Strain-rate Dependent Stiffness of Articular Cartilage in Unconfined Compression

The stiffness of articular cartilage is a nonlinear function of the strain amplitude and strain rate as well as the loading history, as a consequence of the flow of interstitial water and the stiffening of the collagen fibril network. This paper presents a full investigation of the interplay between the fluid kinetics and fibril stiffening of unconfined cartilage disks by analyzing over 200 cases with diverse material properties. The lower and upper elastic limits of the stress (under a given strain) are uniquely established by the instantaneous and equilibrium stiffness (obtained numerically for finite deformations and analytically for small deformations). These limits could be used to determine safe loading protocols in order that the stress in each solid constituent remains within its own elastic limit. For a given compressive strain applied at a low rate, the loading is close to the lower limit and is mostly borne directly by the solid constituents (with little contribution from the fluid). In contrast, however, in case of faster compression, the extra loading is predominantly transported to the fibrillar matrix via rising fluid pressure with little increase of stress in the nonfibrillar matrix. The fibrillar matrix absorbs the loading increment by self-stiffening: the quicker the loading the faster the fibril stiffening until the upper elastic loading limit is reached. This self-protective mechanism prevents cartilage from damage since the fibrils are strong in tension. The present work demonstrates the ability of the fibril reinforced poroelastic models to describe the strain rate dependent behavior of articular cartilage in unconfined compression using a mechanism of fibril stiffening mainly induced by the fluid flow. [DOI: 10.1115/1.1560142]

Keywords: Cartilage Mechanics, Fibril Reinforcement, Finite Element Analysis, Nonlinear Biomechanics, Poroelasticity

1 Introduction

Articular cartilage (of the knee joint, for example) is typically subjected to physiological loading involving large strains at high rates. At sufficiently large compressive strains (e.g., 20%), the cartilage stiffness at high-speed loadings can reach 10–15 times as high as that for static loadings. It is essential to understand how the cartilage behavior changes with the strain amplitude and strain rate as well as the implications of such changes in cartilage functioning. Much experimental work has been done in observing the consequences of impact loadings, including mechanical failure, changes in biochemical composition and cartilage pathology due to the resulting strains [1–8]. It has been found that the strain (stress) rates as well as the strain (stress) amplitudes are important factors influencing the normal function of cartilage [9–12]. However, previous model studies, which consider the fibrils and proteoglycans as one constituent, have experienced difficulties in predicting the relevant mechanical behavior especially that of the nonlinear transient response. For example, the widely used isotropic biphasic model predicts a maximum transient stiffness of unconfined cartilage disk at only 1.5 times of the equilibrium stiffness [13]. Obviously such models are not adequate to study cartilage response to high rate loadings. This is probably why extensive analyses have not yet been reported in correlating the transient stiffness of cartilage to the strain amplitude and strain rate, and hence in determining the individual role of each of the three constituents in the transient response.

The fibril reinforced poroelastic models [14–16] recently developed distinguish the role of the collagen fibrillar network from that of the proteoglycan matrix and thus have the potential to model the interactions among three material constituents: collagen fibrils, proteoglycan matrix and water containing diffusible ions. One of the advantages of such modeling is the ability of the models to predict the nonlinear transient response in unconfined compression. Previous work on these models has been focused on formulation and verification of the method. It has been shown analytically that the fibril reinforcement plays a predominant role in the transient behavior of cartilage in unconfined compression. The objectives of the present work are to explore the mechanism of the transient response, especially at high strain rates not previously considered, and to determine the strain amplitude/rate dependent stiffness of cartilage in unconfined compression by employing the fibril reinforced models. The work is likely helpful in describing the experimental findings, such as cartilage damage is loading rate dependent [10], that may not be explainable if the solid skeleton of cartilage is formulated as one single constituent. It is hypothesized that the collagen fibrils play an important role in supporting high rate loadings whereas the proteoglycan matrix is relatively more crucial in supporting low rate (or equilibrium) loadings.

