

# New Insights about the Relationship between Bone Strength and Muscle Strength

H.Schießl, J. Willnecker Ph.D.

Stratec Medizintechnik GmbH

Durlacher Str. 35

D-751 72 Pforzheim, Germany

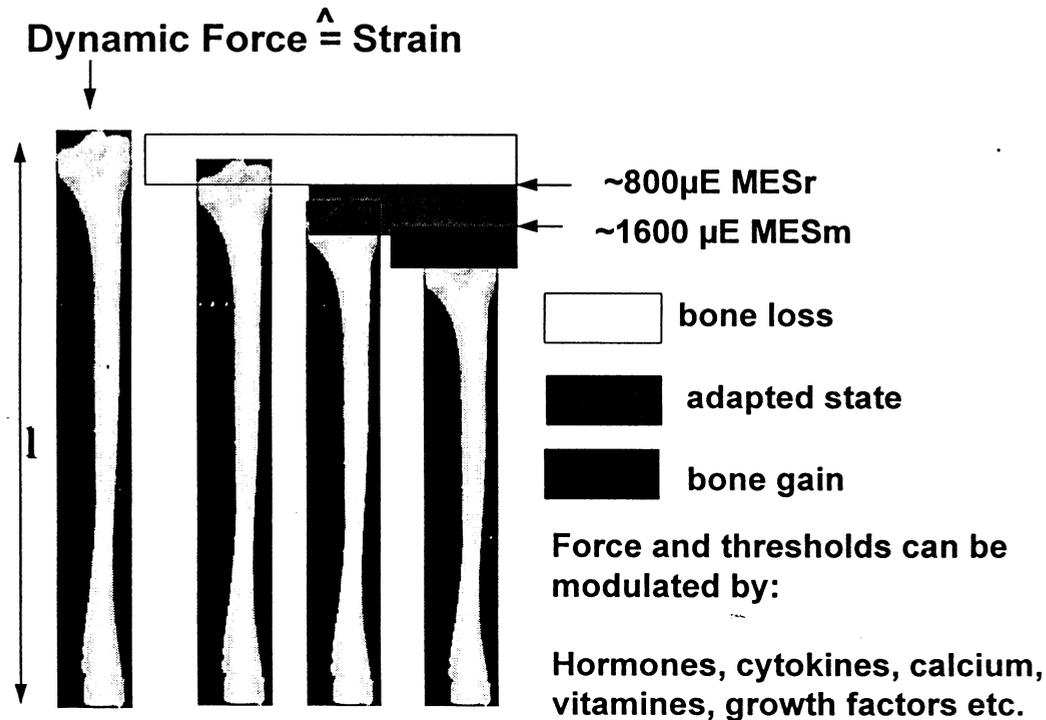
## **Abstract**

According to the mechanostat theory, the mechanical loads on bones help to determine bone strength, and the largest loads come from muscle forces. Indeed, recent studies found that muscle strength and the bending strength of bone are highly correlated ( $r > 0.93$ ), and bone „mass“ and muscle mass likewise ( $r > 0.93$ ). Two strain thresholds seem to help to control bone strength. If the mean voluntary loads on bone do not exceed the minimum threshold for remodeling (MESr), remodeling removes bone until this threshold is exceeded. Between 800 and 1600  $\mu$ Strain bone is preserved. If mean loads on bone regularly exceed 1600  $\mu$ train Strain ( minimum threshold for modeling MESm) bone is added to make it stronger. This suggests that any physical training that does not exceed 1600  $\mu$ train will not increase bone strength.

Growing muscle strength and body weight in children can cause bone strains that exceed the modeling threshold. This could help to explain the active bone modeling and increases in bone „mass“ and strength that occur in growing children. Data in an Argentine absorptiometric study show that in children bone and muscle mass both increase linearly until puberty, but in girls at 12 years of age bone „mass“ begins increasing faster than muscle mass. A similar but smaller increase occurs in boys at age 15. This suggests, estrogen may make girls store more bone than needed for strictly mechanical reasons, possibly to provide calcium stores during lactation after pregnancy.

