TH		Reported in Crandall et al [119]. This study reported time to treatment of osteoporosis.	Unadjusted time to treatment level by age, for FRAX with BMD group: 7.6 y. 70−74 y: 6.94 y, 75−79 y: 5.1 y. For FRAX without BMD, 65−69 y: 5.43 y, 70−74 y: 2.94 y, 75−79 y: 3.67 y
Gourlay et al. 2016[33]	5415 [a]	Population (MrOs)	73.6 ± 5.9	100	BMI 27.4 kg/m^2	8.7	LS, FN, TH	0, 2, 3, 5, 7		Men with BMD T-scores > −1.50 on a first BMD test were very unlikely to develop osteoporosis during follow-up. Additional BMD testing may be most informative in older men with T-scores ≤ −1.50
Leslie et al 2015[13]	542[b]	Population	62 ± 8	0	Excluded subjects on medications affecting BMD	6.9		0, 3.4, 6.9	For interval 1 and 2, LS: −0.6 % and −0.3 % FN: −0.5 % and −0.6	Mean FN BMD loss was stable with weak negative correlation between intervals.
Berry et al 2013[120]	802 [b]	Population (Framingham)	78.8 ± 4.5	38.6		9	FN	0, 4, 8	Annual BMD change −0.005 ± 0.01 g/cm2; −0.6 ±1.8 %	In untreated men and women of mean age 75 years, a second BMD measure after 4 years did not improve the prediction of hip or major osteoporotic fracture.
Greendale et al 2012[121]	862 [a]	Population (SWAN); multi-ethnic population, W 44.5 %, AA 28.0 %, C 13.6 %, J 13.8 %	46.7 ± 2.6	0	Mean age at FMP 51.6 ± 2.4 Y; pre-M 58 %, early peri-M 41 %; BMI 27,4; smokers 16 %	10	LS, FN	Annually	LS: **TM** W: −2.46 % **PMP** W: −1.04 % (10Y W: −10.6 %, AA −9.6 %, C −12.6 %, J −9.1 %) FN: **TM** W: −1.76 % AA: −1.42 % C: −2.17 % J: −2.13 % **PMP** W: −1.15 % (10Y W: −9.1 %)	Relatively well excluded patients on treatment. Greater loss in the trans menopause period than postmenopausal period.

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Table 1 (*Continued*)

Studies	N	Participants	Age (Mean ± SD, y)	M (%)	Demographic information	Follow-up (y)	Bone site(s)	DXA (y)	Annual BMD change	Comments
Gourlay et al 2012[122]	4957	Population (SOF), W > 99 %	≥ 67	0	Population-based listing recruitment.	15	FN	0, 2, 6, 8, 10, 16	Time for 10 % to develop osteoporosis by FN T-score: > −1.5: 15 y −1.5 to −1.99: 5 y −2.0 to −2.49: 1 y	
Cawthon et al 2012[123]	4470[a]	Population (MrOS)	≥65	100	M ≥ 65 Y, community-dwelling, able to walk without aid, not have bilateral hip replacement	4.6	FN, TH	2 times 1−3 y apart	TH Maintained Gp: + 1.3 % Expected loss Gp: −1.9 % Accelerated Gp: −6.8 %	BMD change was categorized as "accelerated" (≤−0.034 g/cm2), "expected" (between 0 and −0.034 g/cm2), or "maintained" (≥0 g/cm2). Accelerated decrease in BMD is a strong, independent risk factor for hip and other nonspine fractures in men.
Cawthon et al. 2009[124]	4720 [a]	Population (MrOS)	≥65	100	M ≥ 65 Y, community-dwelling, able to walk without aid, not have bilateral hip replacement	4.6	FN, TH, Trch, ITrch	2 times 1−3 y apart	FN: −1.72 TH: −1.82 Trch: −1.60 ITrch: −2.02	Men ≥ 85 y/o lose BMD twice as fast as ≥ 65 y/o. Low BMD, low body weight associated with higher bone loss
Berger et al 2009[125]	3635 [aorb]	Population (CaMos)	50−85[d]	39.0	Age 50−85 y; not using oral or parenteral glucocorticoids for >3 m at baseline or during the first 5 y of follow-up	7	LS, FN, TH, GT	0, 3, 5	LS (F/M): + 0.42 / + 0.56 FN (F/M): −0.26 / −0.26 TH (F/M): −0.27 / −0.25 GT (F/M): −0.12 / −0.14	
Bruyere et al. 2009[18]	1775	Placebo arm of SOTI and TROPOS trials	73.3 ± 6.1	0		3	LS(L2-L4), FN, TH	0,3	Used 3 % bone loss as a cutoff	Bone loss >3 % in 3 years is significant and may predict fracture
Zhai et al 2008[126]	955 [a]	Population, W 100 %	54.7 ± 6.0	0		15	LS, FN	2, 3, 4, 5, 6, 8, 10, 15	LS quadratic: 3.12 % FN linear: 1.67 %	
Hillier et al 2007[127]	4124 [a]	Population, SOF cohort	72.4 ± 4	0	Community dwelling women	8(6.3−9.8)	TH	2 DXA over follow-up duration	−0.59 (0.91)%	repeating a measurement of BMD up to 8 years later provides little additional value to initial measurement to predict incident fractures
Sorney-Rendu et al 2005[128]	671 [a]	Population, OFELY	62.2 ± 9	0	31−89 years of age, randomly selected from insurance data	11.2 ± 1.1	MR, DR, UDR	Annually	MR: −0.30 ± 0.76 % DR: −0.55 ± 0.79 % UDR: −0.40 ± 0.96 %	The rate of bone loss in postmenopausal women is significantly associated with fracture risk independently of other well-known predictors such as BMD and history of fractures.
Nguyen et al 2005[129]	966 [b]	Population (Dubbo)	69.9 ± 6.7	0		10.7(2.7−13)	LS, FN	2.7(0.8−4.2)	Annual BMD change in any Fx/ hip Fx / no Fx at LS:−0.3± 2.8 %/−1.1± 2.8 % /−0.1± 2.8 %; at FN: −1.4± 4.1 % /−2.1±4.2 % / −0.8± 2.9 %	Bone loss at FN is a predictor of fracture risk in elderly women, independent of baseline BMD and age
Melton III et al 2000[130]	304 [a]	Population, W 100 %	60 (30−94) [c]	0	Age stratified random sample from medical records of Rochester Epidemiology project.	7.9 (0−16.6) [c]	FN	Every 3 y	FN: −1.0 (−10.0− + 13.4)[d]; not statistically significant difference over life	Rates of loss in non-HRT: Slow (<−1.5 %/y): 59 % Intermediate (−1.5 to −3.0 %/y): 33 % Fast (>−3.0 %): 8 %. Correlation of baseline with FN BMD at 16 years was 0.83

