

# Does the anterior cruciate have a modeling threshold? A case for the affirmative

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## Abstract

Growing evidence supports a 1972 proposal that dynamic tension strains of a ligament above a threshold range, but below its ultimate strength, would make its cells synthesize more collagen to thicken and strengthen it. If so, when that strengthening reduced later strains to the bottom of that threshold range this “diametric modeling” would stop. A) Such a mechanism *must* create a “strength-safety factor” that would minimize or prevent voluntary activities from rupturing healthy ligaments, so chiefly injuries would rupture them. B) Such a mechanism should also make the usual largest loads on a healthy anterior cruciate ligament (ACL) determine its strength, and would make smaller loads mixed with large ones have little effect on its strength. C) In principle, when an ACL’s strains exceeded that threshold range, diametric modeling would turn on, strengthen it, and reduce subsequent strains from the same loads. When its strains remained smaller, this mechanically-controlled modeling would turn off. Normal ACLs do have a strength-safety factor so they could have a diametric modeling threshold too, as we now know bone does. In healthy young adult humans available evidence suggests that threshold’s value could lie in the region of 23 Newtons/mm<sup>2</sup> of the ACL’s cross section area. If similar relationships applied to fascia, tendons and other ligaments (I suggest they do), they would form fundamental biomechanical properties of collagenous tissue organs.

**Keywords:** Ligament, Tendon, Fascia, Biomechanics, Knee, Mechanical Influences, Collagen

## I. Introduction

An extensive literature concerns the causes and mechanisms of anterior cruciate ligament (ACL) ruptures, which nearly always follow injuries. That raised the question of why healthy ACLs rarely rupture spontaneously. This article concerns some physiology that could help to answer that question. Technical considerations aside, that physiology could also affect the long-term results and rehabilitation of surgical ACL reconstructions. The physiology is embodied in the still-evolving Utah paradigm of skeletal physiology<sup>1,2</sup> that adds later facts and ideas to former ones. A brief summary follows of its features that bear on this matter. Table 1 lists abbreviations used in the text.

## II. Pertinent features of the Utah paradigm

In 1960 it was generally thought that the health, disorders

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and other features of ligaments and bones depended on their effector cells and regulation by humoral, genetic and other nonmechanical factors (effector cells are fibroblasts in collagenous tissues, and osteoblasts and osteoclasts in bone). Back then no tissue-level mechanisms and functions, and no control by mechanical influences, were recognized<sup>3-5</sup>.

For bones further things became clear after 1960<sup>2,6-8</sup>.

A) Bones do have tissue-level mechanisms, and one of them called “modeling” can increase the strength of load-bearing bones, partly by adding more structural tissue<sup>9</sup>.

B) Loads on bones create strain-dependent signals such as streaming and piezoelectric potentials, fluid streaming effects on cell membranes, some chemical effects and perhaps other things<sup>8,9,12</sup>.

C) Presumably the existence and value of a genetically-encoded threshold signal strength for controlling bone modeling resides in some skeletal cells (which ones are still unknown).

D) Those and/or other cells would compare that threshold to strain-dependent bone signals.

E) When that comparison revealed an error an “error signal” would arise to make modeling correct the error, which would be too little strength for the voluntary loads on

the bone (“voluntary loads” mean those caused by conscious, deliberate efforts; they exclude loads due to injuries and occasional jumps from a height). Lacking an error signal, mechanically-controlled bone modeling would turn off.

F) Various studies put this modeling threshold in the 20 mpa region in healthy young adult bone<sup>2,13</sup>. For comparison, cortical bone’s ultimate strength in young adults lies in the 120 mpa region (somewhat more in older adults and less during growth)<sup>14</sup>.

G) Thus bone’s modeling threshold lies well below its ultimate strength.

