ΝΕΟΤΕΡΑ ΣΤΗΝ ΟΣΤΙΚΗ ΔΙΑΤΑΡΑΧΗ ΑΠΟ ΚΑΚΩΣΕΙΣ ΝΩΤΙΑΙΟΥ ΜΥΕΛΟΥ

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MSc (Γήρανση & Χρόνια Νοσήματα) / PhD (ΚΝΜ & Μεταβολικά Νοσήματα των Οστών)

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Πανεπιστήμιο Πατρών

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The International Spinal Cord Society

ΠΑΝΕΠΙΣΤΗΜΙΟ ΠΑΤΡΩΝ
UNIVERSITY OF PATRAS

ΚΛΙΝΙΚΗ ΑΠΟΚΑΤΑΣΤΑΣΗΣ

Προς τί το ενδιαφέρον για την οστική διαταραχή στην κάκωση Ν.Μ.

- Η κάκωση Ν.Μ. προκαλεί μια ακραία και αιφνίδια μορφή ακινητοποίησης των άνω ή και κάτω άκρων (ανάλογα με το επίπεδο της βλάβης).
- Η οστική απώλεια ατόμων με τραυματική κάκωση Νωτιαίου μυελού είναι η «ταχύτερη» του είδους.
- Η ομάδα αυτή των νέων (συνήθως) ανθρώπων είναι ακριβώς αυτή που έχει μεγαλύτερη ανάγκη από αντιμετώπιση.
- Η κάκωση πιθανόν να επισυμβεί πριν από την ανάπτυξη της κορυφαίας οστικής πυκνότητας.
- Κατά τη διάρκεια της παράλυσης μειώνεται η οστική εναλλαγή (κυρίως κάτωθεν του επιπέδου βλάβης), οπότε αναμένονται ακόμη χαμηλότερες τιμές οστικής πυκνότητας με επακόλουθα κατάγματα!

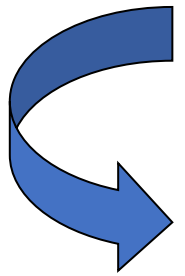
ΚΑΚΩΣΕΙΣ (ΒΛΑΒΕΣ) ΝΩΤΙΑΙΟΥ ΜΥΕΛΟΥ

- Κάκωση Νωτιαίου Μυελού (Ν.Μ.) παραπληγία-τετραπληγία

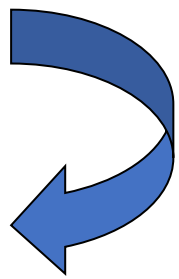
❖πλήρης: καμία αίσθηση ή κινητική λειτουργικότητα δεν διατηρείται στα ιερά τμήματα I4-I5

❖ατελής: διατηρείται η αισθητικότητα αλλά όχι η κινητική λειτουργία κάτω από το επίπεδο της βλάβης μέχρι και τα ιερά τμήματα I4-I5

- Κεντρομυελική βλάβη Ν.Μ. (central cord syndrome)
- Σύνδρομο Brown-Sequard
- Μηνιγγομυελοκήλη



- Σ.Κ.Π. (εντόπιση απομυελινωτικών πλακών στο Ν.Μ. π.χ. ΑΜΣΣ)
- Μυελίτις – μυελοπάθεια
- Πλάγια Μυατροφική Σκλήρυνση (νωτιαία μορφή)



ΔΙΑΦΟΡΕΣ

Στις τραυματικές και παθολογικές βλάβες του Ν.Μ. υπάρχουν διαφορές ως

- *προς την εξέλιξη ή όχι της βλάβης,*
- *το προσδόκιμο επιβίωσης,*
- *την υπολειμματική κινητικότητα και λειτουργικότητα,*
- *την ικανότητα ορθοστάτησης και βάδισης,*
- *τη φαρμακευτική θεραπεία και*
- *τη σπαστικότητα που εμφανίζουν οι ασθενείς.*

...ΑΛΛΑ ΥΠΑΡΧΟΥΝ ΚΑΙ...

ΟΜΟΙΟΤΗΤΕΣ

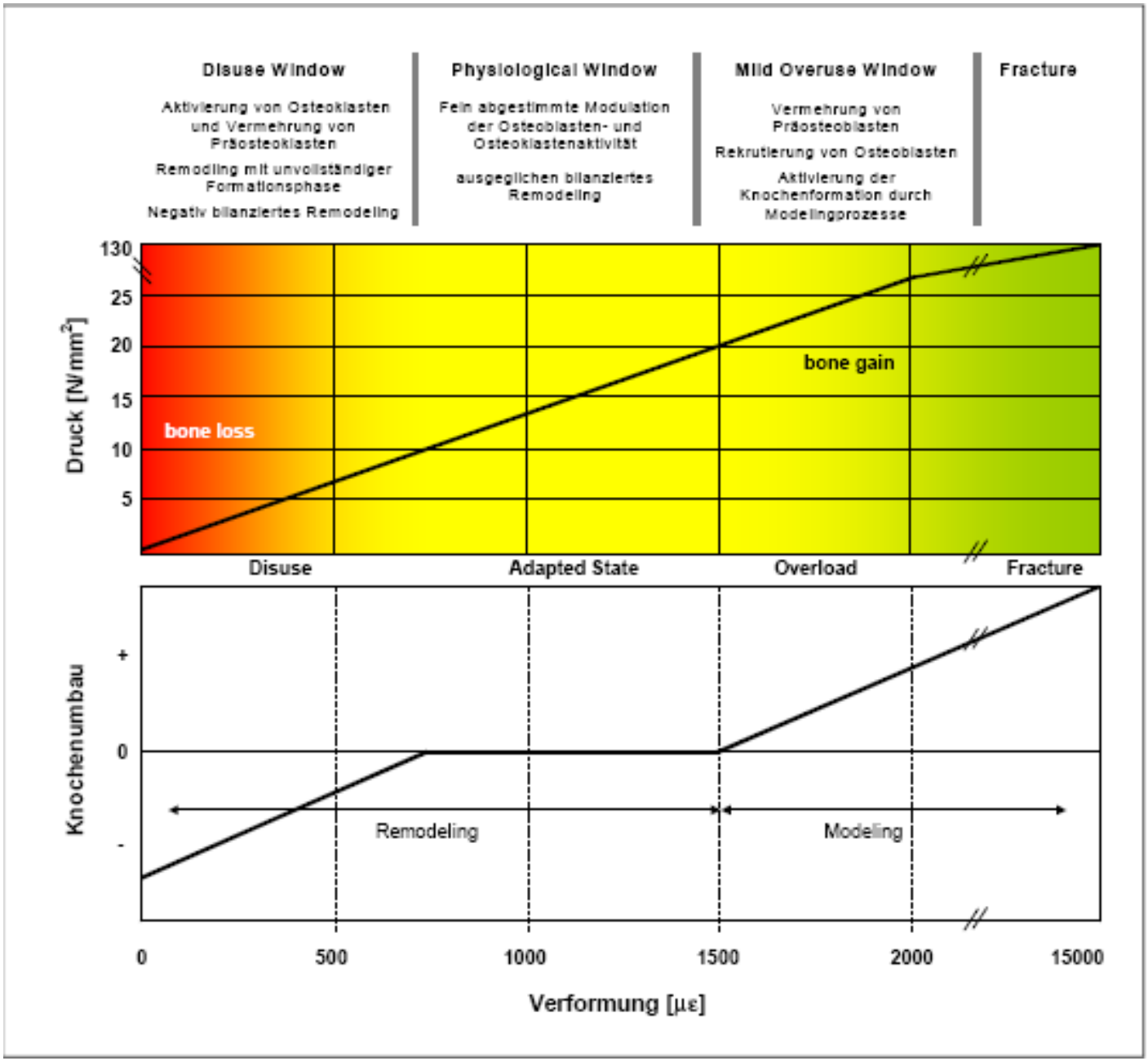
ως

- *προς την κλινική εικόνα*
- *κινητικότητα*
- ***λειτουργικότητα***

Denirel G et al . Spinal Cord 1998;36:822-5.
Kannisto M et al. Spinal Cord 1998;36:641-6.
Frey-Rindova P et al. Spinal Cord 2000;38:26-32.

Θεωρία του ΜΗΧΑΝΟΣΤΑΤΗ κατά FROST

- **Η θεωρία του μηχανοστάτη περιγράφει ένα σύστημα ελέγχου, στο οποίο μια ελάχιστη δραστική καταπόνηση (MES) είναι απαραίτητη για τη διατήρηση του οστού.**
- **Η οστική αντοχή καθορίζεται / ρυθμίζεται από τις μέγιστες μυϊκές δυνάμεις**
- Frost HM. The mechanostat: a proposed pathogenic mechanism of osteoporoses and the bone mass effects of mechanical and nonmechanical agents. Bone Miner. 1987 Apr;2(2):73-85.



Ισχύει ο μηχανοστάτης στη βλάβη Ν.Μ.;

J Musculoskelet Neuronal Interact 2006; 6(3):269-276

Original Article

Hylonome

Bone adaptation to altered loading after spinal cord injury: A study of bone and muscle strength

