Estimating the Strength of an Elastic Network Using Linear Response

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Disordered networks of fragile elastic elements have been proposed as a model for inner porous regions of large bones [Gunaratne et.al., cond-mat/0009221, http://xyz.lanl.gov]. In numerical studies, weakening of such networks is seen to be accompanied by reductions in the fraction of load carrying bonds. This observation is used to show that the ratio $\Gamma$ of linear responses of networks to DC and AC driving can be used as a surrogate for their strength. The possibility of using $\Gamma$ as a non-invasive diagnostic of osteoporotic bone is discussed.

Osteoporosis is a major socio-economic problem in an aging population [1]. Non-invasive diagnostic tools to determine the need for therapeutic intervention are essential for effective management of the disease. Bone Mineral Density (BMD), or the effective bone density is the principal such investigative tool [2]. Ultrasound transmission through bone [3] and geometrical characteristics of the inner porous region or trabecular architecture (TA) [4] are being studied as complementary diagnostics. In this Letter, we use results from a model system to suggest an additional diagnostic for osteoporosis.

The TA is the principal load carrier in bone of older adults [5], and cellular models have been used to study TAs [6]. In Ref. [6], a disordered network of fragile elastic elements [7] was proposed as a system to model mechanical properties of a TA. Preliminary studies are conducted on two dimensional square networks which include elastic and bond-bending forces. Motivated by conclusions from mechanical studies of bone [8], the springs are assumed to satisfy a strain-based fracture criterion; specifically the fracture strains of elastic elements are chosen from a Weibull distribution [9] (with parameters $\gamma_e$ and $m$). Bonds are assumed to fracture when changed beyond a critical angle; these fracture-angles are chosen from a second Weibull distribution (with parameters $\gamma_b$ and $m$). Osteoporosis is modeled by random removal of a fraction $\nu$ of springs from the network.

The characteristic introduced below includes response of a network to an AC strain, which depends on the mass distribution on the network. It is modeled by placing masses $m$ at the vertices. Viscous effects of the surrounding medium are modeled by a dissipative force proportional to the speed of each mass. In studies reported here, points located on the sides were constrained to move vertically. Numerical studies of the system support the conjecture that these elastic networks are a suitable model to study mechanical properties of bone [10].

Figure 1 shows the stress distributions on two networks subjected to uniform compression [10,11]. For clarity only the compressed bonds are shown, and darker hues represent larger stresses. On the “healthy” network (where $\nu = 10\%$) the large stresses supporting propagation are seen to be distributed evenly over the network. In contrast, elastic elements supporting a “weak” network (\(\nu = 30\%\)) form a few coherent pathways. We refer to the set active in load transmission as the “stress backbone” of a network [12].

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.

In Figure 1, the X’s denote long horizontal fractures; specifically, locations where four or more consecutive ver-
tical bonds are missing. It is clear that these fractures prevent the participation of many bonds in the stress backbone. Since the number of long fractures increases with $\nu$, a progressively smaller fraction of bonds are able to be load carriers. The assumption that this dilution of the stress backbone is related to the ultimate (or breaking) stress of the network motivates the measure introduced below.

Consider first an ordered $N \times M$ square network of identical springs, each of whose breaking stress, breaking strain and elastic modulus are denoted by $u_0$, $\zeta_0$ and $Y = u_0/\zeta_0$. Then the ultimate stress and strain of the pure (i.e., $\nu = 0$) network are $U(0) = Nu_0$ and $M\zeta_0$. Denote by $U(\nu)$ and $\zeta(\nu)$ their values for a network obtained by removing bonds with a probability $\nu$. Each horizontal layer of the network is approximately compressed by $\zeta(\nu)$ and a fracture will propagate when one of the bonds bordering it is stressed beyond $u_0$. In this approximation, the DC response of the network is

$$\chi_0 = \frac{U(\nu)}{\zeta(\nu)} \approx \frac{NY}{M} \cdot u(\nu), \quad (1)$$

where $u(\nu) = U(\nu)/U(0)$.

