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Perspective Article

Hylonome

On the strength-safety factor (SSF) for load-bearing skeletal organs

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Abstract

The strength of healthy postnatal mammalian load-bearing bones, growth plates, joints, fascia, ligaments and tendons exceeds the minimum strength needed to keep voluntary mechanical usage from breaking or rupturing them or from causing arthroses. Thus, they have a *strength-safety factor* (SSF). Some general features of the physiology in the Utah paradigm of skeletal physiology can explain two things: *(i)* Why load-bearing bones should have an SSF, *(ii)* and why its numerical value should ≈ 6 in healthy young adult mammals. The number and kinds of studies and facts that revealed those two things for load-bearing bones do not yet exist for the extraosseous load-bearing organs that are made with cartilage and collagenous tissue. However, clinical-pathologic observations suggest the latter organs' SSFs should depend on features analogous to those that create SSFs for load-bearing bones. If so, the physiology on which bone's SSF depends could suggest directions for future studies of the SSF determinants of load-bearing extraosseous organs. Biomechanicians currently favor strain above stress when discussing biomechanical roles in the functional adaptations of bones to mechanical loading. However, an SSF is best expressed in stress terms, so a Table in this article provides corresponding strain/stress/unit-load values for bone's three important thresholds, and for its ultimate strength.

Keywords: Biomechanics, Bone, Joints, Ligaments, Tendons, Modeling, Growth

I. Introduction

In healthy postnatal mammals, it is an elementary observation that their load-bearing bones, joints, growth plates, fascia, ligaments and tendons have enough strength to keep voluntary mechanical usage from fracturing or rupturing them or from causing arthroses[1]. Thus, such organs have a strength-safety factor (SSF)[2-5]. Table 1 lists and defines the abbreviations in this article.

Stress equals force per unit area, and unit loads such as kg/mm^2 of a load-bearing organ's cross section area provide one way to express stress. A stress of 100 mpa (megapascals) corresponds to a unit load of 100 Newtons/mm^2 and to ≈ 10 kg/mm^2 [6]. Given that, dividing the ultimate stress (Fx) of a load-bearing skeletal organ by the typical peak stress (PS) caused by a subject's voluntary physical activities can define and equal that organ's strength-safety factor (SSF)[3], so:

$$Fx \div PS = SSF \qquad \text{Relation 1}$$

Two questions about such organs concern this article: What biologic mechanisms create the SSF? How many times stronger is a healthy load-bearing organ than the minimum needed to keep voluntary loads from breaking it?

Herein, "load-bearing" organs exclude bones like the cranial vault and the ethmoid, nasal and turbinate bones. Such organs also exclude the cartilages in the nose, ear and trachea, and the collagenous periosteum and perichondrium. Herein, "voluntary" means intentional, not due to trauma and not due to jumping from heights. Therefore, such loads would include muscle forces.

This article summarizes some physiology that can explain why load-bearing bones have an SSF, and that can reveal the SSF's magnitude for bones. "Connecting the dots" between many ideas and facts from many lines of inquiry provided by many people revealed those features and led to the Utah paradigm of skeletal physiology[3-6] (over 80 years ago, connecting the dots between many kinds of facts in physics that many people found let a Swiss postal clerk realize that $E = mc^2$). This article concludes by suggesting that features analogous to those for bone and bones could explain SSFs for extraosseous load-bearing organs (joints, growth plates, fascia, ligaments, tendons).

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Abbreviations used in this article

BMU:	basic multicellular unit of bone remodeling
Fx:	a load-bearing organ's ultimate strength expressed as a stress
kg:	kilograms of force
M:	the meter, a unit of length
MDx:	microscopic fatigue damage
MESm:	a structural tissue's modeling threshold range expressed as a stress
MESp:	the microdamage threshold range
MESr:	the threshold range that turns disuse-mode remodeling on
mpa:	megapascals, = millions of Newtons/M^2
PS:	typical peak stress caused by voluntary activities, not by trauma
SSF:	a load-bearing organ's strength-safety factor
≈:	approximately equals

Table 1.