J. Rittweger[1], K. Gerrits[2], T. Altenburg[2], N. Reeves[1], C.N. Maganaris[1], A. de Haan[1,2]

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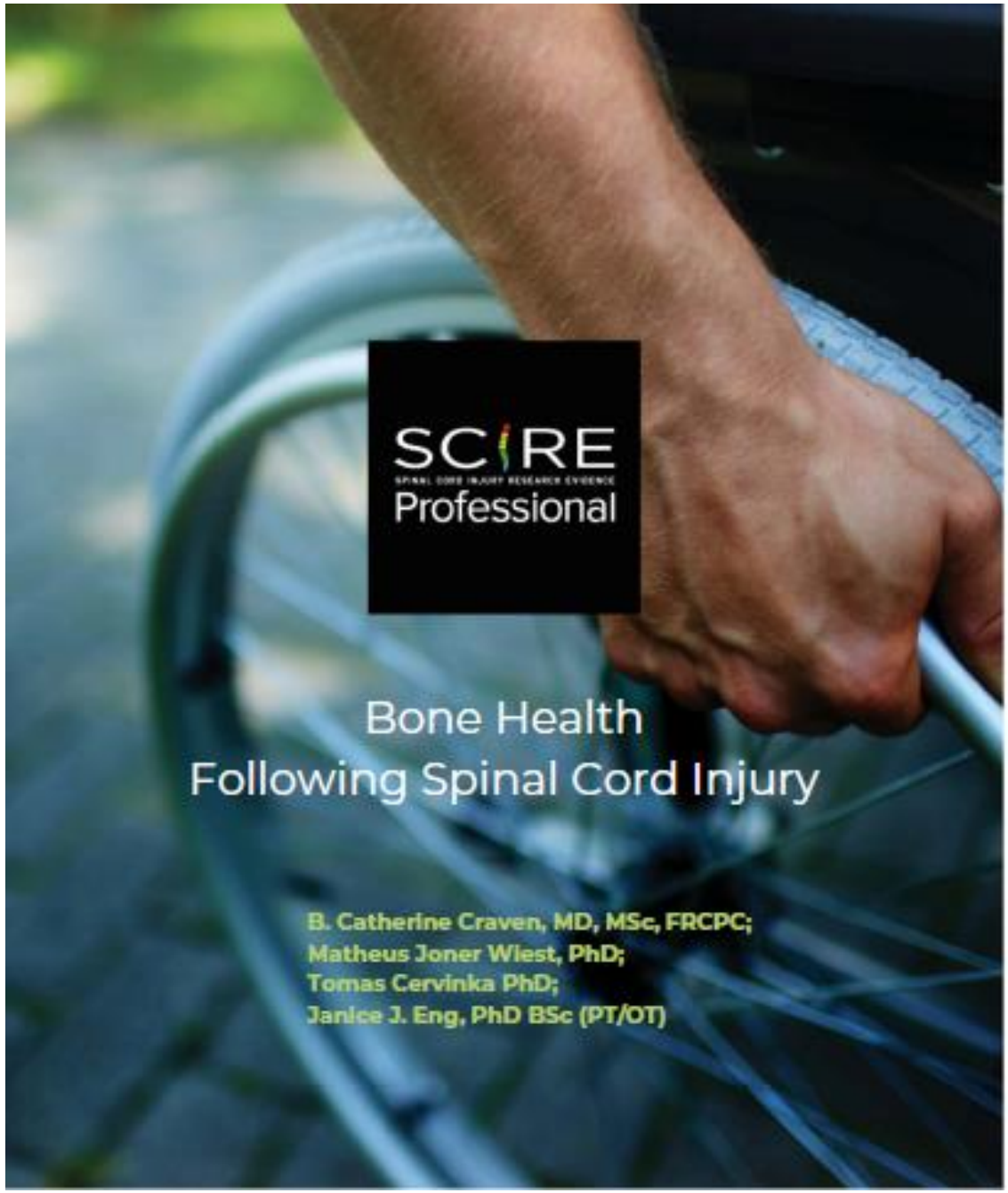
- Υπό φυσιολογικές συνθήκες, οι μεγαλύτερες δυνάμεις προκύπτουν από τις συσπάσεις των μυών.
- Μετά την SCI, οι αισθητικές και κινητικές λειτουργίες διακόπτονται λόγω βλάβης του νευρικού ιστού εντός του σπονδυλικού σωλήνα.
- **Ωστόσο, στις περισσότερες περιπτώσεις, αυτό δεν συνεπάγεται μια πλήρη απώλεια των μυϊκών συσπάσεων, καθώς πολύ συχνά στους περισσότερους ασθενείς SCI εμφανίζονται μυϊκοί σπασμοί και παθολογικά νωτιαία αντανακλαστικά. ***
- Θα μπορούσε να υποστηριχθεί ότι η μείωση της μυϊκής δύναμης δεν θα ήταν η αιτία, αλλά μάλλον ένα παράλληλο συμβάν με τη μείωση αντοχής των οστών μετά από SCI , *καθώς υπάρχει συσσώρευση αποδεικτικών δεδομένων για την εμπλοκή του κεντρικού νευρικού συστήματος στον οστικό μεταβολισμό.*
- *Για παράδειγμα σχετικά με το συμπαθητικό νευρικό σύστημα, πιστεύεται ότι εμποδίζει το σχηματισμό των οστών και ενεργοποιεί την οστική απορρόφηση.*
- **Ωστόσο, η συμπαθητική δραστηριότητα μειώνεται έπειτα από την SCI, και, ως εκ τούτου θα πρέπει να αναμένεται αύξηση της οστικής μάζας και δύναμης μέσω αυτής της οδού.***

Θέματα ορολογίας

- στη διεθνή βιβλιογραφία χρησιμοποιείται και στις αναπηρικές παθήσεις ο όρος *οστεοπόρωση (osteoporosis)* και οι ασθενείς κατατάσσονται σύμφωνα με τα κριτήρια του Παγκόσμιου Οργανισμού Υγείας (WHO) για τις μετεμμηνοπαυσιακές γυναίκες.
- η οστική απώλεια από βλάβη νωτιαίου μυελού (SCI) *λανθασμένα* κατηγοριοποιείται ως εξ' ακινητοποίησης IIπαθής οστεοπόρωση καθώς:

1) η οστική απώλεια είναι πολλή μεγαλύτερη του 10% της απώλειας ακινητοποίησης και
2) δεν αναστρέφεται πλήρως μετά από πιθανή κινητοποίηση ή άλλες παρεμβάσεις (βλ. παρακάτω ορθοστάτηση, ορθώσεις κτλ.)



Craven BC, Wiest MJ, Cervinka T, Eng JJ (2020). Bone Health Following Spinal Cord Injury. In Eng JJ, Teasell RW, Miller WC, Wolfe DL, Townson AF, Hsieh JTC, Connolly SJ, Noonan VK, Loh E, Sproule S, McIntyre A, Querée M, editors. Spinal Cord Injury Rehabilitation Evidence. Version 7.0. Vancouver: p 1- 126.

Sublesional osteoporosis (SLOP)

Table 5. Definition of sublesional osteoporosis (SLOP)

Age range	Definition
Men ≥ 60 years or postmenopausal women	Hip or knee region T score ≤-2.5
Men <59 years or premenopausal women	Hip or knee region Z score <-2.0 with ≥3 risk factors for fracture
Men or women age 16–90	Prior fragility fracture and no identifiable etiology of osteoporosis other than SCI

Craven et al. Top Spinal Cord Inj Rehabil 2009;14(4): –22

Table 3: Risk Factors for Lower Extremity Fragility Fracture after SCI

Yes	Risk Factors
☐	Age at Injury < 16 years
☐	Alcohol Intake > 5 servings/day
☐	Body Mass Index < 19
☐	Duration of SCI ≥ 10 years
☐	Woman
☐	Motor Complete (AIS A-B)
☐	Paraplegia
☐	Family history of fracture in men
☐	Anticonvulsant use (i.e., Tegretol, Depakote Gabapentin – Neurontin)
☐	Spasticity Medication
☐	Opioid analgesia use (≥28 mg morphine for 3 months)
☐	**Prior fragility fracture****
☐	SSRI
☐	PPI
☐	**Knee region BMD below the fracture threshold****
☐	****The big 2****

Fracture thresholds are values below which fragility fractures begin to occur, whereas *fracture breakpoints* are values below which the majority of fractures occur (Garland et al. 2005). Knee region areal BMD (aBMD) and volumetric (vBMD) thresholds for fracture and breakpoint have been identified (Mazess 1990; Eser et al. 2005; Garland et al. 2005). BMD thresholds are described on Table 2.

Table 2: BMD thresholds for fracture and fracture breakpoint.

Name	Value	Definition
Fracture threshold	≤ 0.78 g/cm^2 (aBMD) < 114 mg/cm^3 (vBMD-femur) < 72 mg/cm^3 (vBMD-tibia)	Knee region BMD values below which fragility fractures occur
Fracture breakpoint	< 0.49 g/cm^2 (aBMD)	Knee region BMD values at which the majority of fragility fractures occur
BMD = bone mineral density; aBMD = areal BMD; vBMD = volumetric BMD.		

επίπεδο βλάβης – απώλεια οστού

- Ο ΟΡΟΣ ΟΣΤΕΟΠΟΡΩΣΗ ΚΑΤΩ ΑΠΟ ΤΟ ΕΠΙΠΕΔΟ ΤΗΣ ΒΛΑΒΗΣ **(sublesional osteoporosis)** ΠΡΕΠΕΙ ΝΑ ΧΡΗΣΙΜΟΠΟΙΕΙΤΑΙ ΜΕ ΠΡΟΣΟΧΗ ΙΔΙΑΙΤΕΡΑ ΣΤΗΝ ΠΑΡΑΠΛΗΓΙΑ ΑΛΛΑ ΚΑΙ ΣΕ ΙΣΟΔΥΝΑΜΕΣ ΚΑΤΑΣΤΑΣΕΙΣ (MS)

- Η ΕΞΗΓΗΣΗ ΟΦΕΙΛΕΤΑΙ ΣΕ ΔΥΟ ΛΟΓΟΥΣ

Α) ΥΠΑΡΧΕΙ <ΔΙΑΤΗΡΗΣΗ> ΟΣΤΙΚΗΣ ΜΑΖΑΣ ΣΤΗ Σ.Σ. ΣΕ ΠΕΡΙΟΧΕΣ ΚΑΤΩΘΕΝ ΤΟΥ ΕΠΙΠΕΔΟΥ ΒΛΑΒΗΣ ΛΟΓΩ ΤΗΣ ΦΟΡΤΙΣΗΣ ΠΟΥ ΥΦΙΣΤΑΝΤΑΙ ΑΠΟ ΤΗΝ ΚΑΘΙΣΤΗ ΘΕΣΗ ΣΤΟ ΑΝΑΠΗΡΙΚΟ ΑΜΑΞΙΔΙΟ.

Β) ΥΠΑΡΧΕΙ ΜΗΧΑΝΙΚΗ ΦΟΡΤΙΣΗ ΣΤΗΝ ΠΕΡΙΟΧΗ ΤΗΣ ΒΛΑΒΗΣ ΑΠΟ ΤΑ ΥΛΙΚΑ ΤΗΣ ΣΠΟΝΔΥΛΟΔΕΣΙΑΣ ΠΟΥ ΕΧΕΙ ΓΙΝΕΙ ΜΕΤΑ ΤΗ ΒΛΑΒΗ

Bone Impairment and Spinal Cord Injury

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Charles A. Stewart, M.D.
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Rancho Los Amigos National Rehabilitation Center
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Garland DE, Adkins RH. 2001. Bone loss at the knee in spinal cord injury. Topics in Spinal Cord Injury Rehabilitation 6:37-46.

Garland DE, Adkins RH, Kushwaha V, Stewart C. 2004b. Risk factors for osteoporosis at the knee in the spinal cord injury population. Journal of Spinal Cord Medicine 27(3):202-206.

Garland DE, Adkins RH, Rah A, Stewart CA. 2001a. Bone loss with aging and the impact of SCI. Topics in Spinal Cord Injury Rehabilitation 6:47-60.

Garland DE, Adkins RH, Scott M, Singh H, Massih M, Stewart CA. 2004a. Bone loss at the os calcis compared with bone loss at the knee in individuals with spinal cord injury. Journal of Spinal Cord Medicine 27(3):207-211.

Garland DE, Adkins, RH, Stewart CA. 2005a. The natural history of bone loss in the lower extremity of complete spinal cord injured males. Topics in Spinal Cord Injury Rehabilitation 11:48-60.

Garland DE, Adkins RH, Stewart CA. 2005b. Fracture threshold and risk for osteoporosis and pathological fracture individuals with spinal cord injury. Topics in Spinal Cord Injury Rehabilitation 11:61-69.

Garland DE, Adkins RH, Stewart CA. 2008. Five-year longitudinal bone evaluations of the lumbar spine, hip, knee and lower extremity in individuals with chronic, complete SCI. Journal of Spinal Cord Medicine 31(5): 543-550.

Garland DE, Adkins RH, Stewart CA, Ashford R, Vigil D. 2001b. Regional osteoporosis in women who have a complete spinal cord injury. Journal of Bone Joint Surgery. American edition. 83A(8):1195-1200.

Garland DE, Foulkes GD, Adkins RH, Stewart CA, Yakura JS. 1994. Regional osteoporosis following incomplete spinal cord injury. Contemporary Orthopaedics 28:134-139.

Garland DE, Maric Z, Adkins RH, Stewart CA. 1993. Bone density about the knee in spinal cord injured patients with pathologic fractures. Contemporary Orthopaedics 26:75-79.

•Ειδικότερα σε βλάβη Ν.Μ. η ορολογία **οστική διαταραχή λόγω βλάβης Ν.Μ. (SCI-related bone impairment)** κρίνεται ως πιο ακριβής

•Ο όρος bone impairment προκρίνεται και του όρου *bone disorder* καθώς ενέχει ορολογία Αποκατάστασης που σχετίζεται άμεσα με τη μακροχρόνια πορεία των ατόμων αυτών

Εικόνα 3. Διεθνής ταξινόμηση της λειτουργικότητας, της ανικανότητας και της υγείας (ICF)[23].

