Estimating the strength of bone using linear response

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Accurate diagnostic tools are required for effective management of osteoporosis; one method to identify additional diagnostics is to search for observable consequences of bone loss. An analysis of a model system is used to show that weakening of a bone is accompanied by a reduction of the fraction of the bone that participates in load transmission. On the basis of this observation, it is argued that the ratio Γ of linear responses of a network to dc and high-frequency ac driving can be used as a surrogate for their strength. Protocols needed to obtain Γ for bone samples are discussed.

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I. INTRODUCTION

Osteoporosis is a major socioeconomic problem in western societies [1]. Since excessive use of therapeutic agents often has adverse effects on patients [2], noninvasive diagnostic tools to determine their need are essential for effective management of the disease. Bone mineral density (BMD) is the principal such investigative tool [3]. Ultrasound scans [4] and structural characteristics of the inner porous region or trabecular architecture (TA) [5–7] are being studied as complementary diagnostics. These characteristics account only partially for the strength of a bone and its propensity for fracture. In this paper, results from the analysis of a model system are used to propose a different diagnostic for osteoporosis.

Porous segments are the principal load carriers in bone from older adults [4], and their structure is reminiscent of disordered cubic networks [8,9]. Most microfractures formed during routine activity are repaired by a turnover process, which prevents significant degradation of the quality and fracture toughness [10] of trabecular bone [4]. However, more traumatic events can sever trabecular elements causing a systematic loss of connectivity and strength of TA with aging.

In Ref. [9], a disordered cubic network of fragile elastic elements [11] with elastic and bond-bending forces was proposed as a system to model mechanical properties of TA. As suggested by experiments on bone samples [12,13], elastic elements are assumed to satisfy a strain-based fracture criterion; specifically their fracture strains are chosen from a Weibull distribution [14,15] (with parameters γ_e and n). Bond angles are assigned a similar fracture criterion; their fracture angles are chosen from a second Weibull distribution (with parameters γ_b and n). Bone loss is modeled by random removal of a fraction ν of elastic elements from the network; the remaining struts are assumed to retain their elastic moduli.

The diagnostic introduced below includes the response of a network to an ac strain, which depends on the mass distribution on the network; it is modeled by placing masses m on the vertices. The viscous effects of the surrounding medium are modeled by a dissipative force proportional to the speed of each mass. In the studies reported here, networks are subjected to uniform compression and points located on transverse boundaries are constrained to lie within these planes. Numerical studies of the system support the conjecture that these networks are a suitable model to study the mechanical properties of bone [9].

The use of a model system to identify possible diagnostic tools can be justified as follows. Most structural details of trabecular bone are patient and location dependent [9]. In contrast, for a diagnostic tool to be useful, it needs to be broad based; i.e., it should predict the bone strength from many subjects. In other words, *measures that are viable as diagnostic tools can only depend on a few generic aspects, but should transcend most details of the TA*. Model systems can be expected to be useful in determining such ''configuration independent'' aspects of trabecular bone.

II. BREAKDOWN OF NETWORKS AND STRESS BACKBONES

It is very difficult to provide theoretical estimates for breakdown properties of a network because they depend on the largest fractures present [16–18]. However, it has been argued that statistical analysis of breakdown properties can be carried out using methods similar to those used to study transport properties [18]. Analysis of our model shows that the (mean) fracture strains depend only weakly on the level of bone loss, ν. Consequently, the ultimate (or breaking) strength of such a network is proportional to its elastic modulus χ_0. Analogous observations have been made in mechanical studies; i.e., bone samples from a given anatomical site fail at a fixed level of stain *independent of age* [12], and their yield stresses are strongly correlated with their elastic moduli [19].

Stress transmissions in ''healthy'' and ''weak'' networks under uniform compression exhibit distinct characteristics, as illustrated in Fig. 1 [20]. For clarity only the compressed elements of each network are shown, and darker hues represent larger stresses. On the ''healthy'' network ($\nu=10\%$) large stresses supporting propagation are distributed evenly. In contrast, elastic elements supporting a ''weak'' network ($\nu=30\%$) form a few coherent pathways. The set of elastic elements active in load transmission will be referred to as the ''stress backbone'' of a network [21].