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Table 1 (*Continued*)

Studies	N	Participants	Age (Mean ± SD, y)	M (%)	Demographic information	Follow-up (y)	Bone site(s)	DXA (y)	Annual BMD change	Comments
Huang et al 1998[131]	>500 [a]	Population (HOS)	62.9 ± 5.1	0		11	DR, LS	2–3, 8–11	DR: −1.04 % LS: + 0.48 %	Recent bone mass measurements predict vertebral fractures only slightly better compared to measurements made 10 y earlier.
Hansen et al 1995[132]	178 [b](1977) 70 [b](1983), 129 completed	Population	50 ± 2	0	Danish females, BMI 24.2 kg/m^2	15	LS, FN, TH, GT, mean of both femurs	0, 8, 11, 14, 16	DF: −1 % FN: −1 % LS and Greater Troch: non-significant	
Jones et al 1994[133]	626 [b]	Population (Dubbo osteoporosis study)	≥60	61.5	Dubbo, NSW, AU	4	LS, FN	0, 2.5	LS: F: −0.04 % M: + 0.56 % FN: F: −0.96 % M: 0.82 %	Excluded all subjects who were also taking drugs that may affect bone. Low number of subjects > 75 y
Gärdsell et al 1991[134]	366	Population	20–70	0		15	Forearm BMC by SPA	0, 14.6 ± 2.2	40–49 y: nFx: −0.72 to −1.56 % Fx: + 0.01 to −1.98 % 50–59 y: nFx: + 0.20 % to −2.21 %/ Fx: + 0.4 to −2.31 % 60 −69 y: nFx: + 0.95 to −1.26 % Fx: + 1.44 to −2.00 %	Rate of bone loss did not differ between those with or without fracture. Initial bone mass was the best predictor

[a]= Hologic®
[b]= Lunar®
[c]median (range)
[d]range

AA = African American, AI = aromatase inhibitor; AU = Australia; BMC = Bone mass content; BMI = Body mass index; C = Chinese; CaMos = Canadian Multicentre Osteoporosis study; CI = confidence interval; DR = distal radius; F = female; FMP = final menstrual period; FN = femoral neck; FU = follow=up; Fx = fractured group; Gp = group; GT = greater trochanter; HOS = Hawaii Osteoporosis study; ITrch = intertrochanter; J = Japanese; LS = lumbar spine; M = male; MrOS = Osteoporotic Fractures in Men study; m = month; nFx = non-fractured group; NSW = New South Wales; OP = osteoporosis; OSTPRE = Kuopio Osteoporosis Risk Factor and Prevention study; peri-M = perimenopause; PMP = post menopause; pre-M = pre-menopause; PTM = pre-trans menopause (5− 1Y prior to FMP); SD = standard deviation; SES = socioeconomic status; SOF = Study of Osteoporotic Fractures; SPA = Single photon absorptiometry; SWAN = Study of Women's Health Across the Nation; TH = total hip; TM = trans menopause (1 y before and 2 y after FMP); Trch = trochanter; W = white; WHI = Women's Health Initiative study; y = year(s)