Στην Αποκατάσταση και σύμφωνα με την διεθνή ταξινόμηση της λειτουργικότητας, της ανικανότητας και της υγείας (ICF), **με τον όρο διαταραχή αναφερόμαστε σε πρόβλημα μιας δομής ή οργάνου του σώματος, σε αυτή την περίπτωση της οστικής δομής.**

S1 Guidelines on Bone Impairment in Spinal Cord Injury

Yannis Dionyssiotis,[1,6,] Yorck-Bernhard Kalke,[2] Angela Frotzler,[3] Jürgen Moosburger,[4] Georgios Trovas,[5,6] Evangelia Kaskani,[7] Belgin Erhan,[8] Calogero Foti,[9] Jannis Papathanasiou,[10,11] Jose Luis Ferretti,[12] Marta Imamura,[13] and Anastasia-Christina Rapidi[14], HeSCOSPRM Bone and Spinal Cord Injury Working Group Attendants*

[1] 1st Physical Medicine and Rehabilitation Department, National Rehabilitation Center EKA, Athens, Greece; [2] SCI Centre, Orthopaedic Department, Ulm University, Ulm, Germany; [3] Clinical Trial Unit, Swiss Paraplegic Centre, Nottwil, Switzerland; [4] Medical Rehabilitation Center for Spinal Cord Injured "Heinrich-Sommer-Klinik", Bad Wildbad, Germany; [5] Laboratory for Research of the Musculoskeletal System, "Th. Garofalidis" Medical School, National and Kapodistrian University of Athens, Kifissia, Greece; [6] Hellenic Osteoporosis Foundation, Kifissia, Greece; [7] Hellenic Society for the Study of Bone Metabolism, Maroussi, Greece; [8] Physical Medicine and Rehabilitation Department, İstanbul Medeniyet University Faculty of Medicine, Instabul, Turkey; [9] Clinical Medicine and Translational Department Tor Vergata University, Rome, Italy; [10] Department of Medical Imaging, Allergology and Physiotherapy, Faculty of Dental Medicine, Medical University of Plovdiv, Plovdiv, Bulgaria; [11] Department of Kinesitherapy, Medical University of Sofia, Sofia, Bulgaria; [12] Center of P-Ca Metabolism Studies (CEMFoC), Natl Univ of Rosario and Arg NRC (CONICET), Rosario, Argentina; [13] Faculdade de Medicina, Universidade de Sao Paulo, Sao Paulo, Brazil; and [14] Physical Medicine and Rehabilitation Department, General Hospital "G. Gennimatas", Athens, Greece

Journal of Clinical Densitometry: Assessment & Management of Musculoskeletal Health, vol. 24, no. 3, 490-501, 2021

Prophylaxe, Diagnostik und Therapie der OSTEOPOROSE

bei postmenopausalen Frauen und bei Männern

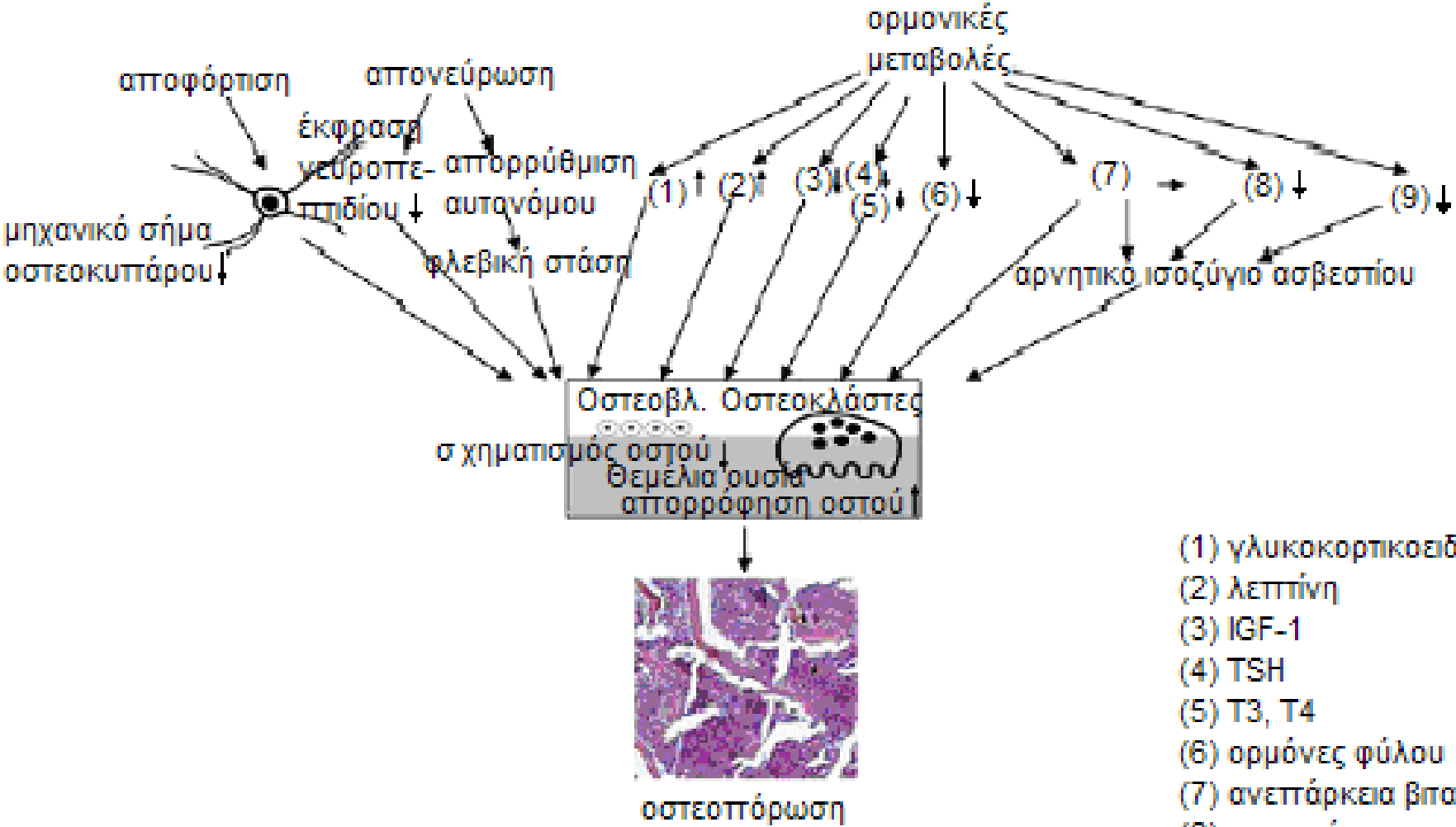
Leitlinie des Dachverbands der Deutschsprachigen Wissenschaftlichen Osteologischen Gesellschaften e.V.

2017

Deutschsprachige Medizinische Gesellschaft für Paraplegie e.V.

Spinal Cord Injury Research Evidence

πολυπαραγοντική παθοφυσιολογία στην οξεία και στη χρόνια φάση

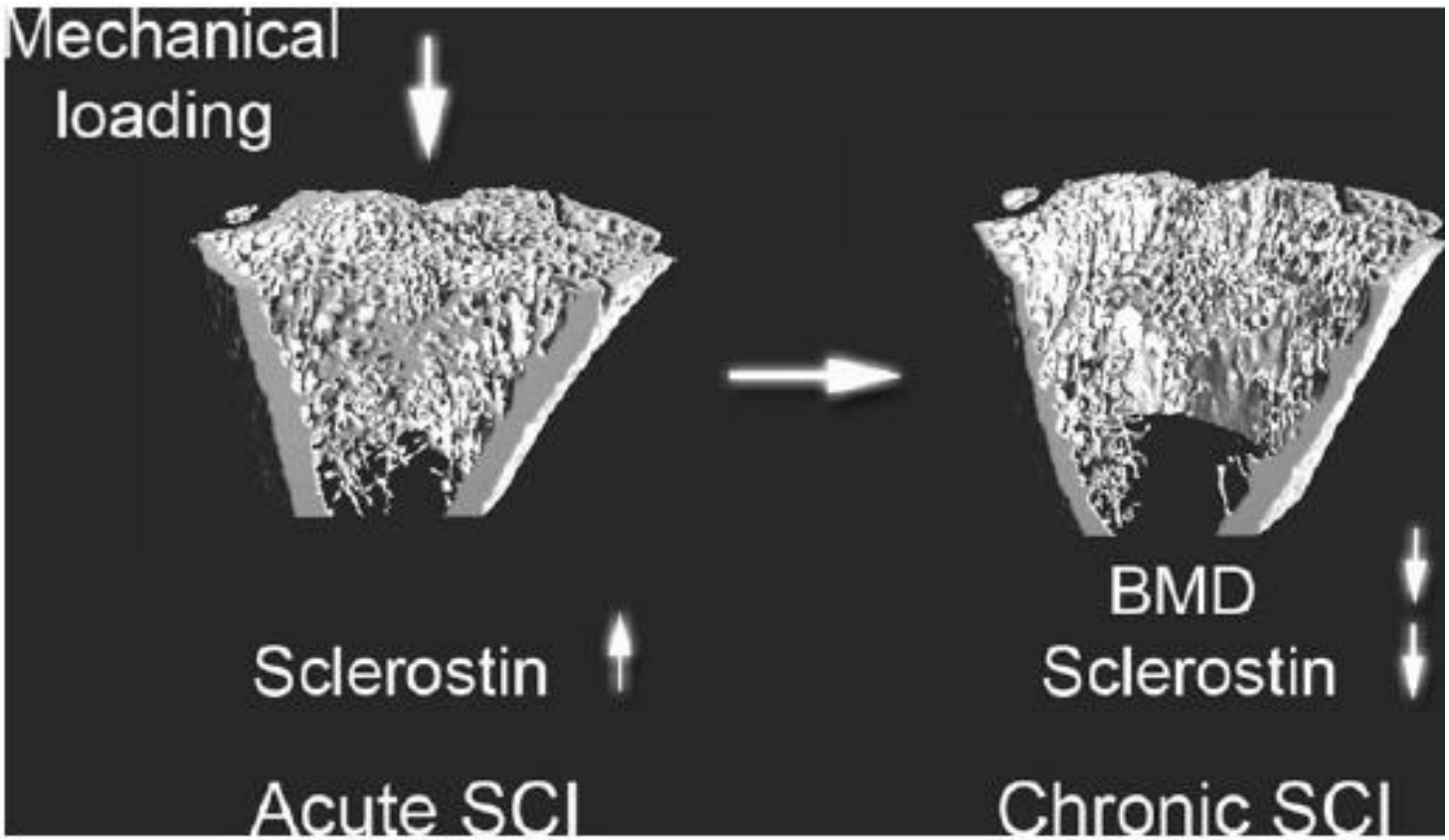


Curr Osteoporos Rep
DOI 10.1007/s11914-012-0117-0

EPIDEMIOLOGY AND PATHOPHYSIOLOGY (RA ADLER, SECTION EDITOR)