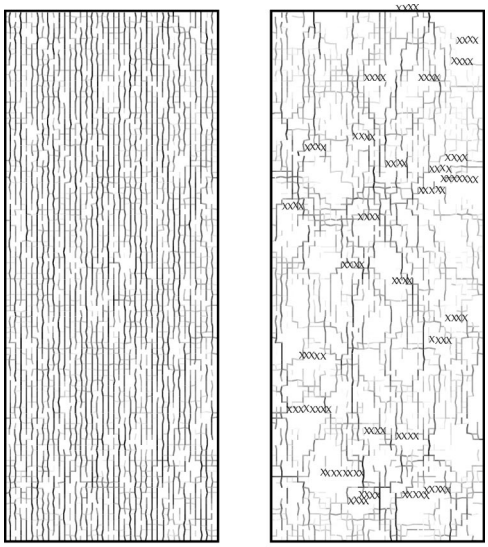

FIG. 1. The stress distributions on networks of size 40×100 with (a) $\nu=10\%$, and (b) $\nu=30\%$ representing "healthy" and "osteoporotic" bone, respectively. For clarity only the compressed bonds are shown, and darker hues represent larger stresses. The crosses denote locations of long horizontal fractures.

Reduction in the extent of the stress backbone is caused by the presence of long fractures on the network. In Fig. 1, the crosses denote locations where four or more consecutive vertical bonds are missing. It is clear that these fractures prevent the participation of many additional bonds in the stress backbone. Since the number of long fractures increases with ν, a progressively smaller fraction of elastic elements are able to be load carriers. We conjecture that the breaking strength of a network is related to the fraction of elastic elements belonging to the stress backbone. We show next that the total number of elements on a network and the number that belong to the stress backbone can be estimated from its linear response to external strain.

III. LINEAR RESPONSE

Consider first an ordered $N\times M$ square network of identical elastic elements, each of whose breaking stress, breaking strain, and elastic modulus are denoted by t_0, s_0, and $Y=t_0/s_0$. Then the ultimate stress and strain of the complete (i.e., $\nu=0$) network are $T(0)=Nt_0$ and $\zeta(0)=Ms_0$. Denote by $T(\nu)$ and $\zeta(\nu)$ their values when a fraction ν of randomly chosen struts are removed. The dc response (or elastic modulus) of the network can be written as

$$\chi_0=\frac{T(\nu)}{\zeta(\nu)}=\frac{Nt_0}{Ms_0}\frac{T(\nu)}{T(0)}\frac{\zeta(0)}{\zeta(\nu)}=\frac{NY}{M}\tau(\nu)\frac{\zeta(0)}{\zeta(\nu)}, \qquad (1)$$

where $\tau(\nu)=T(\nu)/T(0)$ is the nondimensionalized breaking stress of the network. But as was discussed earlier $\zeta(\nu)$ is only weakly dependent on ν. If we approximate $\zeta(\nu)$ by $\zeta(0)$, then

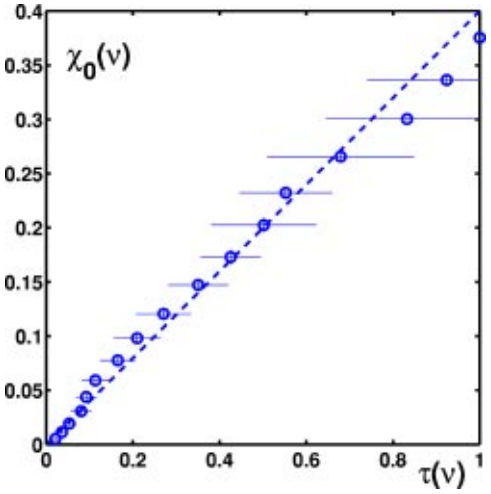

FIG. 2. The mean values and ranges of χ_0 and $\tau(\nu)$ for five nominally identical networks. As expected the variability in the breakdown property $\tau(\nu)$ is significantly larger than that in the transport coefficient χ_0.