## Introduction

Postmenopausal bone loss is regarded as a main factor for increased fracture risk in women. Estrogen supplementation is commonly used to prevent this bone loss. But the effects of increased estrogen secretion on bone in girls at puberty have not been well described.



**Fig. 1**

*If bone is compressed, it is shortened. The change in length  $\Delta l$  divided by the original length  $l$  is called strain. The unit is microstrain ( $\mu$ E). If bone strains do not exceed the threshold for remodeling (MESr) centered at around 800  $\mu$ E remodeling removes bone. Between 800 - 1600  $\mu$ E bone is preserved. This corresponds to loads between 11 and 22 N/mm<sup>2</sup>. If strains exceed the threshold for modeling (MESm) at around 1600  $\mu$ E, modeling adds bone.*

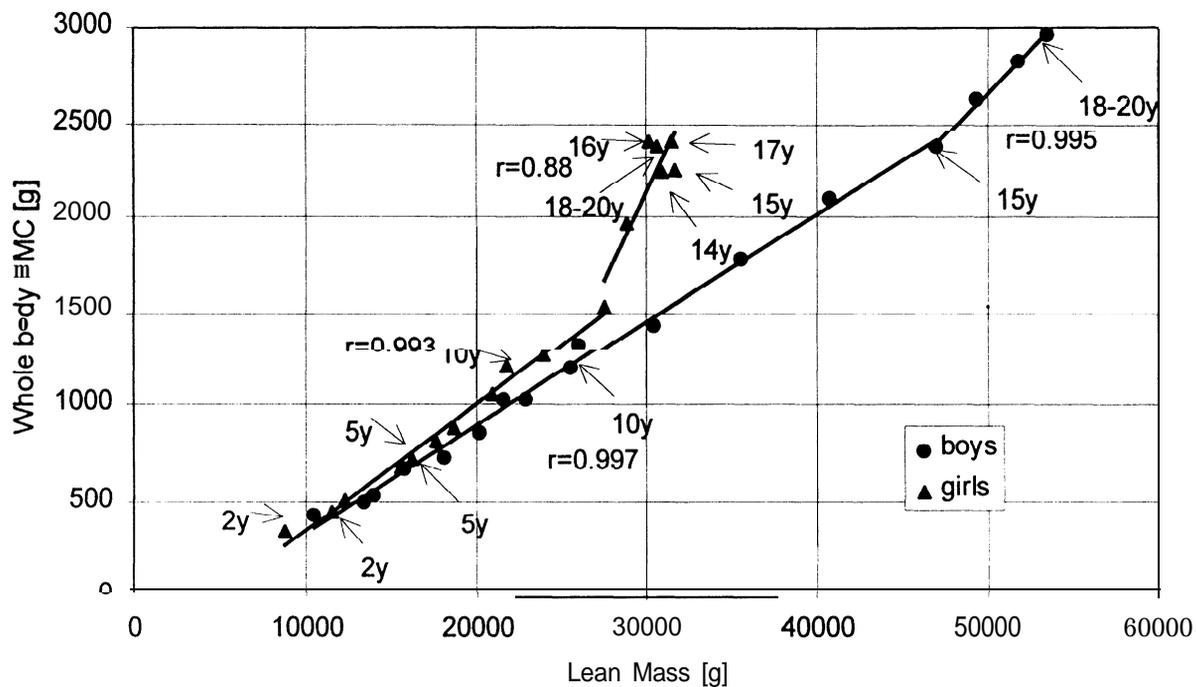
In the Utah paradigm of skeletal physiology [1], bone strength is controlled by mechanical loads on bone. These loads arise from muscle forces rather than body weight [2]. Thus, bone and muscle strength should be highly correlated. This could indeed be shown [3]. Loads create a change of the length on bone which means that under a certain load the bone is deformed. The change in length divided by the original length is called strain. The strains are commonly expressed as microstrains ( $\mu$ E). 10000  $\mu$ E are equivalent to a deformation of bone by 1% of its original length.