Spinal Cord Injury-Induced Osteoporosis: Pathogenesis and Emerging Therapies

Ricardo A. Battaglino · Antonio A. Lazzari · Eric Garshick · Leslie R. Morse

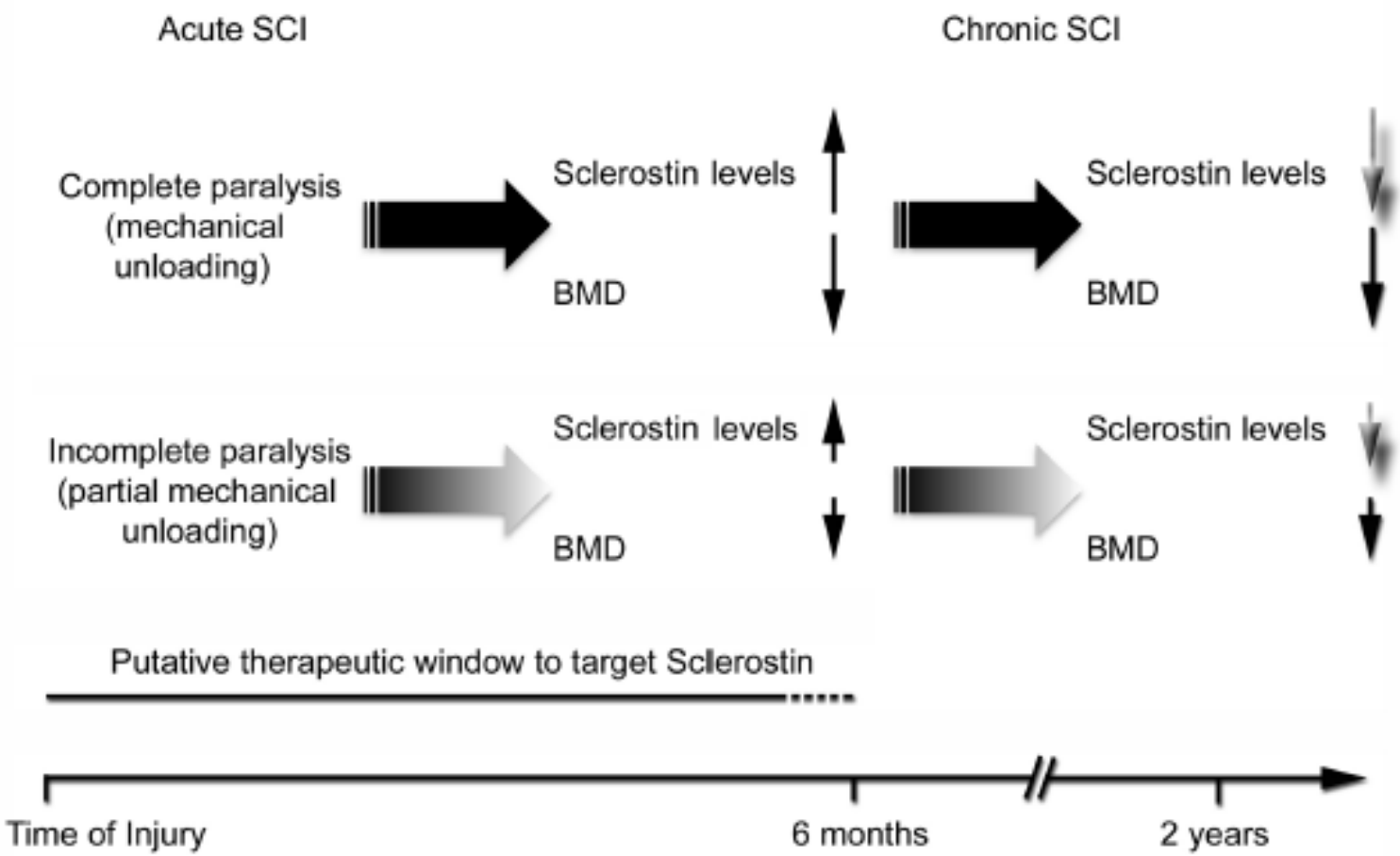


J Bone Miner Res. 2012 February ; 27(2): 352–359. doi:10.1002/jbmr.546.

Association between Sclerostin and Bone Density in Chronic SCI

Leslie R. Morse, DO, CCD[1,2,3,4], Supreetha Sudhakar, MPH[2], Valery Danilack, MPH[5], Carlos Tun, MD[6], Antonio Lazzari, MD, CCD[7], David R. Gagnon, MD, MPH, PhD[8], Eric Garshick, MD[9,10], and Ricardo A. Battaglino, PhD[3,11]

Conceptual Model of Sclerostin-Mediated Bone Loss Following SCI



- The ***hypercalciuria***

❑ seen in 10 days after neurological injury

❑ reaches its peak at 1.6 months (is 2-4 times the calciuria observed in long bed ridden subjects) and still in the next 12 months with various degrees.

- *Excessive secretion of calcium in urine leads to hypercalcemia*

- *Hypercalcaemia*

❑ usually develops 4-8 weeks after SCI but can be started early by 2 weeks or late at 6 months after injury.

Especially in renal insufficiency

Διαταραχή του άξονα Ca-PTH

- Η υπερασβεστιαιμία και η οστική απορρόφηση που εμφανίζονται αμέσως μετά τη βλάβη είναι υπεύθυνα για τα χαμηλά επίπεδα της PTH.
- Η μειωμένη PTH σχετίζεται με αυξημένες συγκεντρώσεις φωσφόρου και μειωμένη σύνθεση 1,25-dihydroxyvitamin D.
- Οι παραθυρεοειδείς αδένες είναι σχεδόν ανενεργοί με χαμηλά επίπεδα PTH να παρατηρούνται μέχρι και 1 έτος μετά τη βλάβη SCI.
- Τα επίπεδα της PTH έχουν αντίστροφή συσχέτιση με το επίπεδο της βλάβης με μικρότερα σε τετραπληγία κ.ο.κ και σε πλήρεις βλάβες Ν.Μ.
- *Το αποτέλεσμα είναι μια αύξηση της οστικής απορρόφησης που σχετίζεται με τη δυσλειτουργία των παραθυρεοειδών στη χρόνια φάση της βλάβης.*
- Σταδιακά σε φάσμα 1-9 ετών αποκαθίσταται η λειτουργία των παραθυρεοειδών

Spinal Cord Series and Cases (2021)7:12
https://doi.org/10.1038/s41394-020-00361-7

ISCoS The International Spinal Cord Society

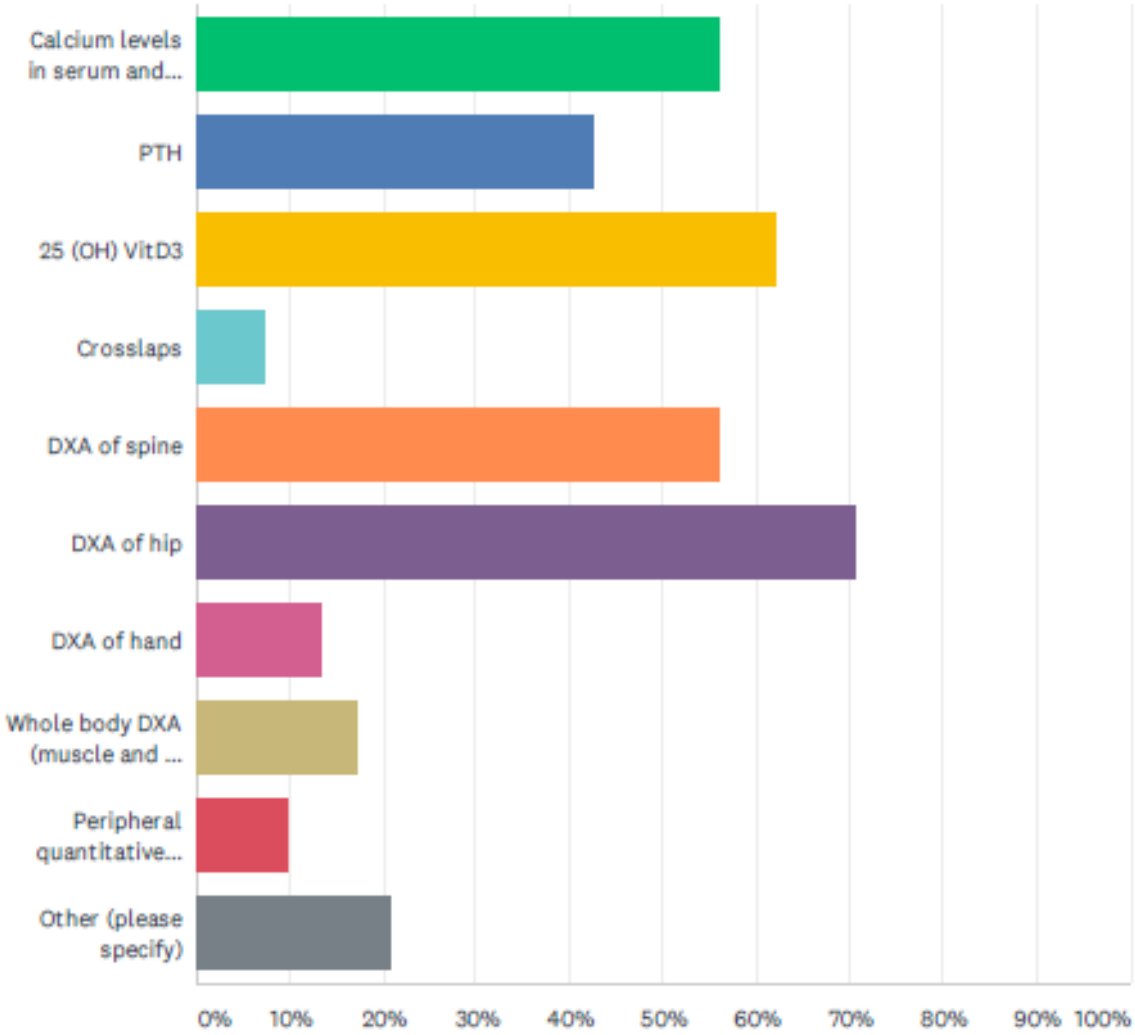
ARTICLE

A pulse survey: assessment and management of bone loss in spinal cord injury

Yannis Dionyssiotis[1] · Ruth Marshall[2,3] · Michael D. Stillman[4] · Leslie R. Morse[5] · Anastasia-Christina Rapidi[6]

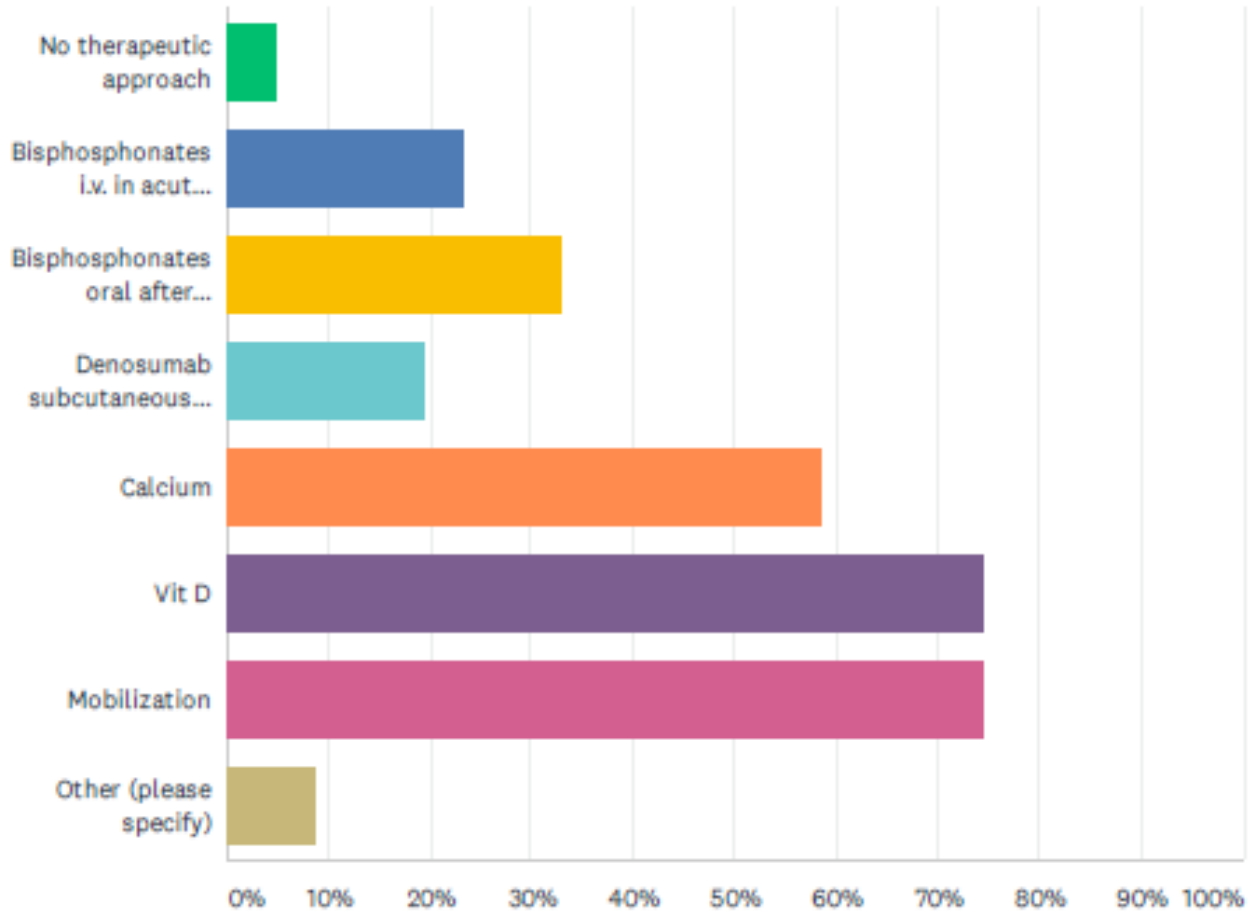
Q5 If you measure bone loss in persons with SCI, which tests do you use? (more than one answer can be chosen)

Answered: 82 Skipped: 0



Q7 Which therapeutic approach do you prefer for continued bone loss (abnormal blood and urine tests) during the acute & subacute phase in order to stop bone loss (more than one answer can be chosen)

Answered: 82 Skipped: 0



DIAGNOSIS

MEASUREMENTS
BLOOD AND URINE TESTS - DXA

Table 1
Biochemical Parameters in Serum and Urine.