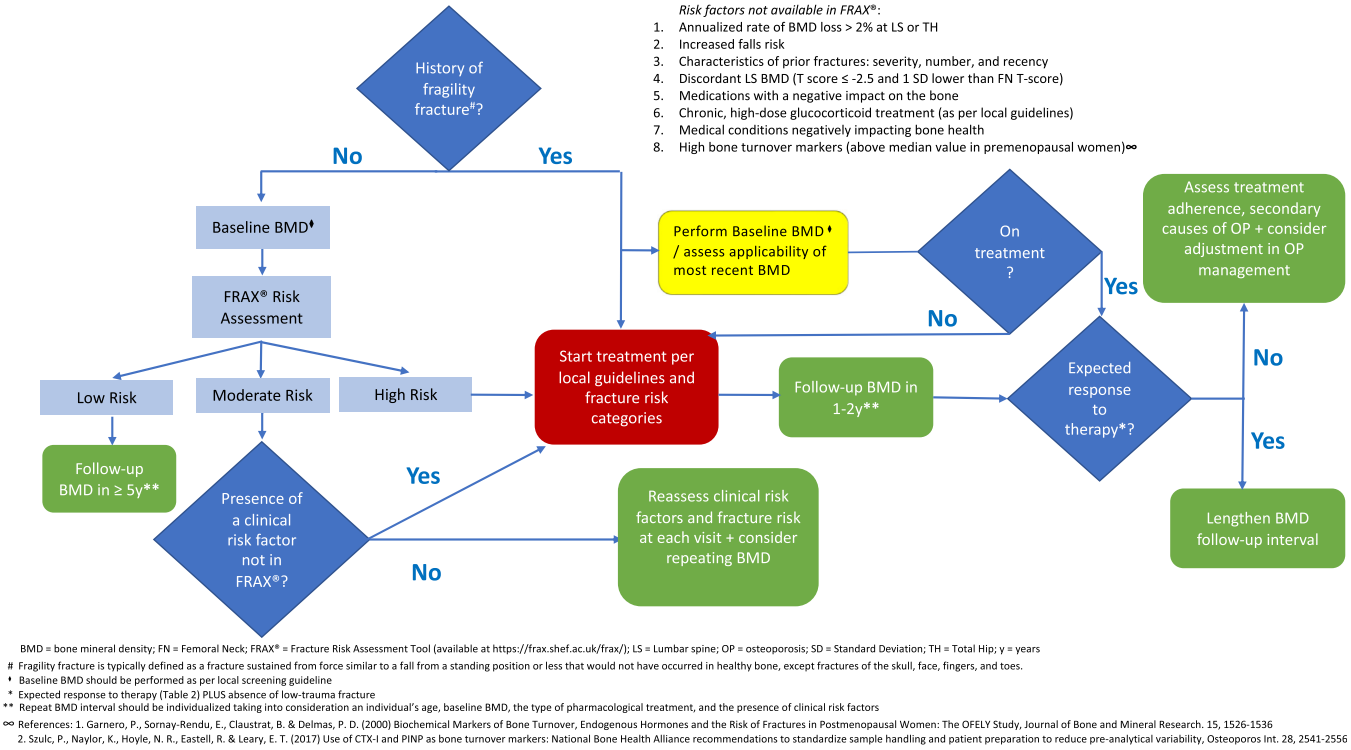

Fig. 2. Follow-up BMD testing algorithm.

and a repeat BMD within 3 to 7 years did not provide additional information for fracture prediction compared to using the baseline study alone.[9,10,12]

Measurement error has also been cited as a concern with serial BMD comparisons with recent literature showing that in instances where there is initial BMD decline, serial studies at short intervals often show regression of the results toward the baseline values.[13] Therefore, to avoid the risk of measurement errors, a quality follow-up BMD scan is essential for clinicians to make clinical decisions based on genuine changes or stability of BMD.[4,14]

Repeating DXA in older patients unlikely to experience a further decline in BMD may have little value in guiding treatment decisions.[15] Conversely, an individualized approach to repeating BMD should be considered in those at risk of accelerated bone loss. For example, those with premature ovarian insufficiency, gastric bypass, other malabsorption disease states, and those receiving glucocorticoids, aromatase inhibitors, or androgen deprivation therapy are candidates for follow-up testing (Supplementary Table 1–3). Previous studies have also shown there may be a subset of patients who are at a risk of more rapid bone loss with older age, low body weight, and a lower baseline BMD being cited as the main risk factors.[16-18]

Systematically repeating BMD without clear objectives may not be useful and contributes to low value care.[2] We present a schematic diagram on the suggested role of follow-up BMD testing in patients (Fig. 2). The ordering health care provider needs to consider the goal of a follow-up BMD testing in each patient. If the aim of a follow-up DXA in an individual patient is fracture prevention, the interval for BMD repetition can be recommended on the baseline fracture risk of the patient.[19,20] BMD is just one of the criteria considered in most fracture risk assessment tools and its strength in the prediction of fractures varies according to each individual's clinical context. Established clinical risk factors for fracture development include those that have been widely used in risk calculators such as FRAX.[21] FRAX-based intervention thresholds vary between countries according to country-specific fracture data, life expectancy and economic assumptions.[22-24] A recent study using the Manitoba DXA data estimated the time needed for 10 % of a population to cross an intervention threshold as a function of baseline fracture risk.[25] In the setting of unchanged clinical risk factors, the 10 % transition time was 2.7–3 years for those with fracture risks initially at 75–99 % of threshold while 10 % transition time for those initially at < 25 % of threshold may exceed 15 years. Furthermore, changes in clinical risk factors had a substantial impact on transition times. FRAX may underestimate fracture probability in patients with risk factors such as history of falls,[26] multiple or recent fractures,[27] lumbar spine (LS) BMD much lower than femoral neck (FN) BMD,[28] high-dose glucocorticoid exposure (prednisolone > 7.5 mg / day or equivalent),[29] and history of diabetes

mellitus.[30] DXA availability varies by country and in places where there is limited access, use of clinical risk factors to assess treatment need has been shown to be of value.[31]

Clinicians also repeat DXA to assess the development of osteoporosis by BMD criteria. Most studies agree that age and baseline BMD are the strongest factors for determining the interval of follow-up BMD assessment in this setting. In healthier younger patients with low-risk osteopenia (BMD T score −1.0 to −1.4), the development of osteoporosis by BMD criteria may take up to five to ten years.[20,32] However, BMD testing cannot identify every individual who will have a future fragility fracture. In fact, longitudinal studies have confirmed that most patients who sustain a hip or clinical vertebral fracture do not have pre-existing osteoporosis diagnosed by BMD.[33,34] Thus, as discussed earlier, other screening approaches to fracture risk assessments should be continued as part of optimal bone health management.

It is important to highlight that these recommendations are limited to healthy, community-dwelling older individuals with and without a history of fractures, as the majority of the studies (above) were conducted in this group. In patients who have sustained a fracture or developed new risk factors, repeating a BMD may support a change in management.