![Image](a) ![Image](b)

**FIG. 2.** The distribution of stresses on the disordered network of Figure 1(b) due to small amplitude AC compressions with (a) $\Omega = 10$ and (b) $\Omega = 500$. As $\Omega$ increases, so does the attenuation of the signal, and a progressively thinner slice of the network is effected. For sufficiently large $\Omega$ only bonds belonging to the top and bottom layers experience an AC stress.

Next we argue that the linear response to an external AC strain can be used to estimate the number of bonds in a network. First subject the network to a DC compression ($\zeta_{DC}$) below the yield point, so that there is no fracture of elastic elements. Next introduce an additional AC compression, given by $\zeta(t) = \zeta_{AC} \exp(i\Omega t)$, where $\zeta_{AC} \ll \zeta_{DC}$. When $\Omega$ increases so does the attenuation of the signal, and a progressively thinner slice of the network is effected by the AC signal. Figure 2 shows the AC response of the network of Fig. 1(b) driven at two frequencies.

Denote by $T(t)$ the sum of vertical forces on the top layer due to the AC strain. The linear response of the network $\hat{\chi}(\Omega)$ is given by $T(\Omega) = \hat{\chi}(\Omega) \cdot \zeta(\Omega)$, where $\zeta(\Omega)$ and $T(\Omega)$ are the Fourier transforms of $\zeta(t)$ and $T(t)$ respectively. Figure 3 shows the behavior of $|\hat{\chi}(\Omega)|$ for a disordered network with $\nu = 30\%$. When $\Omega \rightarrow 0$, there is no attenuation and hence $\hat{\chi}(\Omega)$ approaches $\chi_0$. On the other hand, for sufficiently large $\Omega$ only the two edges are excited and hence $\hat{\chi}(\Omega)$ can be expected to approach a limit. Then each of the $(1 - \nu)N$ bonds of the top layer are strained by the same amount and hence

$$\hat{\chi}(\Omega) \approx N(1 - \nu)\bar{Y}, \quad (2)$$

where $\bar{Y}$ denotes the mean elastic constant for bonds on the top layer. Numerical integrations of disordered networks confirm these conclusions, see Fig. 3.

![Graph](3)

**FIG. 3.** The dependence of $|\hat{\chi}(\Omega)|$ on $\Omega$ for a network with $\nu = 30\%$. For $\Omega \rightarrow 0$, $\hat{\chi}(\Omega) \rightarrow \chi_0$, while for sufficiently large $\Omega$, $\hat{\chi}(\Omega) \approx N(1 - \nu)\bar{Y}$. In this limit, only bonds bordering the top and bottom layers experience AC stress. The fluctuations for intermediate values of $\Omega$ are configuration dependent.

Consider once again the ordered pure network. When only the top and bottom layers experience AC stress the effective height of the layer is reduced by a factor $M$; thus, $\hat{\chi}(\Omega)$ will be larger than $\chi_0$ by a factor $M$. For $\nu = 0$, this can be expected to hold (approximately) even for disordered networks since the stress backbone
covers the entire network. Even though both $\chi_0$ and $\chi(\Omega)$ will decrease with increasing $\nu$, the latter will be affected less because long fractures in the middle of the network (which reduce the stress backbone) have no effect on $\chi(\Omega)$. These arguments motivate the use of
\[
\Gamma(\nu) \equiv \lim_{\nu \to \infty} M \frac{\chi_0}{|\chi(\Omega)|}
\] (3)
to estimate the reduction in the extent of the stress backbone. Approximating $Y$ by $Y$ and using Eqs. (1), (2) and (3) gives
\[
u \approx (1 - \nu) \cdot \Gamma(\nu).
\] (4)

To complete the derivation, we propose a relationship $\nu = F(u)$. Since $u(\nu)$ for a 2D square network vanishes at the percolation threshold $\nu = \nu_0 = \frac{1}{2}$,
\[
u_0 = F(0).
\] (5)