Biochemical parameter	Why is important to calculate
Serum Calcium (corrected to serum albumin)	↑ Primary Hyperparathyroidism or other causes of Hypercalcaemia ↓ e.g., secondary hyperparathyroidism, malabsorption, Hypocalcemia as a contraindication to several osteoporosis medications
Serum - Phosphorus	↑ renal insufficiency stage IV ↑ secondary renal hyperparathyroidism ↓Malabsorption
Serum creatinine	
Alkaline Phosphatase (ALP) (Serum)	↑ i.e., Osteomalacia
BSG /C-Reactive Protein	↑ Differential diagnosis inflammatory Causes of vertebral deformities, inflammatory rheumatic diseases
Blood cell account	evidence of inflammatory and malignant diseases or celiac disease
Serum – protein electrophoresis	Evidence of a monoclonal gammopathy or hypogamma-globulinemia as an indication of MGUS Monoclonal gammopathy of unclear significance or multiple myeloma or a systemic inflammatory disease
TSH	<0.3 mU/L endogenous or caused by L-thyroxine medication as a risk factor for fractures
Testosteron in men	Testosteron deficiency
25-Hydroxy-Vitamin D3	Vitamin D deficiency
24 h creatinin secretion	Correct volume collection
24 h Urine Calcium secretion	↑ hypercalciuria ↓ hypocalciuria

Measurement of specific parameters is important for differential diagnosis of other diseases.

ΜΕΘΟΔΟΙ ΑΠΕΙΚΟΝΙΣΗΣ

Οι μέθοδοι απεικόνισης που χρησιμοποιούνται για την αξιολόγηση των οστών περιλαμβάνουν (DXA), (DPA) και τυπική και υψηλής ανάλυσης (pQCT, HR-pQCT).

Ωστόσο, η διαθεσιμότητα των pQCT περιορίζεται κυρίως σε ερευνητικά ιδρύματα, εν μέρει λόγω της ασυμβατότητας των δεδομένων pQCT με τα T σκορ που προέρχονται από το DXA, την έλλειψη μελετών σε φυσιολογικούς πληθυσμούς και τα κατώφλια θεραπείας (Engelke et al. 2008, Adams et al. 2014, Zysset et al. 2015).

Craven C, Lynch CL, Eng JJ (2014). Bone Health Following Spinal Cord Injury. In Eng JJ, Teasell RW, Miller WC, Wolfe DL, Townson AF, Hsieh JTC, Connolly SJ, Noonan VK, Loh E, McIntyre A, editors. Spinal Cord Injury Rehabilitation Evidence. Version 5.0. Vancouver: p 1- 37.

Υπάρχουν περισσότεροι από 50.000 σαρωτές DXA ολόκληρου σώματος, περίπου 800 pQCT (XCT 2000 και 3000) και μόλις 50 σαρωτές HR-pQCT (XtremeCT και XtremeCT II) που χρησιμοποιούνται στην κλινική πρακτική/έρευνα παγκοσμίως (Shepherd et al. 2014· (Medizintechnik GmbH και Scanco Medical).

ELSEVIER

Journal of Clinical Densitometry

Volume 22, Issue 4, October–December 2019, Pages 554-566

2019 ISCD Official Position

Bone Mineral Density Testing in Spinal Cord Injury: 2019 ISCD Official Position

Leslie R. Morse [1], Fin Biering-Soerensen [2], Laura D. Carbone [3, 4], Tomas Cervinka [5], Christopher M. Cirnigliaro [6], Therese E. Johnston [7], Nan Liu [8], Karen L. Troy [9], Frances M. Weaver [10, 11], Christopher Shuhart [12], Beverley C. Craven [13]

4 ερωτήσεις που σχετίζονται με την κλινική χρησιμότητα της αξιολόγησης της οστικής πυκνότητας με (DXA) σε άτομα με ΚΝΜ

- Question 1: What are the indications for initial DXA in individuals with spinal cord injury?

ISCD Official Position Statement

· All adults with spinal cord injury resulting in permanent motor or sensory dysfunction should have a DXA scan of the total hip, proximal tibia, and distal femur as soon as medically stable.

Grade: Fair, B, W

Question 2: Can bone densitometry by DXA be used to diagnose osteoporosis, assess fracture risk, or monitor response to therapy in individuals with spinal cord injury?

ISCD Official Position

· In adults with SCI, total hip, distal femur and proximal tibia bone density should be used to diagnose osteoporosis, predict lower extremity fracture risk and monitor response to therapy where normative data are available.

Grade: Fair, B, W

Question 3: How should DXA be used to monitor osteoporosis therapy (drug, nutraceuticals, rehabilitation interventions) in individuals with SCI?

ISCD Official Position

· Serial DXA assessment of treatment effectiveness among individuals with SCI should include evaluation at the total hip, distal femur, and proximal tibia, following a minimum of 12 months of therapy at 1- to 2-year intervals. Segmental analysis of total hip, distal femur and proximal tibia sub-regions from a whole-body scan should not be used for monitoring treatment.

Grade Fair, B, W

Question 4: Are there DXA based criteria that are absolute or relative contra-indication to exercise-based therapy?

ISCD Official Position

· There is no established threshold BMD value below which weight-bearing activities are absolutely contraindicated. BMD and clinical risk factors should be used to assess fracture risk prior to engaging in weight-bearing activities.

Grade: Poor, C, W

prevention and treatment

Pros vs. Cons

Spinal Cord Series and Cases (2019)5:27
https://doi.org/10.1038/s41394-019-0165-0

PERSPECTIVE

Osteoporosis prophylaxis in acute SCI

Leslie Morse[1,2]

Is prophylaxis for osteoporosis indicated after acute spinal cord injury?

Yannis Dionyssiotis[1]

Prophylactic treatment of osteoporosis after SCI: promising research, but not yet indicated

Dustin Anderson[1] · Andrew J Park[1]

CALCIUM IN ACUTE PHASE RIGHT OR WRONG?

- In acute SCI, calcium dietary intake is beneficial
- The suppression of PTH results in reduced absorption of calcium in gut.

- *Others highlight the risk of calcium nephrolithiasis after acute SCI due to high bone turnover*
- *However, hypercalciuria after abrupt immobilization is due to unregulated bone resorption and not appreciably influenced by dietary intake*
- *This may misguide people with SCI to avoid calcium intake or dairy products.*

Should calcium be used in the prevention and treatment in SCI subjects?

- Basic therapy includes the administration of calcium.
- A total calcium intake of 1000 mg daily is recommended, but the conventional diet is generally considered insufficient.

Low calcium levels, what they cause?

- Provoke production of PTH to produce more 1,25(OH)2D.
- Increased values of PTH initiate bone remodeling, resulting in further bone resorption and increasing released calcium ions in the blood.

- *Before starting therapy with antiresorptive drugs supplementation with vitamin D is necessary.*

WHAT ABOUT VITAMIN D

- If 25(OH)D levels are below normal, calcium is inadequately absorbed in gut and
- because an antiresorptive agent like bisphosphonates or denosumab strongly suppresses bone resorption,
- severe iatrogenic hypocalcaemia may develop.

serum 25(OH)D > 30 ng/ml is desirable

Αναφορικά με την έλλειψη ή ανεπάρκεια βιταμίνης D έχει βρεθεί πως στο 64% των παραπληγικών υπάρχει ένδεια vitamin D (< 15ng/ml)

An effective oral vitamin D replacement therapy in persons with spinal cord injury

William A. Bauman[1,2], Racine R. Emmons[1,3], Christopher M. Cirnigliaro[1], Steven C. Kirshblum[4,5], Ann M. Spungen[1,2]

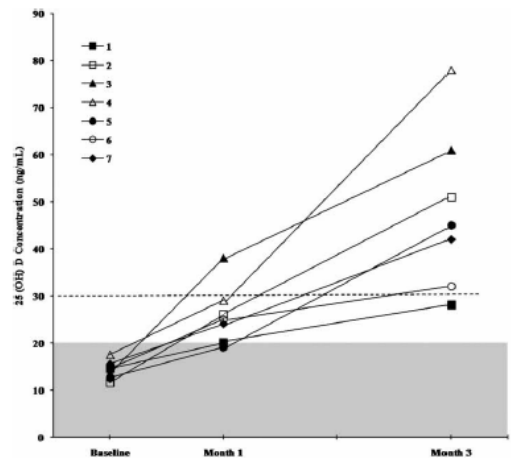


Figure 1 Serum vitamin D [25(OH)D] levels after supplementation with 2000 IU/day. Values are displayed for seven subjects after month 1 and month 3 of replacement vitamin administration. Note that six of seven subjects had 25(OH)D levels >30 ng/ml by month 3; the only subject who did not normalize the 25(OH)D level by month 3 approached the lower limit of normal (i.e. 28 ng/ml) from a baseline level of 17 ng/ml.

What is the role of vitamin D in the prevention and treatment of bone loss in SCI?

Basic therapy includes the administration of vitamin D3.

vitamin D3 recommended dose should be 800 - 1000 IU per day.

Other countermeasures except neutraceuticals and drugs?? YES


STANDING


WALKING WITH BRACES


FES


FES-CYCLING


VIBRATION


PEMF

EARLY MOBILIZATION – TILL TABLE - STANDING

DECREASES HYPERCALCIURIA

FOCUS ON ALL RISK FACTORS WHICH CAN CHANGE (INCLUDING FALLS, LIFESTYLE MODIFICATION, etc.)

MONITORING REGULARLY (BIOCHEMICAL INDICES AND DXA)

CHECK FOR COMPLIANCE ISSUES

PREVENTION &MANAGEMENT

- **Lifestyle Modifications:**

•Explain the anticipated decline in hip and knee region BMD for 18-24 months after SCI and the associated future risk of lower extremity fragility fracture. Discuss the differences between an incident and fragility fracture. Acquisition of optimal transfer technique in and out of his wheelchair, car and motocross bikes will minimize his future fracture risk.
•Encourage the pursuit of an alternate role within his preferred recreational activity (i.e. official versus competitor, recreational versus competitive moto-cross, etc.) or involvement in alternate sports/activities.
•Restrict caffeine intake to ≤ three 8 oz servings per day.
•Discuss smoking cessation strategies, ask him to pick a date for quitting, and offer smoking cessation therapy as appropriate (i.e. cognitive behavior therapy, nicotine patch, Zyban®, etc. alone/in combination).