An incident fracture in untreated patients should trigger reassessment of the need for pharmacotherapy and a search for new risk factors for poor bone health. There are limited studies assessing the utility of a follow-up BMD in those who have fractured; however, most studies are consistent in the finding that patients who undergo DXA in the setting of a fragility fracture are more likely to commence treatment[35] and have a lower risk of refracture.[36] Patients' understanding of their BMD results and beliefs in treatment effects are also strongly associated with treatment initiation and persistence.[37]

There may be clinical scenarios where patients would benefit from a shorter interval between BMD testing. Examples include the use of certain medications such as glucocorticoids, aromatase inhibitors, androgen deprivation therapy, and osteoanabolic therapies, medical disorders such as malabsorption and severe systemic inflammatory diseases, and other conditions such as prolonged immobilization, bariatric surgery, premature and surgical menopause. As stated in the past ISCD Official Positions, patients at risk for more rapid bone loss such as glucocorticoid exposure, aromatase inhibitors, and androgen deprivation therapy should have an earlier follow-up BMD testing to assess the development of osteoporosis.[3] A list of relevant studies can be found in Supplementary Table 2.

Discussions of various conditions causing secondary osteoporosis and the associated BMD changes are beyond the scope of this PDC. However, the task force would like to highlight the importance of identifying secondary osteoporosis. In particular, certain cancer patients and survivors may be at risk for short or long-term increased risk of fractures. These could be due to active treatments (e.g., aromatase inhibitors, antiandrogens, or gonadotropin-releasing hormone agonists or chemotherapy induced ovarian failure).[38] Women with breast cancer, and especially premenopausal women undergoing ovarian function suppression with gonadotropin releasing hormone agonist (GnRH agonist) in combination with aromatase inhibitors, are expected to have the largest decrease in BMD, with BMD loss in the LS estimated to be as high as 9.3 % in the first year and 13.6 % in the second year[39] (Supplementary Table 1). In men with prostate cancer undergoing androgen deprivation therapy, expected BMD loss has been variable, depending on age and duration of the treatment, with highest losses reported at 1 year of 5.77 % in the LS and 5.55 % in the FN[40,41] (Supplementary Table 3). Data are still limited regarding the appropriate interval between bone density evaluations among patients with cancer and current expert opinion suggests monitoring at intervals based on expected bone loss according to the specific therapy.[38]

Key Question #2*:* Can follow-up BMD assess response to treatment?

ISCD official position

Follow-up BMD testing can aid in monitoring response to therapy.
Grade: good-B-W

ISCD official position

Repeat BMD testing intervals must be individualized considering an individual's age, baseline BMD, the type of pharmacological treatment and the presence of clinical risk factors which are associated with bone loss
Grade: good-B-W

ISCD official position

Shorter intervals between BMD testing may be indicated in the presence of factors associated with rapid change in bone mineral density. Examples include the use of certain medications such as glucocorticoids, aromatase inhibitors, androgen deprivation therapy, and osteoanabolic therapies, medical disorders such as malabsorption and severe systemic inflammatory diseases, and other conditions such as prolonged immobilization, bariatric surgery, and premature menopause.

Grade: fair-A-W

Rationale

The goal of osteoporosis treatment should be an acceptably low risk of fracture. Treatment decisions should be guided by processes that will help patients achieve the goal, balanced with the inherent drawbacks of pharmacotherapy side effects. As the most widely used and well-studied tool, BMD response is an attractive

option that is frequently employed assess treatment efficacy or lack thereof and/or adherence to medication. However, in practice this is complex and the role of a follow-up DXA scan in monitoring therapy has been long debated with a lack of clear consensus.

Evidence regarding whether follow-up BMD measurement while on therapy can predict outcomes is mixed.[42-47] No specifically designed trial has tested whether monitoring treated patients with BMD would improve fracture outcomes, so most evidence is observational.

Changes in BMD are not always consistently correlated with fracture risk. This is demonstrated in studies on oral bisphosphonate treatments which show that decreases in BMD may not reliably predict fracture risk.[46,48] Most recently, another study demonstrated that among postmenopausal women highly adherent to antiresorptive therapy, even when there was apparent BMD loss, there is poor (less than 1 %) reproducibility of the BMD loss in individuals for 3 − 3.2 years.[47] This could be due to the presence of fracture prevention benefit that is multifaceted, such as improvement in bone microstructure and quality which is not always reflected as change in bone density.

While the changes in BMD do not account for all of the fracture risk reduction seen with therapy, several post hoc studies and large meta regression studies have shown that larger increases in BMD on therapy are associated with greater fracture risk reduction.[45,49,50] Increasingly, BMD T-score attainment has also been considered as a therapeutic target for the treatment of osteoporosis. This is supported by extension studies of large clinical trials demonstrating the reduction of fracture risk with larger BMD gains with antiresorptive therapies.[51-54] In these studies, total hip (TH) or FN T-scores achieved were associated with subsequent nonvertebral and vertebral fracture rates and the relationships were independent of the treatment received. TH T-scores exceeding −2.0 were associated with reduced fracture risk and TH was a better monitoring site than FN and LS.[53-55]

A recent large meta-regression of 38 placebo-controlled trials evaluating 19 therapeutic agents also concluded that TH BMD gain on treatment is associated with fracture risk reduction.[49] However, statistical analysis based on summary data and those based on individual data have been shown to produce different results.[56,57] Thus, these group level summary statistics (as opposed to individual patient data) model trial level associations and may not be reflective of the association at the individual patient level. This implies that the results of these large meta-regressions may not be applicable to the individual patient as variables in each individual are not sufficiently reflected in large statistical calculation.