2 Methods

In an experimental study [17], 3 mm diameter bovine articular cartilage (0.9–1.2 mm thick) disks with underlying bone (0.3–0.6 mm thick) were compressed in unconfined geometry with compression amplitudes ranging from 12.5 to 300 μm and speeds from 0.5 to 50 μm/s. The compressive stiffness of the disks during the transient was found to vary non-monotonically with the compression amplitude and to increase substantially with the compression speed. In a preliminary model study, the cartilage and bone were both assumed as homogeneous materials [18]. In another
attempt to simulate the experimental data, a nonhomogeneous model involving depth-varying material properties [15] was employed in the present work to reduce the extent of material discontinuity at the cartilage/bone interface, in an effort to improve the numerical results. In the current simulation, the disk radius was taken to be 1.5 mm and the thickness of cartilage and bone were taken to be 1.1 and 0.4 mm respectively (which were representative dimensions for the explants used in the tests [17]). Both bone and cartilage were considered as poroelastic. The bone was defined by the Young’s modulus \( E_m \), Poisson’s ratio \( \nu \) and a constant permeability \( k \). The cartilage skeleton was considered to consist of a fibrillar matrix (collagenous) and a nonfibrillar matrix (mainly proteoglycans). The fibrillar matrix was presented by the Young’s modulus \( E_f \left( = \omega (E_f^0 \times \text{fibril strain} + E_f^0) \right) \), where \( \omega \) was a function of cartilage depth [15, \( \omega = 1 \) for homogeneous model]. The nonfibrillar matrix was defined by \( E_m \), \( \nu_m \) and \( k \) \( = k \exp(M \times \text{Dilatation}) \). Linear depth variations were assumed for \( E_m \) and \( \nu_m \), as well as the initial void ratio [15]. The parameters used for simulation (given in the caption of Fig. 1) were chosen to be compatible with measurements reported and our previous modeling work. The finite element mesh (axisymmetric) for continuum elements was 14×7 (7 in the axial direction: 5 for the cartilage layer, 2 for the bone).

Further analyses focused on exploring the behavior of cartilage without the presence of the bone. Thus only the cartilage layer was considered in finite element analysis. A homogeneous model [14] was then employed to reduce computation time. This simplification would not produce qualitative changes in the results, since only the overall response of the disks was of interest in this study (determined by the total load and displacement amplitudes). The depth dependence of cartilage properties was previously found to have little influence on the load pattern when the bone was not attached [15]. The radius and thickness of the cartilage disks were taken to be 1.5 and 1.0 mm respectively. Only half the thickness of the disk was meshed (axisymmetric 14×4). In each case, five compression rates were investigated: the static response (zero compression speed), 0.05%, 0.5% and 5% compressive axial strain per second and the instantaneous response (infinite compression speed). The compression was applied continuously at one of the axial strain rates from the undeformed state to 20% compressive nominal strain. This loading protocol was different from that of the experiments [17] where a sequence of compression-relaxation-release ramps of desired strain amplitudes was imposed, accompanied by interspersed witness ramps to monitor possible changes in cartilage properties during the loading cycles. Such repeated loadings were not necessary in computation since the material properties were assumed to be unaffected by the loading. It is to be noted that the strain amplitudes and strain rates adopted in the purely theoretical analyses were higher than those used in the experimental data fitting.

Various cases with diverse material properties were investigated (Table 1, concerns will be addressed in the discussion). Considering the five compression speeds, 224 cases in total were involved, noting that the static and instantaneous responses are independent of \( M \). The fibril’s nonlinear property (\( E_f > 0 \)), the permeability nonlinearity (\( M > 0 \)) and the effect of finite deformation were considered except when specifically noted otherwise.