A modeling threshold that lies below bone’s ultimate strength should have at least two effects.

a) That arrangement must make a bone stronger than needed for its usual largest voluntary loads, and in the ratio of *ultimate strength* ÷ *modeling threshold* (because that threshold determines the largest normally-allowed stress – and corresponding strain – in healthy bones during voluntary activities). Thus healthy bones have a strength-safety factor.

b) That arrangement must also make the typical largest voluntary loads on a weight-bearing bone dominate the control of its strength (of its stiffness really, but strength provides a convenient surrogate for it here)<sup>15,16</sup>.

For ligaments it was inferred by 1972 that tension strains above a different threshold range should make a ligament’s (or tendon’s) cells synthesize more collagen to thicken, strengthen and stiffen it. That *diametric modeling* would continue until it reduced the size of the strains to the bottom of that threshold range, when it would stop. Hence the 1972 “stretch-hypertrophy rule”: “*Intermittent stretch causes collagenous tissues to hypertrophy until the resulting increase in strength reduces elongation in tension to some minimum level*”<sup>1</sup>. Equally, a room’s cooling system would turn on when the temperature exceeded the thermostat setting and it would continue until the temperature fell below that setting, when it would turn off. In principle that arrangement should have ligament effects analogous to the above “a,b” bone effects, noting that increasing a ligament’s strength would

usually involve increasing its cross section area too<sup>15</sup>.

In 1972 no experimental or other data could validate or negate that idea, but afterwards such things began to accumulate and support it. A summary follows.

### III. Some ACL evidence and considerations

#### 1) On intermittent stretch

*In vitro*, collagenous tissue cells respond to dynamic tension strains in the 10,000-30,000 microstrain region (a 1%-3% stretch) by synthesizing more collagen<sup>17,19-22</sup>. That supports the idea that a strain-dependent threshold strain range could help to control collagen production in response to strains above the threshold. If that happened *in vivo* it would increase an ACL’s cross section area and strength. Since ligaments usually rupture in a region centered near ≈ 70,000-80,000 microstrain (a 7%-8% stretch)<sup>23</sup>, that arrangement could give an ACL a strength-safety factor, as its modeling threshold does for bone.

#### 2) Some pertinent clinical-pathologic observations

A) In adults the ACL in a nonweight-bearing congenitally or neonatally paralyzed lower limb (as in myelomeningocele or poliomyelitis), has less than half the diameter of the ACL in the normal limb<sup>24</sup>, so it should be weaker. Yet the same blood carries the same humoral agents to both ligaments, the cells of which would have the same genome.

B) At birth the ACL has less than half the diameter of normal adult ACLs<sup>24</sup>, so it should be weaker.

C) If, as formerly thought, genetic, hormonal and/or other nonmechanical blood-borne factors instead of a diametric modeling threshold determined the ACL’s diameter and strength, it should have similar diameters and strengths in the congenitally paralyzed and normal lower limbs of children and adults.

D) But it does not so their *differences* could reveal how normal loads affected ACL modeling in the normal limbs.

E) That voluntary physical activities rarely rupture healthy ACLs shows they are stronger than needed for those activities, i.e., they do have strength-safety factors.

F) A modeling threshold below the organ’s ultimate strength would create such a factor, so our ACL could have a diametric modeling threshold too.

That begs two questions: *Could presently available information support that idea? Could it suggest an approximate value for that threshold?* Material in the next two Sections of this article indicates it could.

#### 3) Illustrative ACL values

To one or two significant figures and in metric units, Table 2 lists approximate values for some ACL features of an average healthy, young adult 70 kg. man. The values include the ligament’s minimum cross section area as determined *in situ* by MRI, an intact ACL’s

Acronyms and Abbreviations		
MESm: the modeling threshold range		≈: approximately equal
CSA: cross section area		W: total load at failure
ULVL: usual largest voluntary total load		SF: safety factor
US: ultimate strength as a material		MESfm: the modeling threshold range expressed as a fraction of the organ’s ultimate strength
Symbols for Metric Units of Measurement		
N = Newton	mpa = megapascal	kg = kilogram
M = meter	mm = millimeter	cm = centimeter

Table 1.

ultimate strength as measured by rupturing it *ex vivo* in laboratories, and its typical peak *in vivo* loads during voluntary activities as estimated with the aid of computer modeling<sup>20,23,25-31</sup>. Calculations in Section #4 below use those values.