A predefined monitoring strategy for patients on pharmacological therapy should take into consideration patient's clinical factors and the class of pharmacotherapy used (Fig. 2) as well as a clear therapeutic objective. Discussion should also be made with patients in anticipation of scenarios when treatment changes are required to avoid treatment inertia.[58] The various fracture preventing medications evoke characteristic BMD responses. Treatment strategies that use anabolic therapies prior to other anti-resorptive treatments are associated with the largest BMD increases.[59-64] In these patients, BMD increases in the LS beyond the LSC can be seen as early 6 months and a larger increase is expected at 12 months compared to those undergoing oral antiresorptive therapy. The more potent anti-resorptive therapies such as denosumab and zoledronic acid have also been demonstrated to show larger BMD increases.[65-68] Oral bisphosphonates are associated with an improvement in LS BMD but are unlikely to result in a change in TH BMD. Table 2 depicts a summary of BMD changes at different skeletal sites according to the treatment used.

Earlier BMD remeasurement may be beneficial in those in whom accelerated bone loss may occur; these include those on higher-dose glucocorticoid therapy, aromatase inhibitors and androgen deprivation therapies. In addition, medical conditions such as malabsorption,[69-71] bariatric surgery,[72-74] immobilization and spinal cord injury[75-77] are associated with greater bone loss. The occurrence of fractures while on treatment should prompt a review of treatment strategy which includes adherence and therapeutic choices appropriate for level of fracture risk.

In summary, BMD monitoring intervals should be individualized based on the class of treatment and individual risk factors. Follow-up BMD can be considered as soon as 6 months after initiation of anabolic therapy and 1−2 years after other osteoporosis medications if the result will either: a) change treatment or b) improve adherence. In patients at high / very high fracture risk on a potent anti-resorptive, earlier BMD testing can be considered to ensure treatment response is as expected, allowing earlier intervention if required. An individual patient's clinical fracture risk should also be included in decision making of when to perform the follow-up BMD. Shorter intervals (annual) can be considered for those who are taking pharmacotherapies with a known association with impaired bone health, such as high dose glucocorticoids, aromatase inhibitors and androgen deprivation therapy.

Key Question #3: Can follow-up BMD guide changes in osteoporosis management?

ISCD official position

If changes in BMD are outside the expected range for an individual patient and scan quality has been confirmed, this should prompt re-evaluation of the patient and plan of care.

Grade: fair-B-W

Rationale

BMD responses may vary according to each patient's clinical status and the type of therapy instituted. It is

Table 2
BMD change in clinical trials of osteoporosis medications

Drug Class	% Increase in LS BMD	% Increase in FN BMD	% Increase in total TH BMD
Oral Bisphosphonate[a,78-89,135,136]	3–8 %	1–3 %	2–3 %
IV Zoledronic Acid[a,66,137-139]	5–7 %	2–4 %	2–5 %
Raloxifene[140]	2–3 %	1 %	1 %
HRT[b141-144]	3–7 %	N/A	1–3 %
Denosumab[a,60,64,68,145-150]	5–9 %	1–4 %	2–5 %
Teriparatide[64,151-155]	4–11 % (1–2 y)	1–3 % (1 –2 y)	2–3 % (1–2 y)
Romosozumab[60,61,90]	13–17 % (1 y)	5–6 % (1 y)	6–8 % (1 y)

BMD = bone mineral density, FN = femoral neck, TH = total hip, LS = lumbar spine, NA = not available, HRT = hormone replacement therapy.
[a]Percent increase in BMD includes clinical trials with antiresorptive durations of 1–3 years.
[b]Percent increase in BMD includes clinical trials with HRT duration of 3–8 years.

important to have an adequate understanding of the underlying fracture risk on an individual commencing osteoporosis therapy and the expected BMD gains on different classes of treatment. We have summarized the current expected BMD gains based on past studies in Table 2. There are substantial limitations in summarizing findings from different studies due to study heterogeneity. Table 2 offers simplified information to guide the health care provider and patients on whether the follow-up BMD results are exhibiting improvements that are in keeping with those documented in the literature. For example, patients on oral bisphosphonates without secondary osteoporosis risk factors are expected to have an increase in BMD of 2–3 % at the TH within treatment duration of 1–3 years,[78-89] while those on potent dual agent therapy or romosozumab can expect an increase of 6–8 % in their TH BMD scores within 1 year in the absence of new secondary risk factors.[60,61,90] No changes in BMD may sometimes be perceived as inadequate treatment response although clinical trials have shown that stability of BMD on therapy is associated with a reduction in fracture risk. In fact, for patients receiving bisphosphonates, it is expected that BMD may increase and then remain stable.[51,91,92] With this in mind, clinicians should plan on repeating BMD in light of the treatment strategy.

Due to the complexity of individual risk factors for fractures, solely relying on BMD changes may be insufficient to monitor therapy. Aside from BMD changes, the following factors have been suggested as important elements of assessing treatment adequacy[93,94]: 1) new vertebral fractures on imaging, 2) more than 2 cm loss in prospective measured height, and 3) new clinical non vertebral fracture. Bone turnover markers (BTM) have also been proposed as a means of assessing response to therapy; however, there may not be universal availability of this service depending on local laboratory capabilities and access through individual insurance or health care reimbursement. Furthermore, there are potential significant intra-patient variability, biologic variability (age, sex, body mass index, circadian rhythm) and poor standardization of most assays which can significantly impact the interpretation of results.[95,96]

Another reason for repeating a BMD is to encourage proactive attitude towards bone health, consideration of treatment commencement and adherence to treatment.[97,98] Repeating BMD may also trigger switching of treatment if there is an observed interval reduction in BMD. A recent study showed that there was an increase in medication possession rates among patients with a decline in mid-treatment DXA-BMD. There were also higher rates of treatment switches in those having observed interval reduction in BMD.[42,47]

Key Question #4*:* Can repeat BMD testing be used to monitor patients during a planned interruption in osteoporosis treatment?