Two separate strain thresholds control bone loss or bone gain [4]. Below the threshold for remodeling (MESr) centered at about 800  $\mu\text{E}$  remodeling removes bone where it touches marrow. In this disuse state bone strength is reduced until MESr is exceeded again. Where strains exceed the modeling threshold MESm, modeling adds bone to increase bone strength and „mass“. This threshold ranges from 1500 to 2000  $\mu\text{E}$  [2]. Here bone strength is increases until mean strains stay below MESm. For comparison, the fracture limit of bone is around 17,000  $\mu\text{E}$ . Between MESr and (MESm) bone is preserved. (Figure 1)

If estrogen tends to lower MESr, after menopause that threshold would increase again. If so, loss of bone next to marrow should begin at menopause. This would make bone weaker and identical forces will now result in a higher strain When they increased to the level of the new remodeling threshold, bone loss should be reduced and tend to plateau. This suggests the opposite effect should occur at puberty in girls: With the start of estrogen production MESr should be reduced and bone resorption next to marrow should be slowed down. Therefore women from puberty until menopause should acquire more bone than needed for pure mechanical reasons A study from Zanchetta et al. 1995 [5] supplies information that could test that idea.

### **Explanation of the results from the Zanchetta study**

In 1995 Zanchetta et al [5] reported measurements of total body bone „mass“ and whole body lean mass with dual energy X-ray absorption in 778 children and adolescents from 2 to 20 years of age. (Table 1) Since total body bone „mass“ is a surrogate for bone strength and whole body lean mass as a surrogate for muscle strength, such data might show the relationship between muscle and bone strength, and also any changes that might occur during puberty. Figure 2 plots those data, with lean body mass on the x axis and total body bone mineral content on the y axis. Each point represents the mean value of the **age** groups in one year intervals. Until



This graph from Argentine data [5] plots the grams of total body mineral content (TBBMC) on the vertical axis that correspond to the grams of lean body mass (LBM) on the horizontal axis, as determined by a NORLAND XR 26 using dynamic filtration. Crosses: girls. Open circles: boys. The LBM provides an index of muscle strength. From the left to the right, each data point represents the mean value of a one-year age group. (Reproduced by permission: Schießl H, Frost HM, Jee WSS, Perspectives: Estrogen and bone- muscle strength and „mass” relationships. Bone (in press) 1997)

**Table 1**

Age	BOYS			GIRLS		
	n	Lean mass (g)	Total BMC(g)	n	Lean mass(g)	Total BMC(g)
2	6	10510	431, 3	5	8730	344, 54
3	10	13380	494, 2	13	11530	446, 38
4	16	13960	526, 6	15	12300	503, 15
5	15	15710	665	17	15610	671, 33
6	17	18140	723, 8	21	16210	716, 9
7	25	20160	855, 9	22	17620	813, 15
8	24	21630	1024, 3	33	18650	878, 4
9	26	22920	1023, 03	37	20930	1049, 12
10	37	25530	1186, 01	49	21810	1196, 85
11	23	26060	1334, 68	34	23930	1257, 3
12	24	30490	1438, 82	29	27510	1532, 6
13	28	35540	1779, 66	35	28860	1963, 73
14	24	40780	2094, 57	23	31710	2238, 55
15	22	46960	2364, 89	31	30900	2228, 1
16	17	49300	2633, 55	16	30220	2397, 22
17	12	51760	2825, 22	26	31540	2396, 88
18-20	19	53470	2964, 9	19	30630	2368, 11

puberty, bone and lean mass increase similarly in boys and girls. But at about 12 years of age, in girls bone „mass“ began increasing faster relative to lean mass than in boys. A similar but smaller increase probably began in boys at around 15 years of age. Since both bone and muscle mass plateaued in girls at age 15 years but were still increasing in 20 year old males, the latter had more muscle and bone than 20 year old girls.