Table 3 provides conversion factors for some English and metric units of measurements.

*Nota bene:* The voluntary loads on a healthy ACL must lie below its ultimate strength because they do not rupture it; the voluntary loads estimated by different workers also lie below its ultimate strength; *in vitro* studies indicate some threshold strain below the rupture strain can make collagenous tissue cells increase collagen synthesis; and healthy ACLs are stronger than needed for our voluntary activities.

Such things at least support the idea of a diametric modeling threshold. Section #4 below shows a general way to determine its value for any ligament (and tendon too). It also calculates approximate values for the illustrative ACL in Table 2.

4) Some general load-modeling-anatomical relationships for the above ACL

Algebraic relations can express such relationships, using total and unit loads and stresses as the referents. When other factors perturb the accuracy of calculated results appropriate additional terms could provide better values (see Comments, Section #3).

A) The ultimate strength (US) of an ACL's material. This would equal the total tension load (W) that usually ruptures a fresh intact ACL at physiologic temperature and loading rates, divided by the ligament's minimum cross section area (CSA), or:

$$US = W \div CSA \quad \text{Equ. (1)}$$

For the ACL in Table 2,  $US = 1950 \div 35 = 55.7 \text{ N/mm}^2$ ,  $\approx 5.6 \text{ kg/mm}^2$ ,  $\approx 56 \text{ mpa}$ . For comparison, bone's ultimate strength as a material  $\approx 12 \text{ kg/mm}^2$  or  $\approx 120 \text{ mpa}$ .

B) The diametric modeling threshold (MESm) of an ACL's material. Let ULVL mean the Usual Largest Voluntary Loads in tension on an ACL *in vivo*, parallel to its grain or length. Let (CSA) mean its minimum Cross Section Area. Then the center of this threshold's range (its set point) as a stress, and for the ACL tissue as a material, should be:

$$MESm = ULVL \div CSA \quad \text{Equ. (2a)}$$

Mean minimum cross section area ( <u>CSA</u> ):	35 mm <sup>2</sup>
Estimated peak total load in walking down a 19% grade ( <u>ULVL</u> ):	800 N
Mean ultimate strength of the intact ligament ( <u>W</u> ):	1950 N

**Table 2.** Approximate values for the human anterior cruciate ligament in a 70 kg healthy young adult male who is not an athlete\*. \*. Taken from<sup>20,23,25-31</sup>. N = Newtons of tensile force.

For the ACL in Table 2,  $MESm = 800 \div 35 = 22.8 \text{ N/mm}^2$ ,  $\approx 2.3 \text{ kg/mm}^2$ ,  $\approx 23 \text{ mpa}$ . For comparison, bone's modeling threshold in similar referents is thought to lie in the region of  $\approx 20 \text{ N/mm}^2$ , or  $\approx 20 \text{ mpa}$ <sup>16</sup>.

One can also calculate that modeling threshold as a fraction of an intact ligament's ultimate strength (US). Where (MESfm) signifies that threshold value:

$$MESfm = ULVL \div US \quad \text{Equ. (2b)}$$

For the ACL in Table 2,  $MESfm = 800 \div 1950 = 0.41$ , meaning that intact ligament's modeling threshold would  $\approx 41\%$  of its ultimate strength when both are expressed as stresses. For comparison, in similar terms a healthy bone's MESfm is currently thought to  $\approx 17\%$  of its ultimate strength<sup>16</sup>.

C) An ACL's strength-safety factor (SF). To calculate this factor, divide an ACL's ultimate strength (US) by its modeling threshold (MESm), each expressed as a material and a stress, or:

$$SF = US \div MESm \quad \text{Equ. (3a)}$$

For the ACL in Table 2,  $SF = 55.7 \div 22.8 \approx 2.4$ . Thus that ACL's ultimate strength as a tissue  $\approx 2.4$  times stronger than needed to carry its typical largest voluntary loads. For comparison, bone's strength-safety factor in similar terms is thought to lie in the 6 x region.