ISCD official position

Repeat BMD testing should be used to monitor individuals prior to a temporary cessation of bisphosphonate therapy and during the period of planned interruption of treatment.

Grade: Fair-B-W

Rationale

When planning a temporary interruption in osteoporosis treatment, a BMD measurement prior to the therapeutic pause can serve as a new baseline and assist in monitoring of bone loss. Significant BMD loss may occur within 1 year following cessation of some osteoporosis therapies. Repeat BMD testing during a planned interruption can guide future treatment decisions – resumption of therapy or continuation of planned interruption.

Several guidelines are available to aid clinician's decision-making on planned interruption in a patient's osteoporosis treatment.[99-101] These guidelines recommend that fracture risk should be re-assessed after 5 years of oral or 3 years of intravenous bisphosphonate use. The guidelines further recommend considering a planned interruption in osteoporosis treatment in patients with low fracture risk, defined as a hip T-score > −2.5, absence of fracture while on treatment and lack of other clinical risk factors for bone loss/fracture. Under this concept of "treat-to-target" for osteoporosis, follow-up BMD testing should be performed 2−3 years following discontinuation of treatment, at which point, BMD and FRAX risk assessment would guide continued monitoring or resumption of treatment.

Evidence for guidance on planned discontinuation of bisphosphonate treatment largely emanated from studies in postmenopausal women. In the Fracture Intervention Trial Long-Term Extension (FLEX) Trial of 10 versus 5 years of alendronate,[102] women with T-score ≤ −2.5 in the FN had a higher risk of nonvertebral fractures, however the overall number of fractures in the study was low. In the analysis of the placebo arm of the FLEX study, older age and lower hip BMD (TH and FN) at the time of discontinuation were significantly related to fracture risk. Follow-up BMD measurement and BTM at 1 year were not associated with fracture risk.[103] In the Health Outcomes and Reduced Incidence with Zoledronic acid Once Yearly-Pivotal Fracture Trial (HORIZON-PFT) 3-year extension study, FN T-score < −2.5, TH T score < −2.5 and recent incidence of fracture were significant predictors of fracture occurrence after interruption of Zoledronic acid therapy.[104] In the second extension of the HORIZON-PFT trial, postmenopausal women treated with Zoledronate for 6 years or 9 years showed no significant difference in efficacy and incidence of fractures.[92]

A model of BMD changes following discontinuation of alendronate developed from data in relatively healthy postmenopausal women suggests that baseline BMD at the time of therapeutic pause may guide timing of the next BMD measurement.[105] As bisphosphonates vary in their binding affinity for bone, a quicker offset of drug effect in alendronate is marked by an earlier decline in BMD compared to zoledronic acid.[106] Patients exposed to risedronate, a bisphosphonate with shortest half-life, are likely to have a decrease in BMD requiring even an earlier reassessment than alendronate and zoledronic acid.[107] Table 3 summarizes the studies on bone loss following interruption of bisphosphonate therapy with the cumulative evidence suggesting that a repeat BMD measurement 1−2 years after discontinuation may be indicated in most patients. In addition, an important factor is the coexistence or occurrence of other clinical risk factors for bone loss in an individual (for example, malabsorption, medications associated with rapid bone loss or a new fragility fracture) that may warrant an earlier assessment of BMD. Therefore, repeat BMD testing intervals should be individualized according to an individual's age and BMD at time of drug interruption, the duration and type of bisphosphonate used in their treatment and the presence of other clinical risk factors.

There are few studies on the role of BMD monitoring to guide treatment decisions during a bisphosphonate drug interruption. While baseline BMD value and age have been shown to be associated with the incidence of fractures in those undergoing bisphosphonate drug interruption, 1-year BMD changes or BTM have not been shown to be associated with fracture risk.[108] Although annualized BMD change may not be helpful in all patients, more frequent BMD monitoring is of value in patients in whom a greater than average decline in BMD is expected over time.

Denosumab discontinuation is characterized by an increase in bone turnover with rapid bone loss within 12 months[109,110] resulting in a rapid reversal of antifracture efficacy and greater risk of subsequent fractures. Therefore, unlike bisphosphonates, a planned interruption is not recommended following denosumab therapy. BMD measurement at the time of discontinuation of denosumab treatment or prior to the transition to other drug classes is meaningful and indicated, similar to that in bisphosphonate treatment discontinuation. In the event of an unplanned or unexpected interruption of denosumab treatment, BMD measurement following denosumab discontinuation is warranted as soon as possible.

Table 4 and Supplementary Table 4 summarize the studies on bone loss following denosumab discontinuation and transition to other drug classes. The number of large prospective studies monitoring patients post denosumab transition therapy and examining the impact of this discontinuation on BMD and fracture incidence are limited. Available studies suggest that the risk of multiple fractures may increase in those with longer duration of denosumab treatment,[111] prior vertebral fractures, longer duration of time off-treatment and higher percentage of annualized TH BMD bone loss.[112] A large observational study with BMD monitoring over 8 years[113] found that the majority of BMD loss occurred within 24 months of the last denosumab administration. However, patients who sustained vertebral fractures post-denosumab transition were not necessarily those who gained the most BMD during treatment or lost the most BMD post-treatment.[113] Longer treatment periods were also found to be associated with more bone loss.[114]

Therefore, further studies are needed to elucidate the optimal treatment transition from denosumab therapy, predictors of bone loss and fractures and the role of BMD monitoring in these patients. This task force concurs with recent recommendation from the European Calcified Tissue Society[115] suggesting the importance of individualizing follow-up according to duration of denosumab exposure with the use of BTM and close monitoring for vertebral fractures.