In sum, before puberty both boys and girls added about 50 g of whole body mineral content per kg of lean body mass. At puberty this ratio changed to 55 g of bone „mass“ per kg lean mass in boys, and to 77 g of bone „mass“ per kg of lean mass in girls. This difference between girls and boys at puberty should be much larger than any errors in the absorptiometric measurements.

### Comments

The study clearly shows a highly linear dependence of muscle mass and bone „mass“ as surrogates for muscle force and bone strength. Further it shows that the response of bone to muscle force can be influenced by hormonal action. In girls at puberty one could infer the estrogen can make the skeleton store more bone that is required for mechanical needs alone., possibly to meet the demands of lactation after pregnancy [6] If so, when estrogen secretion declines at menopause that bone should be removed, and then further losses of bone should tend to plateau to age-normal levels. To repeat, that menopausal related bone loss and its eventual plateau is well known. In support of that analysis , De Scheper at al. [7] independently found a high correlation between bone and muscle mass. Still, since bone and muscle mass are only surrogates for bone strength and muscle strength further as well as direct studies of those strengths would seem desirable. Bone strength indices obtained by peripheral quantitative computed tomography (pQCT) can provide more reliable noninvasive estimates of bone strength than current DXA techniques and software [8]. Also, in humans muscle strength can be measured directly with grip testers or as torque exerted across joints like the elbow, hip and knee [3].

As Burr recently noted [9], these relationships between muscle and bone strength, and the effects of hormonal and other nonmechanical agents on them , inject a new dimension into former ideas of the determinants of bone strength, „mass“ and health. That incites reassessment of some of those former ideas

Acknowledgments: We wish to thank Dr. H.M. Frost for help in preparing this manuscript.

## Glossary \*\*

Because some terms have vague or even different meanings in the medical literature, their meanings **in this** text follows.

**bone „mass“:** The amount of bone tissue in a bone or skeleton, preferably viewed as a volume minus the marrow cavity. In absorptiometry it does not mean mass as used in physics [8]. When in quotes in this text it has the absorptiometric meaning. DXA-based determinations of bone „mass“ and density only estimate the **contribution of bone „mass“** to bone strength. They do not account **for the equally important , and sometimes more important, contribution of** bone architecture to bone strength. The pQCT technique can account for both contributions. [3,8]

**modeling:** the independent resorption and formation modeling drifts that can increase bone strength and „mass“, and that determine the cross sectional size and shape of bones and trabeculae. Modeling adapts bones to their mechanical loads in ways that prevent voluntary activities from breaking them or make them hurt throughout life [4,1 1]. The former idea that the osteoblast alone control additions to bone „mass“ is no longer tenable, although it persists [4,12,13].

**remodeling:** different meanings of this term in the literature cause some confusion. Here it means turnover of bone in small packets by remodeling BMUs (Basic Multicellular units) [4,1 1]. Pre-1964 literature did not distinguish modeling from remodeling and lumped them together as „remodeling“. Some authors still do that, which can be confusing. However while drifts and BMUs seem to create and use the same kinds of osteoblasts and osteoclasts to do their work, in different parts of the same bone at the same time the osteoblasts osteoclasts in drifts and BMUs can even oppositely to the same stimulus [4]. The former idea that osteoclasts alone control losses of bone is no longer tenable, although it too persists [4,12,13]

**strain:** the deformation or change in dimensions and/or shape caused by a load on any structure or material. It includes stretching, shortening, twisting an/or bending.. Special gages can measure it in bone in the laboratory and in vivo. Loads always cause strains, even if very small ones. [4,1 1]. Biomechanicians often express strain

in microstrain units, where 1000 microstrain in compression would shorten a bone by 0.1% of its original length, 10000 microstrain would shorten it by 1% of that length and 100000 microstrain would shorten it by 10% of that length (and break it) [10,1]. Strain seems to be an important signaling mechanism in skeleton that helps to control their structural adaptations to their mechanical usage.