A form for $F(x)$ has been presented in Ref. [4], where it was assumed that the propagation of the longest horizontal fracture causes the collapse of a network. The ingredients used are (1) a horizontal fracture of size $k$ enhances the stress on the bordering elements by $(1 + ak\alpha)$, and (2) the size $km$ of the longest fracture satisfies $M \sim k_m^{m-1} (1 - \nu)^2$ [4]. Since the possible two dimensional-ity of a fracture is ignored, the resulting expression does not satisfy Eq. (5). We propose a modification
\[
\frac{1}{u(\nu)} - 1 \approx a \left( \ln M N \right)^{\alpha},
\] (6)
which agrees with results from numerical integration of disordered networks for $\nu \in (0, 0.4)$ [3, 4]. Parameters $a$ and $\alpha$ are expected to depend on factors such as the relative strengths of the elastic and bond-bending forces.

Using Eqs. (4) and (5),
\[
u(\nu) = (1 - \nu_0) \Gamma + h(\Gamma),
\] (7)
where the nonlinear correction $h(\Gamma)$ can be obtained by inverting (e.g., Born expansion) Eq. (6) and will depend on $a$, $\alpha$, and $\ln(MN)$.

To test the validity of Eq. (6), numerical studies were conducted on a group of five equivalent disordered networks [4]. $\Gamma(\nu)$ and $U(\nu)$ for a given network is evaluated using methods discussed earlier [1]. As can be expected, the normalization of $U(\nu)$ by $U(0)$ reduces the variability between distinct configurations in the group. In evaluating the linear response, the signal $T(t)$ was collected after the transients have settled. For a given $\nu$, there is scatter in the values of both $\Gamma(\nu)$ and $u(\nu)$, and Figure 4 shows their mean and standard errors. The dashed line is the best fit to the data in the form $\Gamma(\nu) = c \Gamma^{1/3}$. Numerical studies on configurations with different parameters show that, unlike the linear term of Eq. (6), the coefficients $\beta$ and $\epsilon$ are parameter dependent.

The reduction of bone strength from its peak value determines the level of osteoporosis. Unfortunately, it is not accessible in-vivo (without breaking a bone!), and surrogates such as bone density are used to identify osteoporotic bone. The BMD of a patient is compared with that of a sample population to determine if and when therapeutic interventions are necessary. However, the ultimate stress is known to depend on other factors of bone including the structure of its TA and the "quality" of bone material. The resulting variations make it difficult to identify individuals susceptible to fracture using measurements of BMD alone [7].

In this Letter, we have shown that linear response of a network can be used as a surrogate for its strength. This conjecture is based on the assumption that the strength is related to the extent of the stress backbone. Measurements required to evaluate $\Gamma$ 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 [13]. Previous studies suggest that when their frequencies larger than $\sim 1.5 MHz$, ultrasonic signals will excite only those trabeculae on the outer edges of a TA [19]. How these measurements can be implemented in-vivo remains to be studied.
Several issues need to be reiterated. To calculate $\chi(\Omega)$, only elastic forces (on vertices of the top layer) were used. In driving a bone sample with an AC strain, the matter (on the outer layer) is accelerated in a dissipative medium. These inertial and dissipative forces are proportional to $\Omega^2$ and $\Omega$ respectively. In contrast, for sufficiently large $\Omega$, $\chi(\Omega)$ is $\Omega$-independent (see Fig. 3). Hence, the latter can be extracted from the response of the TA to AC signals of several frequencies. Secondly, observe that for weak networks ($\nu$ close to $\nu_0$) the relationship between $u(\nu)$ and $\Gamma(\nu)$ depends only on $\nu_0$. For smaller values of $\nu$, however, the nonlinear correction $h(\Gamma)$, which depends on model parameters, becomes relevant. Hence the form of $u(\Gamma)$ may 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 $\Gamma$ includes the number of layers $M$ of the network. Since the lengths of trabeculae from specific anatomical locations are known (typically $\sim 1$mm), the length of the sample can be used to determine $M$.

The author would like to thank S. R. Nagel for pointing out that the response of a network is related to its stress backbone. He also acknowledges discussions with M. P. Marder, G. F. Reiter and S. J. Wimalawansa. This research is partially funded by the National Science Foundation, the Office of Naval Research and the Texas Higher Education Coordinating Board.

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