$$\chi_0\approx\frac{NY}{M}\tau(\nu). \qquad (2)$$

Equation (2) relates a transport property χ_0 to a breakdown property τ of the network. As discussed in the last section, such a relationship can only be expected to hold on average. Figure 2 confirms the behavior given in Eq. (2) for a set of five nominally identical networks [20].

Next, we argue that the linear response to an external ac strain can be used to estimate the number of elastic elements that are present on a network. First subject the network to a dc compression (ξ_{dc}) below the yield point, so that there is no fracture of elastic elements. Next introduce an additional ac compression, given by $\xi(t)=\xi_{ac}\exp(i\Omega t)$, where $\xi_{ac}\ll\xi_{dc}$ [22]. When Ω increases so does the attenuation of the signal, and a progressively thinner slice of the network is affected by the ac signal. Figure 3 shows the response of the network of Fig. 1(b) to external ac signals at two frequencies.

Denote by $F(t)$ the sum of vertical forces on the top layer due to $\xi(t)$. The linear response of the network $\hat{\chi}(\Omega)$ is given by $\hat{F}(\Omega)=\hat{\chi}(\Omega)\hat{\xi}(\Omega)$, where $\hat{\xi}(\Omega)$ and $\hat{F}(\Omega)$ are the Fourier transforms of $\xi(t)$ and $F(t)$, respectively. When $\Omega\rightarrow0$, there is no attenuation and hence $\hat{\chi}(\Omega)$ approaches χ_0. On the other hand, for sufficiently large Ω only those struts belonging to the two edges are excited. In this case, each of the $(1-\nu)N$ bonds of the top layer is strained by a same amount and hence

$$\hat{\chi}(\Omega)\approx N(1-\nu)Y. \qquad (3)$$

Numerical integrations of disordered networks confirm this expression.

Consider once again the ordered pure network. When only the edges experience ac stress the effective height of the layer is reduced by a factor M; thus, $\hat{\chi}(\Omega)$ will be larger than χ_0 by a factor M. For $\nu=0$, this can be expected to hold (approximately) even for disordered networks since the

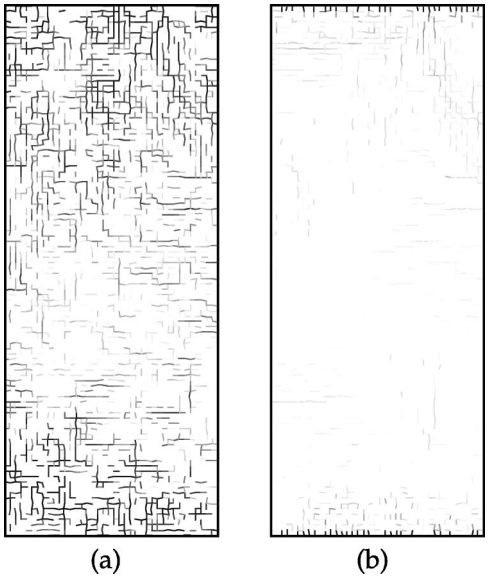

FIG. 3. The distribution of stresses on the disordered network of Fig. 1(b) due to small amplitude ac compressions with (a) $\Omega=10$ and (b) $\Omega=500$. As Ω increases, so does the attenuation of the signal, and a progressively thinner slice of the network is affected. For sufficiently large Ω only bonds belonging to the top and bottom layers experience an ac stress.