Functional electrical stimulation

Functional electrical stimulation (*FES*) is a treatment that applies electrical stimulation to the muscles during activities like standing, walking, or cycling. This treatment involves repeated pulling of the muscles on bones, which could help to strengthen the bones.

There is evidence suggesting that functional electrical stimulation cycling does not prevent bone loss in the lower leg early after SCI but may help to increase bone mineral density in stimulated areas of the leg during the later period after SCI. However, the stimulation needs to be ongoing or the benefits will be lost.

Refer to our chapter on FES for more information!

FES can be applied to the leg muscles during assisted walking.[11]

Craven C, Lynch CL, Eng JJ (2014). Bone Health Following Spinal Cord Injury. In Eng JJ, Teasell RW, Miller WC, Wolfe DL, Townson AF, Hsieh JTC, Connolly SJ, Noonan VK, Loh E, McIntyre A, editors. Spinal Cord Injury Rehabilitation Evidence. Version 5.0. Vancouver: p 1- 37.

Available from: http://scireproject.com/evidence/rehabilitation-evidence/bone-health/

Tilt Table GALILEO ©

Asselin et al. Low-intensity vibration and bone preservation

Available online at www.sciencedirect.com

ScienceDirect

Current Opinion in Pharmacology

Pharmacological approaches for bone health in persons with spinal cord injury

William A. Bauman[1,2,3]

Current Opinion in Pharmacology 2021, 60:346–359

Osteoporos Int
DOI 10.1007/s00198-010-1221-6

ORIGINAL ARTICLE

Early treatment with zoledronic acid prevents bone loss at the hip following acute spinal cord injury

J. S. Bubbear · A. Gall · F. R. I. Middleton · M. Ferguson-Pell · R. Swaminathan · R. W. Keen

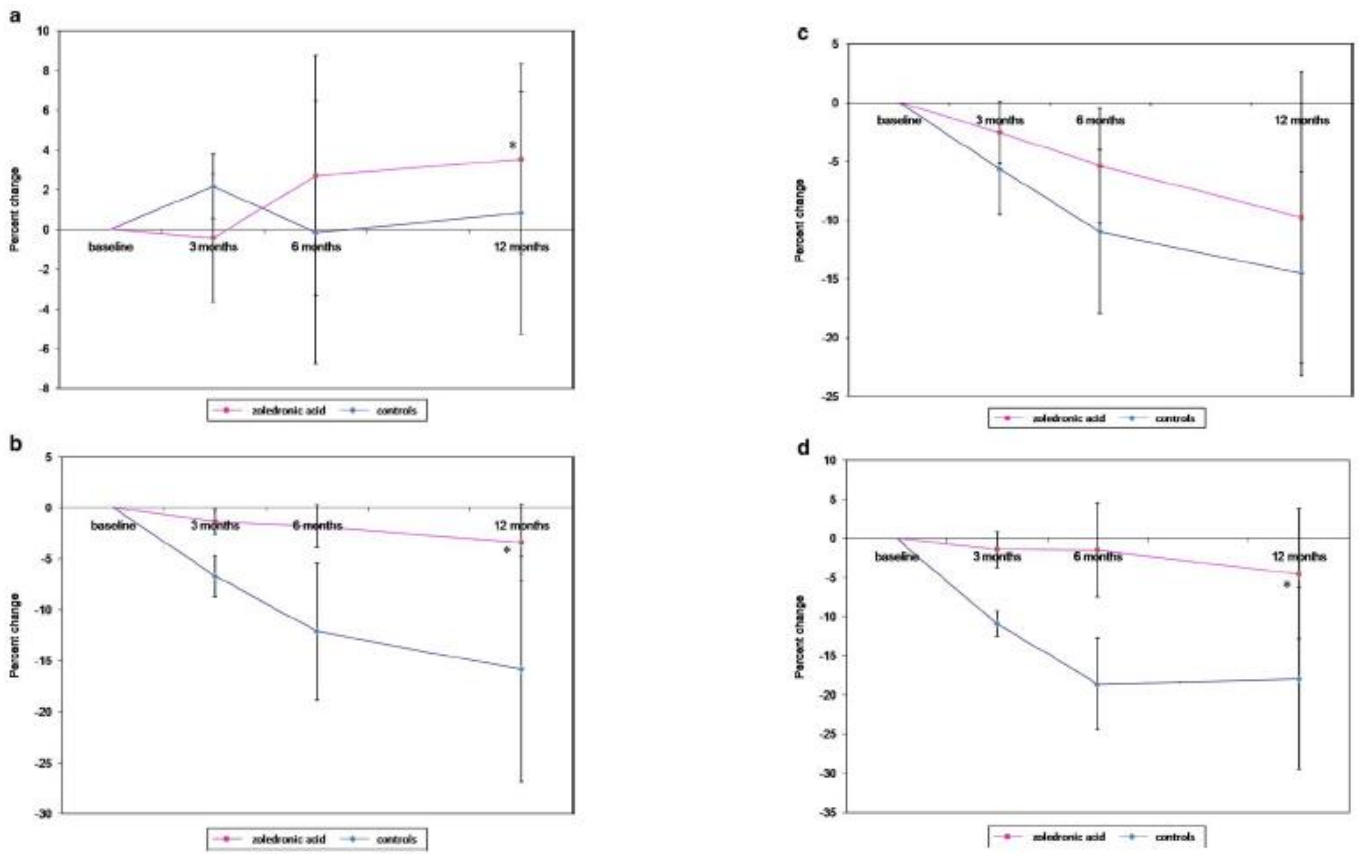


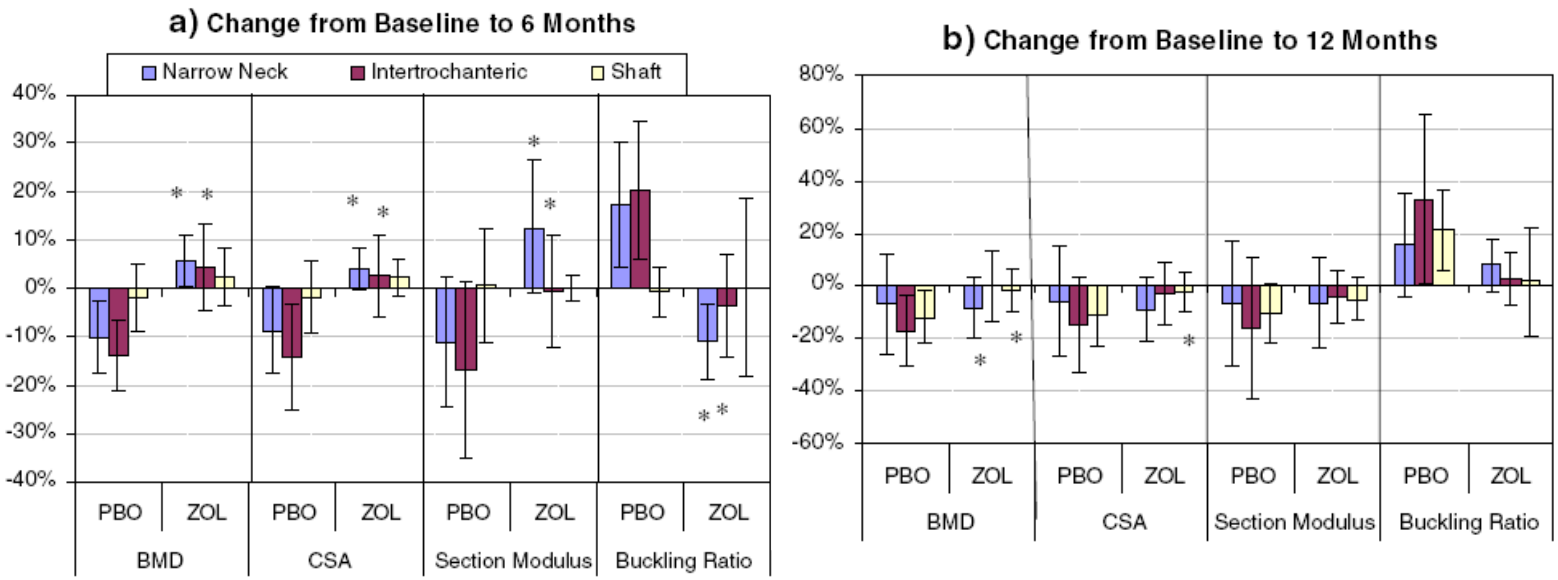
Fig. 2 Mean percentage changes in BMD (±95% confidence intervals). *Difference between groups, $p \leq 0.05$. a Lumbar spine, b Total hip, c Femoral neck, d Trochanter

Calcif Tissue Int (2007) 80:316–322
DOI: 10.1007/s00223-007-9012-6

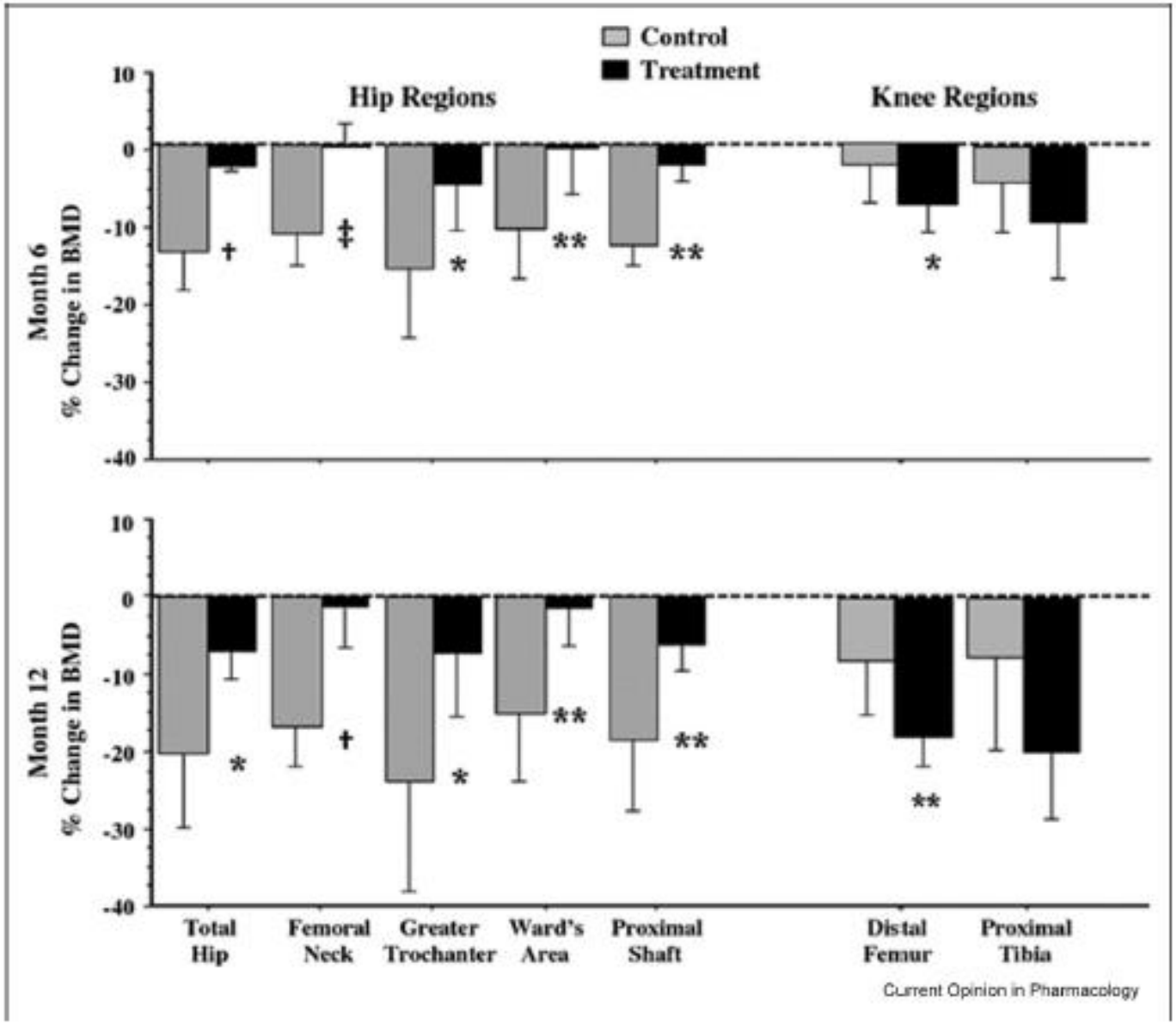
Calcified Tissue International

Treatment with Zoledronic Acid Ameliorates Negative Geometric Changes in the Proximal Femur following Acute Spinal Cord Injury