Table 3
Studies on bone loss post interruption of bisphosphonate

Studies	N	Participants	Age (y)	M (%)	Intervention	Comparison	Duration of intervention	Follow-up	BMD change (%)	Comments
Saag et al. 2021[156]	175 RCT	PMPW ≥ 5 y after BIS ≥ 3 y (ALN only in final y)	Mean 66.9–67.8	0	ALN	Pbo	≥ 3 of 4 y then 1 y of ALN or Pbo	1 y	1 y LS: **ALN**: 1.29 **Pbo**: −0.36 FN: **ALN**: −0.08 **Pbo**: −1.26 TH: **ALN**: 0.46 **Pbo**: −1.44	Greater FN BMD loss with higher BTM levels at 3 − 6 m. younger age and higher LS BMD at baseline were associated with greater LS BMD loss at 12 months
Kim et al. 2019[157]	FLEX: 6459 OBS HORIZON-PFT: 7765 OBS	PMPW randomized to Pbo after core trial (FLEX: ≥ 3 y of ALN; or HORIZON-PFT: 3 y of ZOL)	Mean 73.7 − 75.5	0	Pbo	none	3 y	3 y	3 y LS: **HORIZON-PFT**: + 1.5 **FLEX**: + 0.9 FN: **HORIZON-PFT**: −0.5 **FLEX**: −1.2 TH: **HORIZON-PFT**: −1.3 **FLEX**: −2.4	24.3 % of FLEX and 15.1 % of HOR with TH BMD loss greater than LSC at 3 Y. Oral alendronate quicker offset than ZOL (despite longer duration of treatment a 5 years)
Black et al. 2015[158]	190 RCT	PMPW with osteoporosis treated with ZOL 6 y and 3 y Pbo or Cont. ZOL for 9 years	78.1	0	ZOL 9 y (Z9)	ZOL 6 y and Pbo 3 y (Z6P3)	3 y	3 y	Z6P3/Z9 FN: Y7: −1.24/ −0.78 Y8: −0.88/ 0.00 Y9: −1.17 / −1.11 TH: Y7: −0.83 / −0.28 Y8: −0.14 / −1.06 Y9: −1.13 / −0.54	Almost all patients with 6 annual Zol can stop medications for up to 3 years with apparent maintenance of benefits. There were no significant differences in fracture incidence by treatment.
McNabb et al. 2013[159]	406 OBS	PMPW after average 5 y of ALN (FLEX)	73.6	0	Pbo	None	5 y	none	5 y LS: + 1.27 FN: −1.69 TH: −3.62	Normal distribution of 5 Y BMD changes. Age, smoking, low BMI were associated with BMD loss. No combination of risk factors can predict 5Y BMD changes
Black et al. 2006[160]	1099 (ALN 329 + 333; Pbo 437) RCT	PMPW after 5 y of ALN (FLEX)	72.7–73.7	0	Cont. ALN 5 mg/d or 10 mg/d	Pbo	5 y	none	5 y ALN/Pbo LS: + 5.26 / + 1.52 FN: + 0.46 / −1.48 TH: −1.02 / −3.38 Tch: −0.08 / −3.25 FA: −1.19 / −3.21 TB: + 1.01 / −0.27	Discont. ALN for up to 5 years does not appear to significantly increase fracture risk.
Ensrud et al. 2004[161]	1099 (ALN 329 + 333; Pbo 437) RCT	PMPW after 5 y of ALN (FLEX)	72.7–73.7	0	Cont. ALN 5 mg/d or 10 mg/d	Pbo	3 y	none	3 y ALN/Pbo LS: + 3.5 / + 0.97 FN: + 0.57 / −1.1 TH: −0.34 / −2.38 Tch: + 0.71 / −1.9 FA: −0.90 / −2.31 TB: + 0.62 / −0.31	Discont. ALN did not result in accelerated bone loss.

(*continued on next page*)

Table 3 (*Continued*)

Studies	N	Participants	Age (y)	M (%)	Intervention	Comparison	Duration of intervention	Follow-up	BMD change (%)	Comments
Mortensen et al. 1998[162]	111 (Pbo/RIS cy/RIS dly 36/38/37) ^{Obs}	PMPW with normal LS BMD previously randomized to RIS or Pbo	51.2–52.1	0	Previous intervention RIS 5mg cyclic (RIS cy), RIS 5 mg daily (RIS dly)	Compare among group	2 y	1 y	LS 3 y: Pbo: −5.6 RIS cy: −3.4 RIS dly: −2.3	After stopping RIS, bone mass decreased within 1y.

^{Obs} = observational study
^{RCT} = randomized control trial
BIS = bisphosphonate; BMD = bone mineral density; C = calcitriol; Cont. = continue; Discont. = discontinue/discontinuation; FA = forearm; FLEX = Fracture Intervention Trial Long−term Extension; FN = femoral neck; FU = follow=up; IQR = interquartile range; H = hormonal replacement therapy; HORIZON-PFT = Health Outcomes and Reduced Incidence with Zoledronic Acid Once Yearly−Pivotal Fracture Trial Extension; IV = intravenous; LBM = low bone mass; LS = lumbar spine; M = male; mg = milligram; mg/d = milligram/day; m = month(s); Pbo = placebo; PMPW = postmenopausal women; pre−MW = pre−menopausal women; RIS = risedronate; SD = standard deviation; TB = total body; Tch = trochanter; TH = total hip; wk = week(s); y = year(s); Y = year; ZOL = zoledronic acid