stress backbone covers the entire network. Even though both χ_0 and $\hat{\chi}(\Omega)$ will decrease with increasing ν, the latter will be affected less because long fractures in the middle of the network (which reduce the stress backbone) have no effect on $\hat{\chi}(\Omega)$. These arguments motivate the use of

$$\Gamma(\nu)\equiv \lim_{\Omega\to\infty} M\frac{\chi_0}{|\hat{\chi}(\Omega)|} \tag{4}$$

to estimate the fraction of elastic elements belonging to the stress backbone. Using Eqs. (2), (3), and (4) gives

$$\tau\approx(1-\nu)\Gamma. \tag{5}$$

If the function $\nu=\nu(\tau)$ is known, it can be inverted (e.g., the Born expansion) to obtain $\tau=\tau(\Gamma)$. The linear part of $\tau(\Gamma)$ is universal as can be seen from the following argument. Since the rigidity threshold for networks that include bond-bending forces is the same as the percolation threshold [23], $\tau(\nu_0)=0$ (where ν_0 is 0.5 for square networks [24]); i.e., $\nu_0=\nu(\tau=0)$. Consequently,

$$\tau\approx(1-\nu_0)\Gamma+h(\Gamma), \tag{6}$$

where $h(\Gamma)$ denotes nonlinear terms. In order to test this expression, numerical studies were conducted on a group of five equivalent disordered square networks of size 40×100. In evaluating the linear response, the signal $F(t)$ was collected after the transients settled. For a given ν, there is scatter in the values of both $\Gamma(\nu)$ and $\tau(\nu)$, and Fig. 4(a) shows the mean and range of each variable. The slope of $\tau(\Gamma)$ is seen to approach $(1-\nu_0)$ as $\nu\to\nu_0$.

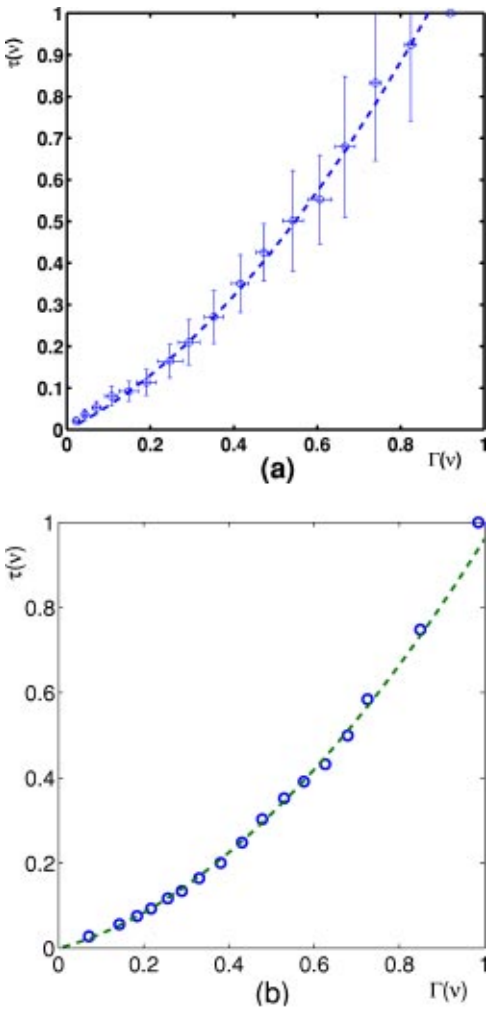

FIG. 4. (a) The relationship between Γ and τ for a set of five equivalent disordered networks. The dashed line shows the best fit of the data to Eq. (6) with $h(\Gamma)=c\Gamma^{\beta}$. For the parameters [20] considered, $c=0.90$ and $\beta=3.20$. (b) shows the analogous relationship for a disordered cubic network.

To proceed further, the expression $\nu=\nu(\tau)$ is required; several forms for it have been proposed in recent studies [25–27,17,28]. As an example, we can use the expression

$$\tau=\frac{1}{1+a_1z^{\alpha}+a_2z^{2\alpha}} \tag{7}$$

proposed in Ref. [28] where $z=\log(\mathcal{N})/\log(\nu_0/\nu)$ and $\mathcal{N}$ is the total number of nodes on the network. The constants a_1, a_2, and α depend on the type of network and model parameters such as the relative strengths of the response coefficients of the elastic and bond-bending terms. The form of Eq. (7) was deduced by an analysis of the effects of a single fracture, and has been tested for a range of values for ν away from the critical point [28]. (Observe that $\tau(0)=1$ and $\tau\to0$ as $\nu\to\nu_0$. However, the correct critical behavior will be obtained only if α is the critical index. This is typically not the case.)