**strength:** the load or strain that, when applied once, usually fractures a bone (also, the „ultimate strength“). Normal lamellar bone's fracture strength is a strain of 25,000 microstrain. That corresponds to a change in length of 2.5%, i.e., from 100% of its original length to 97.5% of that length under compression, or 102.5% of it under tension [11]. In normal bone 25,000 microstrain corresponds to an ultimate or fracture stress of 17,000 pounds per square inch or 120 megapascals.

**stress:** the elastic resistance of the intermolecular bonds in a material to being stretched by strains. Loads cause strains, which then cause stresses (while some think stress causes strain, that is like saying a lake causes the river that fill it). Three principal stresses include tension, compression and shear. Stress cannot be measured directly but must be calculated from other information that often includes strain. Bone's stress strain curve is nonlinear.

\*\* taken from [10]

## References

[1] Jee WSS. Since 1965 this Professor of Anatomy at the University of Utah School of Medicine organized uniquely seminal multidisciplinary Hard Tissue Workshops. Sponsored by the University of Utah, world wide they probably influenced how people think about and study skeletal physiology and disease more than any other meeting in this century. The Utah paradigm of skeletal physiology arose there, with input and critique from hundreds of international authorities in many disciplines. A general summary of that appeared in [IO], but numerous articles by many authors. have described parts of it since 1985

[2] Frost HM, Perspectives: Why Do Long Distance Runners Not Have More Bone? A Vital Biomechanical Explanation and an Estrogen Effect. J Bone Miner Metabol 1997;15:9-16

[3] Schießl H, Ferretti JL , Tysarczyk-Niemeyer G, Willnecker J, Noninvasive Bone Strength Index as Analyzed by Peripheral Quantitative Computed Tomography (pQCT) . In: Pediatric Osteology . New Developments in Diagnostics and Therapy E. Schonau (Ed.) Elsevier, Amsterdam 1996;pp 147-160

[4] Frost HM Bone development during childhood: Insights from a new paradigm. In: Pediatric Osteology . New Developments in Diagnostics and Therapy. E. Schonau (Ed.) Elsevier, Amsterdam 1996;pp. 3-39

[5] Zanchetta, JR, Plotkin H, Alvarez-Figueira ML Bone Mass in Children: Normative Values for the 2-20 Year - Old Population. Bone 1995;16 (Suppl): 393-399

[6] Schießl H, Frost HM, Jee WSS, Perspectives: Estrogen and bone- muscle strength and „mass“ relationships. Bone 1997; (in press)

[7] De Schepper J, Dab I, Louis O, Acquisition Du Contenu Mineral Osseux Chez L 'Enfant Atteint De Mucoviscidose: Correlation Avec Les Facteurs Cliniques Et Anthropometriques. Secondes Recontres d'Osteodensitrometrie Namur, Belgique 1997; p 90

[8] Ferretti JL; Capozza RF Zanchetta JR Mechanical Validation of a Tomographic. (pQCT) index for Noninvasive Estimation of Rat Femur Bending Strength. Bone 18: 97-102

[9] Burr DB, Muscle strength, bone mass, and age related bone loss. J Bone Miner Res 1996;12: 1547-1551;1997

[10] Frost HM, Introduction to a New Skeletal Physiology . Vols I,II. The Pajero Group, Inc. Pueblo, Colorado

[11] Martin RB, Burr DB Structure Function and Adaptation of Compact Bone. Raven Press, New York 1989

[12] Brown W, Haglund k, Landmarks J NIH Res 1995;7: 54-59

[13] Parfitt AM, Problems in the application of in vitro systems to the study of human bone remodeling. Calcif Tiss Int 1995;56 (Suppl 1):S5-S7