The static (equilibrium) and instantaneous responses are fluid-flow independent. Accordingly, the equilibrium stiffness was obtained by employing elastic continuum elements, or by assuming the disks were drained. The instantaneous stiffness, on the other hand, was obtained when the fluid was assumed to be trapped, i.e. all fluid diffusion boundaries were sealed and geometrical boundary conditions were applied in no time. Alternatively, analytical solutions for both static and instantaneous compressions are feasible. The small deformation solutions are provided in Appendix A.

![Fig. 1 Axial compressive stiffness of cartilage disks with bone attached, the nonhomogeneous model versus experiments (mean±SD, \( n = 4 \)). The material parameters of cartilage used for simulation are as follows: at the articular surface, \( E_m = 0.20 \) MPa, \( \nu_m = 0.12 \), the void ratio is 3.6; at the cartilage and bone interface \( E_m = 0.80 \) MPa, \( \nu_m = 0.42 \), the void ratio is 2.88; \( k = 0.004 \text{ mm}^4/\text{Ns} \) and \( M = 13 \); \( E_f = 2800 \) MPa and \( E_f^0 = 3 \) MPa at the surface. For the underlying bone, \( E = 18 \) MPa, \( \nu = 0.25 \), \( k = 0.0004 \text{ mm}^2/\text{Ns} \), the void ratio is 0.5.](image_url)

Table 1 The material parameters for various cases computed. For all cases, the void ratio is 3.5 and the initial permeability is \( k = 0.003 \text{ mm}^3/\text{Ns} (k = k \exp(M \times \text{Dilatation})) \). \( E_m \) and \( \nu_m \) are, respectively, the Young’s modulus and Poisson’s ratio of the nonfibrillar matrix; \( E_f \) is the Young’s modulus of the fibrillar matrix. The dimension for \( E_f \), \( E_m \) and the aggregate modulus \( H \), is MPa. (Each of the seven groups of \( E_m \) and \( \nu_m \), together with other parameters, constitutes a case for computation.)

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**Transactions of the ASME**
3 Results

The axial compressive stiffness of the cartilage disks and the radial strain (logarithmic) at the center are presented versus the compressive nominal axial strain. For convenience, the nominal modulus, defined as the ratio of the total nominal axial stress over nominal axial strain, is generally adopted to represent the stiffness ~exceptions in Fig. 9! During the transient, the stresses are not uniform in the radial direction and the nominal axial stress is calculated using the total load and the initial cross-sectional area of the disk.

The model captures the major trends of the experimental data ~Fig. 1!. The simulation involves modeling the underlying bone, whose material properties are assumed to be homogeneous and independent of strains. It is seen that the predicted growth of the transient stiffness slows down at larger strains for the compression rate 50 m m/s ~Fig. 1!. Should the bone be assumed to stiffen with the strain ~while a smaller E ] f be used!, the model results would match the experimental data better. This is indicated by a faster growth in Fig. 3a ~5%/s! where only cartilage is considered. Moreover, the underlying bone is actually nonhomogeneous: between cartilage and subchondral bone lies a layer of calcified cartilage ~3–8% cartilage thickness [19]! that has a transitional modulus [20]. Pure subchondral bone has been reported to have very high transient stiffness at the order of 1–4 GPa ~e.g. [20]!. However, static measurements by ultrasound obtained a stiffness of 19.8 MPa for normal subchondral bone from human femoral head [21]. Obviously a great deal of work is required to determine the bone and cartilage interactions. For this reason, we only considered cartilage in the remaining part of this paper ~Figs. 2–9!.