To calculate the strength-safety factor as a function of an intact ACL's ultimate strength, divide its ultimate strength (US) by the usual largest voluntary loads (ULVL) on it *in vivo*, or:

$$SF = US \div ULVL \quad \text{Equ. (3b)}$$

For the ACL in Table 2,  $SF = 1950 \div 800 \approx 2.4$ , so again this ACL would be  $\approx 2.4$  times stronger than needed to carry

Force
1 kg = 2.2 lb = 9.8 N. 1 N = .225 lb = .102 kg. 1 million N = 224,000 lb. 1 lb = .45 kg = 4.4 n.
Pressure
1 mpa = 1 million N/M <sup>2</sup> = 145 psi = 1 N/mm <sup>2</sup> = .102 kg/mm <sup>2</sup> . 1 kg/cm <sup>2</sup> = 14.2 psi. 1 kg/mm <sup>2</sup> = 9.8 mpa = 1,420 psi. 120 mpa = 17,400 psi = 12.2 kg/mm <sup>2</sup> = 120 N/mm <sup>2</sup> . 20 mpa = 2,900 psi = 2.04 kg/mm <sup>2</sup> .
Area
1 M <sup>2</sup> = 1,550 in <sup>2</sup> . 1 in <sup>2</sup> = 6.45 cm <sup>2</sup> = 645 mm <sup>2</sup> .
kg = kilogram. lb = English pound. N = Newtons of force. mpa = megapascals. psi = pounds per square inch. mm = millimeters. M = meters. cm = centimeters.

**Table 3.** Some English-metric Conversions.

the typical largest total loads put on it by voluntary activities.

D) The usual largest voluntary loads (ULVL) on an ACL. If accurate values for MESm and CSA became available from other sources, one could solve Equ. 2a for the ULVL on an intact ACL to estimate it in that way:

$$ULVL \approx MESm \times CSA \quad \text{Equ. (4)}$$

From Equ. 2a and for the ACL in Table 2,  $ULVL = 22.8 \times 35 \approx 798 \text{ N}$ ,  $\approx 80 \text{ kg}$ , or a bit less than 1.2 times the body weight of the man in Table 2.

#### IV. Comments

1) Studies incited by the problems of surgical ACL reconstructions helped to produce the evidence in Table 2. While it supports the idea that ACLs have a diametric modeling threshold, most readers would probably agree that it does not suffice to prove the idea is true. The kinds of evidence needed to prove that (and to add strain considerations to the equations in Section #4 of Part III above) could be discussed at another time and place. Yet and as an aside, by 1964 the existence of a modeling threshold for bone was inferred from analogous considerations<sup>18</sup>, and after 1970 others verified it and showed its approximate value<sup>6,8,13</sup>.

Parenthetically, it would be good engineering to make its loads somehow control the strength of a living structure intended to carry loads without breaking or rupturing. We now know bones do that and bone's modeling threshold is essential for doing it<sup>8,16</sup>. Like others, I believe ACLs do it too. If so a diametric modeling threshold should be equally essential for doing it.

*Question:* What tension *strain* might correspond to that diametric modeling threshold? To my knowledge this has not been studied yet, but I suspect it could lie in the 40,000 microstrain region (a 4% stretch). For comparison, bone's modeling threshold as a strain seems to lie in the 1000-1500 microstrain region (a 0.1% - 0.15% elongation or shortening)<sup>8</sup>. In young adults, bone's ultimate strength as a strain is a range centered near 25,000 microstrain (somewhat more during growth and less in aged adults)<sup>14</sup>. Note that the stress-strain curves for bone and ligament are not straight lines and are dissimilar<sup>32</sup>.