Given the values of a_1, a_2, and α [29], Eq. (7) can be inverted to obtain $\nu(\tau)$. Substitution of this expression in Eq. (5), and inversion gives $\tau(\Gamma)$. Unfortunately, these inversions are difficult to implement. Furthermore, in analyzing bone samples, it is significantly easier to determine τ and Γ than to estimate ν. For results from the model system $h(\Gamma)$ can be approximated very well by a two-parameter function such as $c\Gamma^{\beta}$ or $(c_2\Gamma^2+c_3\Gamma^3)$. The dashed line in Fig. 4(a) shows the best fit using the first form.

A similar relationship can be obtained for disordered cubic networks. In the reasoning leading to Eqs. (2) and (3), N needs to be replaced by the number of nodes in the top layer. Thus the only modification in Eq. (6) is the value of ν_0 (which is 0.7508 for cubic networks [24]). As shown in Fig. 4(b), the strength of a disordered cubic network reduces approximately as given by Eq. (6) when its struts are removed randomly.

IV. DISCUSSION

The reduction τ of bone strength from its peak value can be used to determine the need for therapeutic intervention to reduce the propensity for fracture. Unfortunately, it is not accessible *in vivo*, and surrogates such as bone density are used to identify osteoporotic bone. However, there is significant variability between the bone strengths of samples with a fixed BMD; consequently, it is difficult to identify individuals susceptible to fracture using measurements of BMD alone [30]. Since breakdown properties depend on the availability of stress pathways, large variability between the strength and density of a bone is not unexpected.

The diagnostic introduced in this paper bears a strong correlation to the (mean) strength of model networks. Experimental studies are planned to determine if it can accurately predict the strength of bone samples. Measurements required to evaluate Γ can be implemented on *ex vivo* bone samples. dc strain can be imposed using pressure loading, and protocols using ultrasonic techniques have been developed to evaluate the response of bone samples to ac driving [31]. Previous studies suggest that when their frequencies are larger than $\sim$1.5 MHz ultrasonic signals will excite only those trabeculae on the outer edges of a TA [32]. Methods need to be developed to implement these measurements *in vivo*.

Several issues need to be reiterated. To calculate $\hat{\chi}(\Omega)$, only elastic forces (in nodes of the top layer) were used. In driving a bone sample with an ac strain, material (in the outer layer) is accelerated in a dissipative medium. These inertial and dissipative forces are proportional to Ω^2 and Ω, respectively. In contrast, for sufficiently large Ω, $\hat{\chi}(\Omega)$ is Ω independent (since only struts at the edges are excited). Hence, $\hat{\chi}(\Omega)$ can be evaluated using the response of the TA to ac signals over several frequencies. Secondly, although the linear part of $\tau(\Gamma)$ is universal its nonlinear terms depend on model parameters. Hence the form of $\tau(\Gamma)$ will have to be determined for distinct bone locations (e.g., femur, vertebrae) before a complete diagnostic tool for osteoporosis is developed. Finally, notice that the definition of Γ includes the number of layers M of the network, which can be estimated from the height of a sample since the lengths of trabeculae from specific anatomical locations are known (typically $\sim$1 mm).

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tively. The fracture criteria are given by $\gamma_e = 5\%$, $\gamma_b = 0.1$, and $n = 5$. Each mass was 10^{-5} units, and the dissipation coefficient $\eta = 10^{-5}$. The disorder is controlled by a single random seed, which is varied to produce additional ''equivalent'' networks. The equilibrium was evaluated using the conjugate gradient method.

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