Herein, "load-bearing" distinguishes intact organs from the materials or tissues that form them. The cited references provide more details for readers who might want them, or indicate where to find those details.

II. Summary of the bone physiology

1) Four physical factors in mammalian postnatal whole-bone strength[6-12].

These factors include bone's properties as a material (the *material properties* factor); the amount of microdamage (MDx) in the bone (the *MDx* factor); the amount and kind of bone in a bone's cross section – compacta and spongiosa, woven and lamellar bone – (the bone *mass*" factor); and the bone's cross and longitudinal shape and size, and the distribution and orientation of bone tissue within it (the geometry or *architectural* factor).

When compared to effects of the other three factors on whole-bone strength, bone's material properties differ relatively little with age, in different bones and species, and in disease (osteomalacia excepted[9]). Expressed as a stress, cortical bone's ultimate strength ≈ 120 mpa in healthy young adult mammals (a bit less in children and a bit more in aged adults)[9,13-15]. Herein, "whole bone" distinguishes bones as intact organs from bone as a material or tissue.

Similar physical factors should determine the strength of extraosseous load-bearing skeletal organs.

2) Biologic determinants of postnatal mammalian whole-bone strength[3-6].

(i) One multicellular mechanism called *modeling* by separate formation and resorption drifts[16] has the function of increasing whole-bone strength.

(ii) The "disuse mode" of a second multicellular mechanism called *BMU-based remodeling*[16] has the function of reducing whole-bone strength.

Mechanical loads on bones cause corresponding *strains* that generate corresponding *signals* which some cells can detect and respond to[6,17]. Those signals can control how the above modeling and remodeling functions affect a load-bearing bone's strength. Where bone strains approach or exceed one threshold range called the *modeling threshold* (MESm), the modeling function usually turns on to strengthen a bone. When strains stay below a lower *remodeling threshold* range (MESr), the disuse-mode remodeling function usually turns on to reduce that bone's strength. Bone's MESm range seems to center near 20 mpa, while its MESr range may center near one to two mpa[3].

Those two thresholds have at least three effects.

(i) During voluntary activities the thresholds would make the typical largest stresses of a normally-adapted load-bearing bone approach but not exceed the modeling threshold expressed as a corresponding stress. Thus, in normally-adapted bones the PS (peak stress) in Relation 1 above would equal the MESm expressed as a stress, so that relation can be rewritten thus:

$$Fx \div MESm = SSF \qquad \text{Relation 2}$$

(ii) Those thresholds would make the *largest* voluntary bone loads have disproportionately greater effects on whole-bone strength than smaller loads no matter how frequent[6,17]. Trauma excepted, *muscle forces* instead of body weight cause the largest loads on load-bearing bones, and thus their largest stresses too[3,6,9].

(iii) The MESm's effect on bone modeling would make healthy young adult load-bearing bones strong enough *relative to the typical peak voluntary loads on them* to keep their stresses within or below the lower boundary of the MESm range.

Values for bone's thresholds and ultimate strength
(in microstrain, stress and unit load units)*

MESr:	50-100 microstrain; 1-2 mpa; ≈ 0.1 kg/mm^2
MESm:	1,000-1,500 microstrain; ≈ 20 mpa; 2 kg/mm^2
MESp:	3,000 microstrain; ≈ 60 mpa; 6 kg/mm^2
Fx:	25,000 microstrain; 120 mpa; 12 kg/mm^2
*:	values for analogous features for chondral and collagenous tissue organs are currently unknown. The values cited for bone apply to cortical bone and are based on currently available information.

Table 2.

Repeated bone loads and strains (and stresses) cause microscopic fatigue damage or *microdamage* (MDx) in bones[8,18-20]. MDx increases bone fragility without affecting bone architecture or "mass" in any way. Bone has an operational *MDx threshold* range (MESp). Where bone strains and stresses stay below it remodeling BMUs can repair whatever MDx occurs, but strains in or above the MESp range can cause enough MDx to escape repair, accumulate, and cause nontraumatic fractures[8,20]. Hence, pseudofractures in osteomalacia, stress fractures in athletes, and spontaneous fractures in true osteoporoses[3,8]. Bone's MESp range seems to center near 60 mpa.