2) Immediately after an ACL reconstruction the infarcted graft would lack the living cells needed to provide strain-controlled diametric modeling. It should typically take three or more months to repopulate autogenous grafts with competent cells from the host, along with new blood vessels that invade it from the host-bed tissues. Before that repopulation occurred the ligament could not change its strength to adapt to the usual voluntary loads on it. In allografts and xenografts that could take much longer and might even fail. Such things could affect the long-term results of such reconstructions.

3) Like other studies, those cited in Section #3 of Part III

of this article show the ACL's anatomical features and mechanical properties can vary considerably. Its ultimate strength increases during growth, it seems to decline with aging, and it differs in small and large people. Its mean minimum cross section area of  $\approx 35 \text{ mm}^2$  in people aged 48-77 years might be larger in large young athletic adults; it is smaller in children. The amount of collagen per  $\text{mm}^2$  cross section of an ACL, as well as its cross linking, can vary, which would correspondingly affect its ultimate strength and stiffness. Estimates of the peak voluntary loads on the ACL *in vivo* varied from 400 N to over 800 N, but they were always below the ACL's ultimate strength. Differences in neuromuscular coordination and function in different individuals of the same size could also affect the voluntary ACL loads considerably. Finally, different parts of the ACL may carry its total load during different maneuvers of the knee<sup>33</sup>. If so a smaller cross section area than the intact ACL's minimum cross section area would carry most of its momentary total loads. That would mean its ultimate strength as a material and its strength-safety factor were larger than the values calculated in Section #4 of Part III.

Such variations should not affect the general algebraic *relationships* described in Part III, but if not compensated for they would certainly affect the *accuracy* of calculated values, including the examples in Part III. This recalls an old bit of wisdom that should not surprise anyone who studied the ACL: "Many devils can lie in the details".

4) Various things (aging, hormones, drugs, calcium, vitamin D, cytokines, chemokines, etc.) might modulate the ACL's diametric modeling threshold's set point (MESm) when it is defined as the center of the threshold's range. Presumably its existence and value reside as built-in genetically-encoded instructions in some collagenous tissue cells, so genetic factors could -- should -- also affect it.

Anything that raised that threshold should lead to a thinner and weaker ACL, because it would take larger strains than before to turn modeling on to strengthen it. Anything that lowered it should lead to a thicker and stronger ACL, because smaller strains than before could turn its modeling on. That suggests therapeutic agents that reduced this threshold might potentiate the healing of reconstructed ACLs, and might help to make stronger ones in uninjured athletes.

5) Clinical pathologic evidence suggests this diametric modeling in collagenous tissue organs normally works throughout life<sup>24</sup>. As one example, in children and middle-aged adults I have seen attenuated tendons of paralyzed muscles thicken over three times after transferring to them the tendons of other strong muscles to restore or improve hand, knee or ankle function<sup>16</sup>. That took two or so years, so this diametric modeling is slow (subsequent operations provided the opportunities to make those observations).

6) If as I believe the above general relationships or "game rules" extrapolate to other ligaments as well as to fascia and tendons<sup>15</sup>, that could explain why the strength of healthy ligaments in all mammals seems to fit the size of the typical

largest voluntary loads on them. It could explain why the strength of every healthy mammalian tendon from the mouse to the elephant fits the strength of its muscle (which could answer a question about that raised by Ker et al. in 1988<sup>34</sup>. Likewise for the strength and loads on every fascia. Anatomists, biomechanicians, orthopaedic surgeons and sports medicine and surgery clinicians could find such things of special interest.

So could specialists in extraskelatal medicine and surgery<sup>16</sup>. Why? Because collagenous tissues contribute in various ways to the anatomy and physiology of all extraskelatal organs. Ergo and as examples, diametric modeling disorders in their collagenous tissue component might help to cause some inguinal, ventral and hiatal hernias; some aneurysms and varices; some pulmonary fibrosis; some strictures of the esophagus, ureter, urethra and bile duct; some pericardial stenoses; keloid; and some features of Dupuytren's and Peyronie's diseases<sup>16</sup>.

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