In patients transitioning from osteoanabolic therapies, there are a limited number of studies on BMD monitoring and fracture risks following cessation of pharmacological treatments. We summarized the larger studies in Supplementary Table 5. BMD gains achieved from non-bisphosphonate therapies are lost rapidly months following treatment discontinuation, with variable extent and speed, depending on the type of anabolic treatment and other concurrent clinical risk factors. In the placebo arm of a phase 2 study with romosozumab, BMD gains during therapy at the hip were rapidly lost within 1 year.[116] In a study looking at Parathyroid hormone (PTH) 1−84 treatment in post-menopausal women, patients on placebo post PTH 1−84 treatment were observed to have partial reversal of BMD gains at the LS within a year. TH and FN BMD had no changes post PTH 1−84 in the placebo group.[117]

Discussion

This document has summarized the 2023 Official Positions on the role of follow-up BMD testing. Similar to the 2019 ISCD Official Positions on repeating BMD,[6] we have reviewed current evidence on the role of follow-up BMD in initiating treatment, assessing treatment response, guiding changes in treatment and monitoring patients in a period of planned osteoporosis treatment interruption. What is new in the 2023 Official Positions is the emphasis of pre-defined objectives in repeating a BMD to ensure high value care as well as highlighting the importance of avoiding treatment delays in those who have sustained fractures. We further augment the Official Positions by underscoring the consideration of clinical context prompting repeat examination and recommending individualized BMD intervals according to patient's risk factors and expected BMD gains on the selected pharmacological treatment. The task force acknowledges that with the emphasis on individualizing follow up BMD intervals for patients, this may invariably make decision on repeating BMD more complex for the medical provider as there is no "one size fits all". However, as we have outlined in this document, growing evidence and understanding of different clinical circumstances, availability of new treatments has significant implications on changes in BMD and requires medical providers to individualize BMD interval recommendation. We also affirm the role of repeat BMD prior to - bisphosphonate therapy cessation as well as during the period of treatment interruption.

Future directions

With the evolution of the concept of goal directed treatment in osteoporosis, more evidence is needed to better inform clinicians on the role of BMD targets in reducing fracture risk. Studies on expected BMD gains on various osteoporosis therapies, role of BTM bone

Table 4
Studies on bone loss post denosumab transition (study population > 100)

Studies	N	Participants	rowsep) ='1' or string (ancestor:: ce:table/ @rowsep) ='1')"=1? ^!"number (boolean (./@morer-	ows))"=^!"- string(./@mor- erows)"? ^!"count(../fol- lowing-sibling:: row)"[?-} skip143]>Age (y)	M (%)	Intervention	Comparison	Duration of intervention	Follow-up
BMD change (%)	Comments								
Cosman et al. 2022[163]	802 RCT	PMPW with (FREEDOM trial and its open-label extension)	Mean 71.3– 72.4		Dmab	Pbo	≥ 2 doses of Dmab or Pbo	11.4 m	No BMD data	Risk of MVF after Dmab Discont. increases with longer duration of Dmab therapy.
Everts-Graber et al. 2022[114]	282OBS	PMPW with Dmab 2.5–8 y	Mean 65–66	0	1–2 ZOL post Dmab dependent on BTM	none	Dmab 2.5 –8 y	1–2 y	(18–30 m) Short Dmab: LS –3.1 %, TH –2.0 %, FN –1.4 %, Medium Dmab: LS –5.6 %, TH –3.5 %, FN –3.4 %, Long Dmab: LS –5.0 %, TH –3.2 %, FN –2.6 %	More patients in longer Dmab group required 2 ZOL infusions. Patients on medium and long term Dmab lost more BMD than short term Dmab
Everts-Graber et al. 2020[164]	120 OBS	PMPW with 2–5 y Dmab	Mean 65.6	0	1 ZOL infusion	none	Single dose	1–4 y after Dmab	1–4 y (Ave 29m) LS: –3.3 FN: –1.5 TH: –2.2	Percentage of BMD retained post ZOL was 66 % at LS, 49 % at TH, 57 % at FN.
Bone et al. 2011[109]	256RCT	PMPW	Mean 59.1	0	Denosumab 24 m followed by Pbo 24m	Pbo 48 m	24 m	48 m	Denosumab on treatment 24m: LS + 6.4 %, TH + 3.6 %, 1/ 3 Rad + 1.4 %. D -> P 24m: decreased at all sites, greatest decrease 24–36m.	Final BMD at 48 m correlated with BMD at month 0 in Dmab to Pbo. BMD remains above that of participants with placebo for 48 months.

Obs = observational study
RCT = randomized control trial
BMD = bone mineral density; Discont. = discontinue/discontinuation; Dmab = denosumab; FN = femoral neck; FREEDOM = Fracture Reduction Evaluation of Denosumab in Osteoporosis Every 6 Months; FU = follow=up; IV = intravenous; LBM = low bone mass; LS = lumbar spine; M = male; mg = milligram; m = month(s); OP = osteoporosis; Pbo = placebo; PMPW = postmenopausal women; SD = standard deviation; TH = total hip; y = year(s); Y = year; ZOL = zoledronic acid

turnover markers and clinical factors are needed in men and women, and across all populations to guide clinicians on the utility of follow-up BMD in individualizing treatment and attaining treatment goals. Further studies are also needed in patients undergoing treatment transitions across different drug classes. Research on the role of BTM and clinical factors in guiding reinstitution of treatment in patients on drug interruption and prevention of future fractures is needed.

In addition, research is needed to study the role of follow-up BMD testing in altering health care provider and patient behavior. These studies should include assessing optimal ways of communicating results from a follow-up BMD testing. This is important for education and understanding of bone health to ensure adequate treatment and overcoming treatment inertia in both health care providers and patients. As with all chronic disease management, patient engagement, empowerment and education will be the cornerstones of the lifelong management of this condition.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jocd.2023.101440.

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