MDx also occurs in load-bearing organs made from cartilage and collagenous tissue.

Collectively the above things plus some others form a tissue-level negative feedback system called bone's *mechanostat*[6,16,21]. It would orchestrate the biologic activities that make load-bearing bones strong enough so voluntary loads do not break them[3]. If so, persistently strong muscles should usually associate with correspondingly strong bones, and persistently weak muscles should usually associate with correspondingly weak bones. Both things are true[1].

Table 2 lists values for the above three thresholds, and for bone's ultimate strength, in corresponding microstrain, stress and unit-load units. Note that bone modeling and remodeling have further functions[22,23].

III. Bone's SSF

When expressed as unit loads or stresses, the SSF in properly-adapted young adult load-bearing mammalian bones would ≈ 6, since Fx ÷ MESm = 120 ÷ 20 = 6 (see Relation 2 and Table 2). Thus, such bones are about six times stronger than the minimum needed to keep voluntary loads from breaking them. A lower SSF value of ≈ 2 suggested earlier[24] used bone's yield point instead of its MESm to calculate it, and its yield point is a range centered near 60 mpa, like bone's MESp[3]. The yield-point stress is well above the largest allowed stress in healthy bones that defines and equals the MESm.

Question: Why not express the SSF in strain terms? Using the microstrain values in Table 2, Fx ÷ MESm = 25,000 ÷ 1000 = 25(!). Yet healthy bones are clearly not 25 times stronger than needed to carry the typical largest loads on them. That value of 25 may be one way to define an SSF but it does not answer the question that concerns us here. To wit: how much stronger is a healthy bone than the minimum needed to carry its largest voluntary total loads without breaking?

IV. Comments

The threshold "ladder".

Whether expressed as stresses, unit loads or microstrains, bone's MESr lies below its MESm, which lies below its MESp, which lies below its ultimate strength. Thus, where "E" signifies the typical peak stresses in a normally-adapted young adult bone:

$MESr < \text{“}E\text{”} < MESm << MESp <<< Fx$ **Relation 3**

Relation 3 has been called a *general biomechanical relationship*[25].

Please note four things. (i) Section #5 below suggests that relationship would apply to healthy estraosseous load-bearing skeletal organs after birth. (ii) Above its Hookean range (above ≈ 60 mpa or 3000 miscrostrain), bone's stress-strain relationship becomes a curved line, and doubling the strain no longer doubles the stress too[9,15] (the "Hookean range" is that elastic range within which strain and the resulting stresses stay linearly proportional to each other). (iii) A modeling threshold that lies below a load-bearing organ's ultimate strength *must* create an SSF. Why? The mechanostat would normally adjust whole-bone strength to keep voluntary activities (meaning muscle strength – and power?) from causing peak stresses above the MESm range. When that range lies below an organ's ultimate strength (when $MESm << Fx$) an SSF must result, regardless of however else one might choose to name it. (iv) Still, the mere existence of an SSF does not prove an MESm exists and/or causes the SSF.

Some set point and aging considerations.

Cybernetic and other considerations[26,27] suggest the modeling and remodeling thresholds reside in some skeletal cells as genetically-determined internal standards[17,27]. Their values might differ in some people for genetic reasons, and age and some humoral agents such as hormones, vitamins, minerals, drugs, etc., might modify their values[6]. Herein, the centers of those threshold ranges would define their set points.

In principle, modestly supranormal set points could lower bone's SSF from 6 to, say, 4. If so, affected bones would be more susceptible to fractures without significant abnormalities in composition or histology. If the MESm set point increased modestly while the MESp remained normal, affected bones would show a tendency to excessive MDx and stress fractures. Why? An increased MESm would allow larger bone strains and stresses, which usually increase MDx[19]. On the other hand, modestly subnormal MESm and MESr set points would make affected bones stronger than normal and less susceptible to traumatic and stress fractures and MDx[6].