J. Shapiro,[1] B. Smith,[2] T. Beck,[3] P. Ballard,[2] M. Dapthary,[3] K. BrintzenhofeSzoc,[4] J. Caminis[5]



1. In treatment group:
A. Six months after zoledronic acid, BMD, cross-sectional area, and sectional modulus increased at the hip and buckling ratio decreased consistent with improved bone outcomes.
B. At 12 months, narrow-neck femur values declined and intertrochanteric and femoral shaft BMD was maintained.
2. Placebo group showed a decrease in bone outcomes and an increase in buckling ratio at the hip at 6 and 12 months



Percent change in BMD at the hip and knee after treatment with zoledronic acid. The darker boxes represent the treatment group and the lighter boxes represent the control group. Percent change from baseline for treatment versus control: $^{*}p < 0.05$; $^{**}p < 0.01$; $^{\dagger}p < 0.001$; $^{\ddagger}p < 0.0001$. Reproduced with permission from Bauman et al.[49]

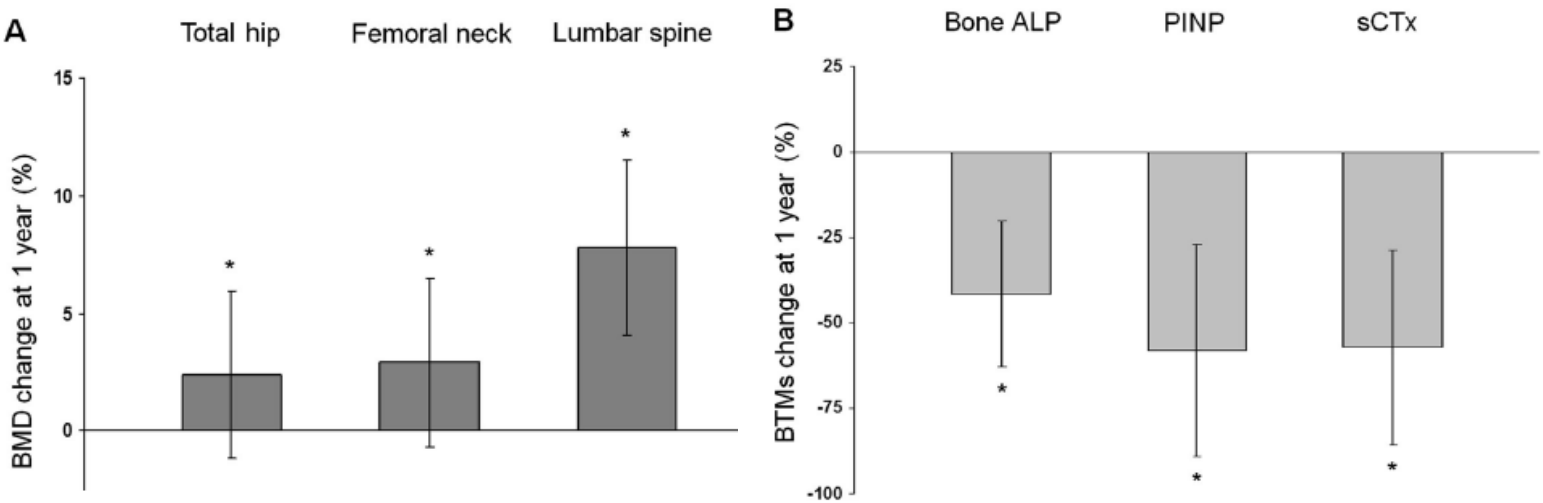
Bauman WA, et al.: Zoledronic acid administration failed to prevent bone loss at the knee in persons with acute spinal cord injury: an observational cohort study. J Bone Miner Metabol 2015, 33:410–421.

Osteoporos Int (2016) 27:405–410
DOI 10.1007/s00198-015-3333-5

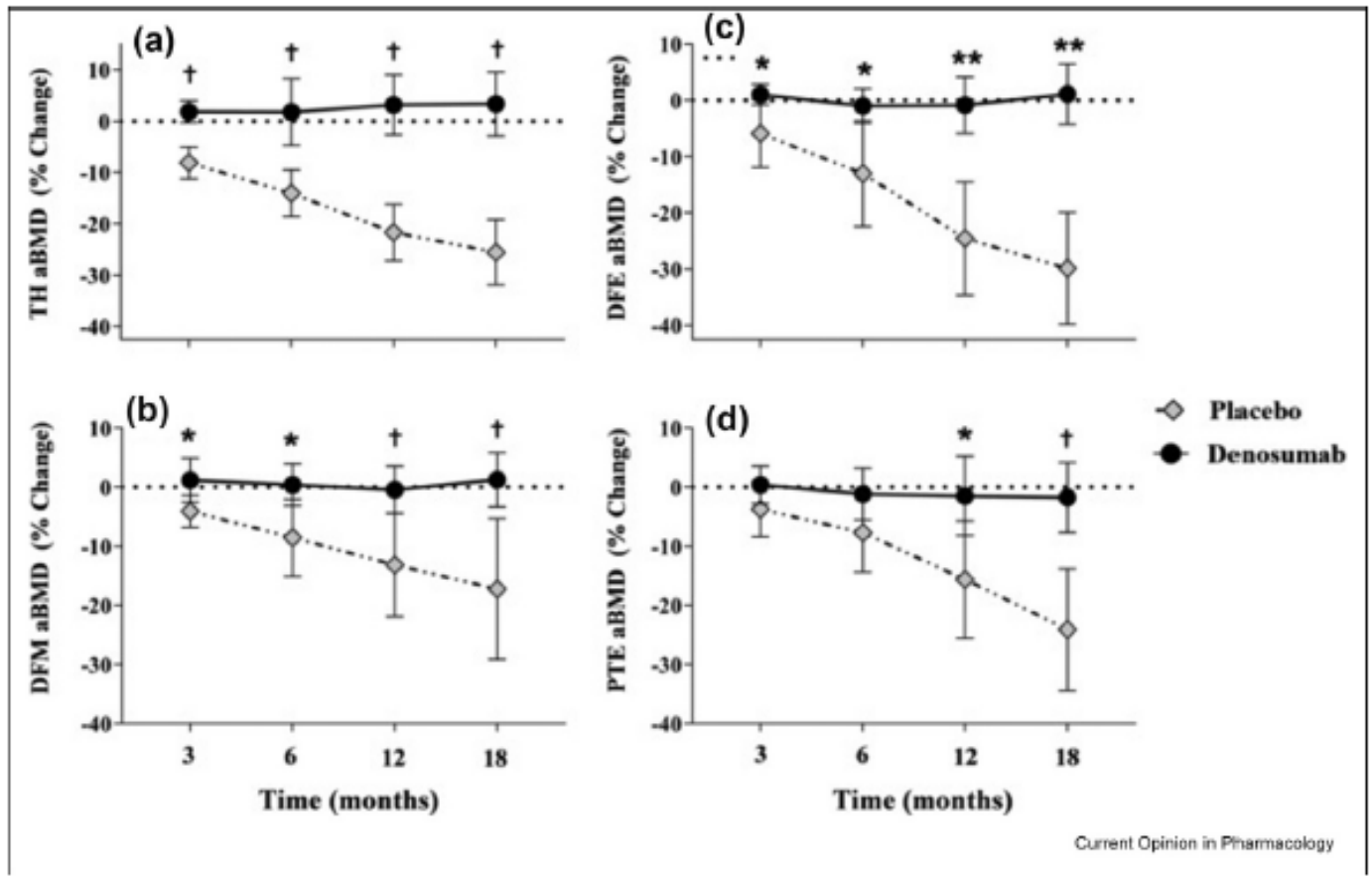
SHORT COMMUNICATION

Denosumab increases sublesional bone mass in osteoporotic individuals with recent spinal cord injury

L. Gifre[1] · J. Vidal[2] · J. L. Carrasco[3] · A. Muxi[4] · E. Portell[2] · A. Monegal[1] · N. Guañabens[1,5] · P. Peris[1,5]