We first consider the cases involving full nonlinearity ~Figs. 2–4!. The equilibrium stiffness is not strongly dependent on either the axial strain or the fibrillar modulus. In the present unconfined compressions ~E m and m as shown!, it is mainly determined by the aggregate modulus H A ~i.e. the confined stiffness!, with less dependence on E m or m varying individually ~Fig. 2a!. The instantaneous stiffness is much higher and grows very quickly with the axial strain ~Fig. 2b!. Obviously the elastic properties of the drained nonfibrillar matrix ~E m and m! have very little influence on it. The instantaneous stiffness is predominantly affected by the fibrillar modulus E f, and its growth is determined by the fibril stiffening ~represented by E f! ~Fig. 2b!. The transient stiffness is largely strain-rate and load-history dependent ~Fig. 3!. When the
The transient stiffness initially increases for a short time, then decreases and again increases with the axial strain. Altering the elastic properties of the nonfibrillar matrix has relatively larger impact on the transient stiffness at the lower rates (~0.05% and 0.5%/s). In contrast, the fibril stiffening with its tensile strain has little impact on the disk stiffness when compression rate is as low as 0.05%/s.

The radial strain (logarithmic) is shown against the axial nominal strain (Fig. 4). The logarithmic axial strain is slightly larger than the nominal one, e.g., when the nominal is 10% and 20%, the logarithmic strain is, respectively, 10.5% and 22.3%). The radial strain at equilibrium is very small compared with the axial strain. At the beginning of compression, the radial strain during transient is close to (but less than) half the axial strain, representing a very small rate of volume change. The radial strain deviates from half the axial strain when the compression continues. The higher the compression strain rate, the closer the radial strain to half the axial strain and the larger the axial strain till which this trend lasts. For the high compression rate 5%/s, the bulk volume changes little at the center of disk until around 8% axial strain. This is observed when the two curves for different $E_f$ are nearly superimposed (Fig. 4b), since the radial strain is independent of material properties when the volume is conserved. On the other hand, when $E_f$ is reduced from 1600 to 1200, the transient radial strain (Fig. 4b) increases less significantly than does the equilibrium strain (Fig. 4a), but it produces larger differences in the stiffness at high strain rates (Fig. 3b vs Fig. 2a).

Now consider the cases for which the nonlinearity in material properties partially disappears, possibly due to cartilage pathology for example (Figs. 5–7). If the permeability is constant while the fibril nonlinearity remains unchanged (Fig. 5), the transient stiffness increases with the axial strain, monotonically for the case with compression rate of 5%/s. In all these cases, the stiffness at high strains is substantially reduced due to loss of nonlinear properties.
When $E_f$ is reduced by 25% (not shown), there is no change in the stiffness for the strain rate 0.05%/s and almost no change for the strain rate 0.5%/s after 10% axial strain. Again $E_m$ (or $E_m$) has a more significant role in the stiffness when the strain rate is low (Fig. 5).

The impact of nonlinear permeability is demonstrated by the radial strain. The permeability nonlinearity makes no differences in the radial strain at low axial strains, but large differences at high axial strains (Fig. 6). Furthermore, the higher the compression rate, the larger the axial strain at which the permeability nonlinearity starts to make a difference (Fig. 6). On the other hand, among the three compression rates, the largest differences in the radial strain produced by the permeability nonlinearity occur at 0.5%/s (Fig. 6). This is so because when the strain rate is too high or too low, the cartilage behavior is mainly elastic; the permeability plays the most significant role in the stiffness at a moderate strain rate. These trends due to the finite deformation and small deformation (SD) theories are employed respectively. In all cases, the stiffness extracted from the SD theory is not axial strain dependent (horizontal lines). (a) Instantaneous moduli as obtained by employing the finite deformation (FD) and small deformation (SD) theories. (b) Transient moduli for the compression rate 0.5%/s ($M=10$, FD theory only).

Fig. 9 Significance of different measures in quantifying cartilage stiffness, for $E_m=0.36$ MPa, $E_m=0.38$, $E_f=1600$ and $E_f=3$ MPa. The tangent modulus is defined as $\frac{d\sigma_z}{d\varepsilon_z}$ and the secant modulus is defined as $\sigma_z/\varepsilon_z$, where $\sigma_z$ is the Cauchy stress in the axial direction and $\varepsilon_z$ is the logarithmic strain. The nominal modulus, generally adopted elsewhere in this paper, is the ratio of nominal axial stress over nominal axial strain. For the convenience of comparison, the moduli are shown as functions of the nominal axial strain. (a) Instantaneous moduli as obtained by employing the finite deformation (FD) and small deformation (SD) theories. (b) Transient moduli for the compression rate 0.5%/s ($M=10$, FD theory only).