In that regard, clinicians do occasionally see patients who seem unusually susceptible to traumatic or stress fractures without any known abnormalities in body or bone biochemistry, histology or composition[1]. Also, R. Recker described patient cohorts in whom stronger and "denser" bones than normal occur[28]. Whether these set point ideas explain such things is currently unknown, but the idea seems attractive.

The modeling activities that increase whole-bone strength during growth are sluggish, so increasing bone strength can lag behind mechanical needs in children, in whom bone loads from body weight and muscle strength steadily increase[29]. If so, typical peak bone strains from voluntary mechanical usage in children should somewhat exceed those in young adults in whom body weight and muscle strength usually plateau, so bone strength could "catch up" to mechanical needs in those young adults. Those things do occur[6,30]. That means more fractures should occur during childhood than in young adult life, as they do[31,32]. Accordingly that "adaptational lag" in children's functional adaptations to bone loading, especially during the adolescent growth spurt[29], could temporarily decrease the SSF in affected bones.

On strain rate, kind, magnitude, frequency.

Studies and discussions continue of the relative roles of such strain features in controlling the functional adaptations of load-bearing bones to their voluntary loads[6,17,33]. While it seems logical that evolution learned to adapt bone strength to the greatest usual threat to structural integrity and, trauma excepted, that typical peak voluntary loads on bones would provide that usual threat, investigators found that loading frequency and rates might have unexpected but therapeutically useful effects on whole-bone strength[33,34]. Also, in my view the role of shear strains in controlling the functional adaptations of load-bearing skeletal organs has probably been underappreciated[3,22,35].

So in these matters devils still lie in the details, and disagreements persist because reasonable people can interpret the same facts differently. More work must resolve such issues.

On some role(s) of genes.

By the time of birth gene expression patterns *in utero* have created the biologic machinery and "game rules" that control postnatal skeletal physiology. After birth that genetically-predetermined machinery and collection of game rules, called the "baseline conditions" elsewhere[3], make the largest typical voluntary loads determine the strength of most load-bearing skeletal organs. Ergo, determining their strength would represent a genetic-biomechanical duet, not a genetic or biomechanical solo. Current disagreements concern whether genetic or biomechanical factors dominate in that duet.

On extraosseous skeletal load-bearing organs (joints, growth plates, fascia, ligaments, tendons).

After birth bone's mechanostat makes voluntary mechanical usage determine a load-bearing bone's strength. While devils still lie in the details, the experimental and other facts on which that general physiology depends seemed fairly robust in 2002 AD, noting that Michael Parfitt recently called bone's mechanostat "…. the most important unsolved problem in bone biology…."[36].

Many clinical, pathologic and other facts suggest to me that load-bearing extraosseous skeletal organs have general features analogous to those of bones. That is, such organs should have their own modeling, disuse and MDx thresholds, their own MDx detection and repair mechanisms, separate biologic mechanisms to increase and decrease their strengths, their own associated signaling mechanisms and cells, their own but analogous responses to muscle forces, and their own mechanostats[37-43]. Normally they are all normally stronger than needed to endure the voluntary loads on them so they do have their own SSFs. As an example, such things could explain why the strength of a healthy tendon always matches the strength of the muscle that pulls on it.

However, the kinds and number of studies that support the above physiology for bone and bones do not yet exist for extraosseous skeletal organs. Nevertheless I would suggest four things about them here. (i) Those extraosseous organs should have their own mechanostats and all that implies. (ii) Those mechanostats would all have the same goal: making their organs strong enough to keep typical peak voluntary loads from rupturing them or causing arthroses[37,38,41-43]. (iii) Relation 3 and the things it implies should apply to those extraosseous organs too. (iv) Those ideas could help to direct some future studies about the roles of biomechanics in the strength and health of our extraosseous load-bearing skeletal organs.

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