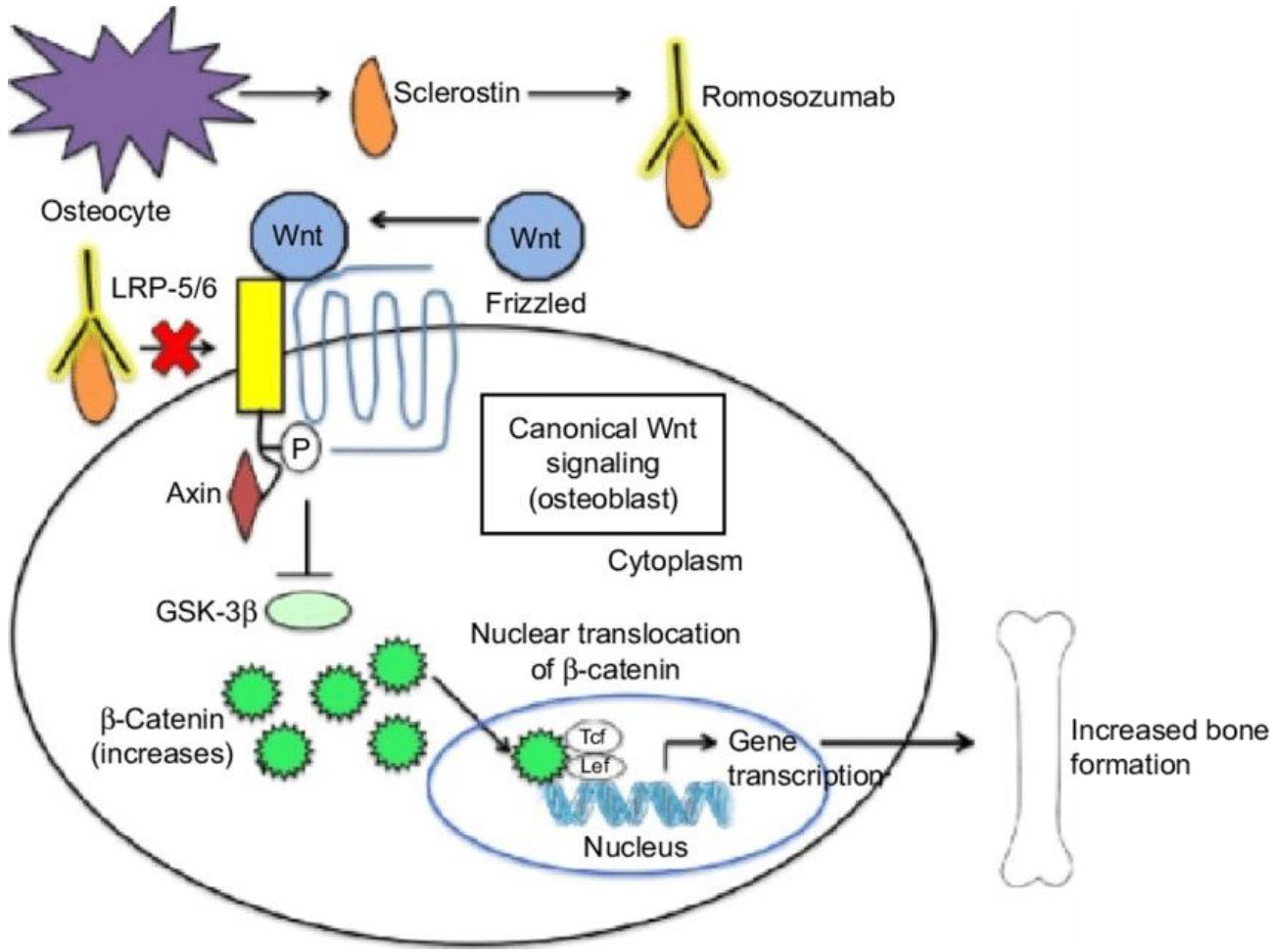


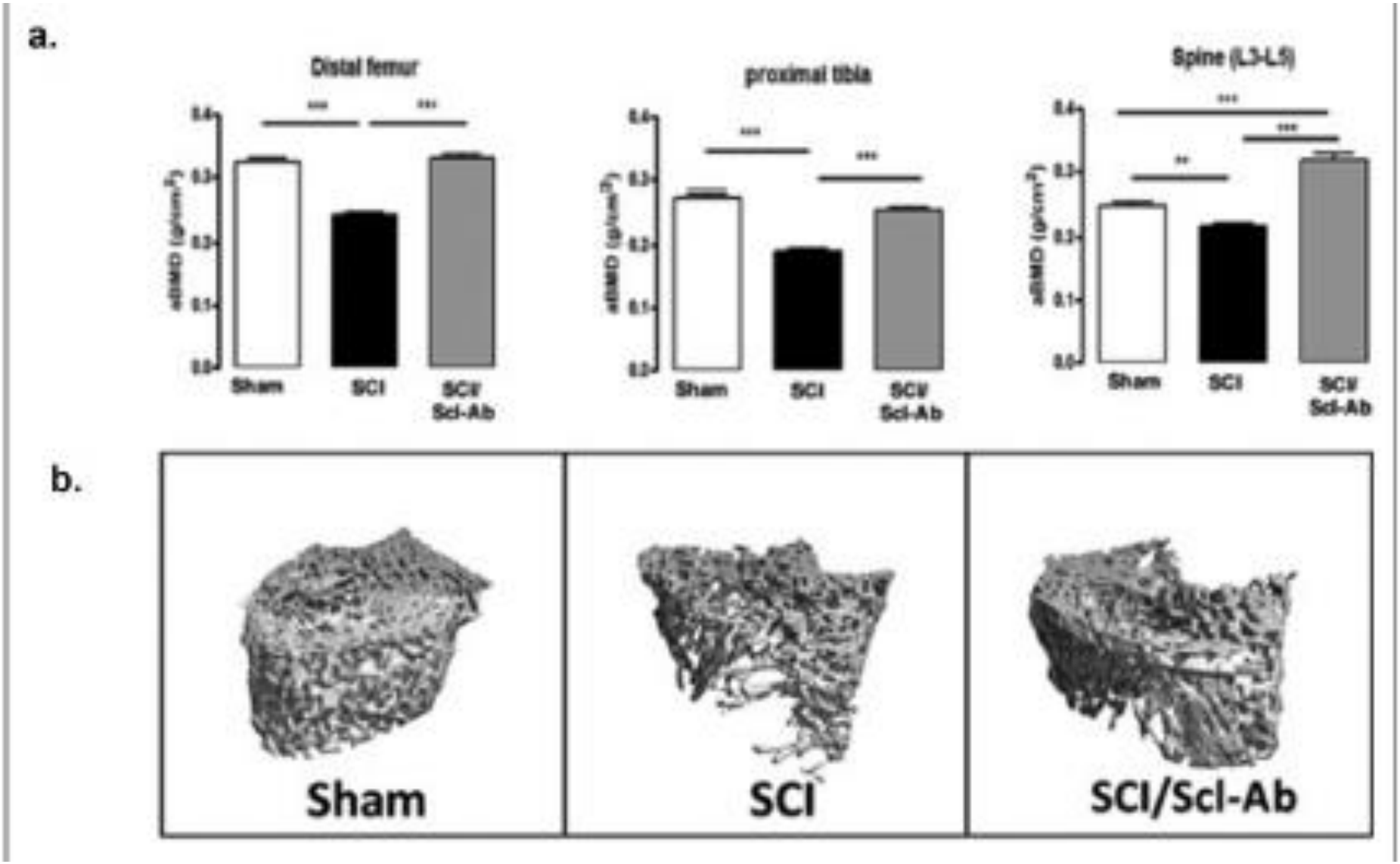
Cirnigliaro CM, et al.: Administration of denosumab preserves bone mineral density at the knee in persons with subacute spinal cord injury: findings from a randomized clinical trial. JBMR Plus 2020, 4, e10375.



Percent change in BMD at the hip and knee after treatment with denosumab. Percent change from baseline in areal BMD (aBMD) at the **(a)** total hip (TH), **(b)** distal femur metaphysis (DFM), **(c)** distal femur epiphyses (DFE), and **(d)** proximal tibia epiphysis (PTE). Percent change from baseline for denosumab versus placebo at the respective time points: $^{*}p < 0.05$; $\dagger p < 0.01$; $^{**}p < 0.001$. Reproduced with permission from Cirnigliaro, et al.[68]

Romosozumab





Effects of sclerostin antibody on bone mass and architecture after acute spinal cord transection. (**a**) aBMD for the indicated sites determined by regional analysis of images acquired by DXA scanning. n = 9–13 per group. (**b–h**) Effect of Scl-Ab on trabecular architecture of the distal femur as assessed by μCT. (**b**) Representative three-dimensional images of trabecular microarchitecture. (**c–h**) Measurements are shown for trabecular bone volume over total volume (BV/TV, %) (**c**), trabecular thickness (Tb.Th, μm) (**d**), trabecular separation (Tb.Sp, μm) (**e**), trabecular number (Tb.N, mm^{-1}) (**f**), Conn.D (mm^{-3}) (**g**), and SMI (range from 0 to 3, with 0 = platelike and 3 = rodlike) (**h**). Data are expressed as mean ± SE. n = 6–7 per group. $^{*}p < 0.05$, $^{**}p < 0.01$, and $^{***}p < 0.001$ versus the indicated group. aBMD, areal BMD; Conn.D, connectivity density; DXA, X-ray absorptiometry; SMI, structure model index. Reproduced with permission from Qin et al.[80]

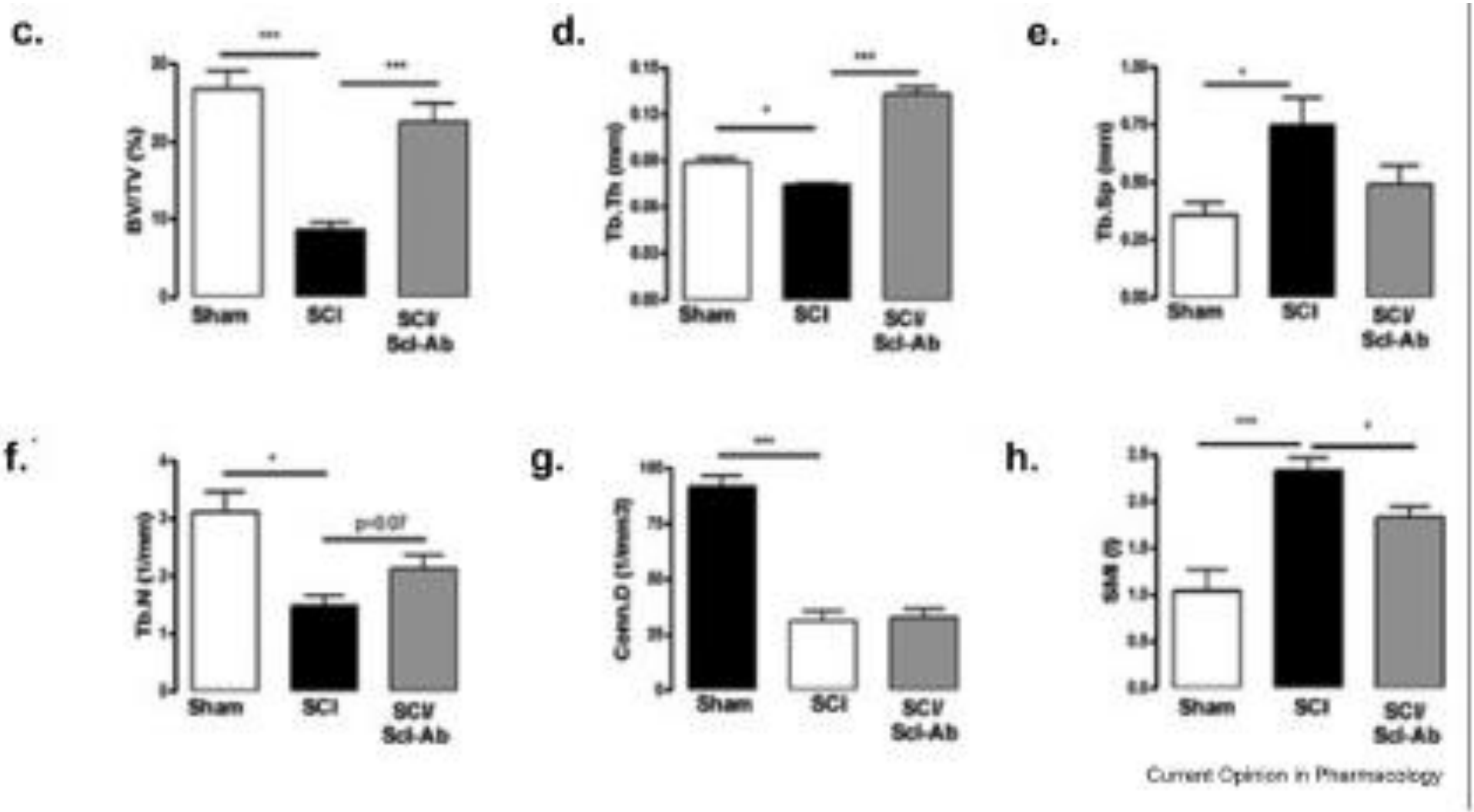


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Table 2
Recommendations for prevention and therapy for SCIs bone impairment.

Recommendations for prevention and therapy for SCIs bone impairment*					
according to SCI phase	implemented diet and lifestyle modifications	vitamin D supplement if vitamin D <20ng/ml	calcium according to international guidelines	Bisphospho nates (per os and i.v.)	FES in SCIs with intact lower motoneuron
1. basic therapy in "persons at risk", including wheel-chair bound paraplegics (prevention).	+	+	+	-	-
2a. therapy (acute phase)	+	+	+	+	-
2b. therapy osteopenic Z scores (chronic phase)	+	+	+	-	-
2c. therapy osteoporotic Z-scores in non-ambulatory persons (chronic phase)	+	+	+	+	+
2d. therapy osteoporotic fracture due to SCI	baseline and specific therapy should be considered in accordance with the recommendations of the DVO Guideline				

*For patients with AIS D who can walk, reference is made to international guidelines *(82)*. Abbr: SCI, Spinal Cord Injury; FES, Functional Electrostimulation, DVO, Dachverband Osteologie.

At what BMD value / T score / Z score etc. is prevention and treatment of osteoporosis indicated in SCI? NOT CLEAR

- Prophylactic osteoporosis-based therapy in "risk individuals", including **wheelchair bound SCI subjects** usually (with severity grades AIS A-C) because of their bone loss promoting pathophysiology.
- *Do we need to wait fractures to occur or to reach fracture threshold??*

- For patients with AIS D who can walk, reference is made to the WHO guidelines

- AIS E *