Fig. 7 Transient stiffness of cartilage with constant fibrillar modulus ($E_f=20$ MPa) for $M=10$ vs $M=0$ while $E_m=0.36$ and $E_m=0.38$. No significant qualitative changes in the stiffness are observed for a larger $E_m$ and thus the results for the comparison group are not included.

Fig. 8 Axial compressive stiffness of cartilage for constant $E_f=20$ MPa and $E_m=0.38$ when no fluid flow is feasible. The finite deformation and small deformation (SD) theories are employed respectively. In all cases, the stiffness extracted from the SD theory is not axial strain dependent (horizontal lines). (a) Elastic static stiffness for $E_m=0.36$, 1.07 and 2.14 MPa respectively. (b) Instantaneous stiffness for which the fluid is trapped.
to alteration in the permeability patterns remain when \( E_i \) is reduced (the fine dots in Fig. 6 vs the data symbols in Fig. 4b).

When \( E_i \) is constant, a weak disk stiffening is possible only if the compression rate is high (5%/s, Fig. 7). Otherwise the transient stiffness decreases significantly with the axial strain at low and moderate strains, and increases slightly at high strains for \( M = 10 \) (nonlinear permeability); the stiffness decreases monotonically if \( M = 0 \) (Fig. 7) except for the case with \( E_m = 2.14 \) and compression rate 0.05%/s for which a very slight increase at high strains is found (not shown).

The significance of employing the finite deformation theory is shown for the cases with linear material properties (Fig. 8). The equilibrium and instantaneous stiffness extracted by the small deformation theory are given by equations (A-11) and (A-4) respectively. Employing the small deformation theory results in smaller errors in the equilibrium stiffness.

The results would be somewhat different if the measure for the stiffness defined in the first paragraph of this section were altered. For the example demonstrated (Fig. 9, nonlinear material properties), the stiffness represented by the solid curves has been shown earlier (Figs. 2b and 3). The moduli are defined as in the caption of the figure, where the Cauchy stress for the transient is obtained by the total compression load and the real cross-sectional area. The instantaneous moduli determined by the small deformation theory (Fig. 9a) are calculated by equations (A-4) and (A-5) respectively; they are linear functions of the axial strain. When the axial strain is not so large (e.g. <8%), the finite deformation theory yields nearly the same results (Fig. 9a). The secant modulus can be replaced by the nominal modulus when the axial strain is small for both transient and instantaneous responses. The tangent modulus for the transient case is found quite different, which was obtained by differentiating the numerical results of the stress and strain with 800 strain intervals (8000 time steps were employed in the finite element computation. However, the results for 9 out of 10 steps were not saved due to large output by ABAQUS).

4 Discussion

It has been shown that the fluid flow dominates the transient response of cartilage. In general, for the fully nonlinear case, there is an initial rise in the stiffness due to tissue resistance to immediate fluid exodus, witnessed by little volume change, i.e. the radial strain is nearly half the axial strain at the beginning of compression (Fig. 4b). This trend of volume conservation will be lost sooner or later depending on the strain rate. Then the disk stiffening diminishes significantly (Figs. 3b and 4b), or even the disk appears softer if the compression rate is sufficiently low since the fluid dissipates relatively quickly with respect to the rate of matrix deformation. However, as the compression strain further increases (at the low compression rate), the permeability is substantially reduced and the flow becomes more difficult, expanding the disk quicker and resulting in fibril stiffening and a rise in pore pressure. This explains the decrease in transient stiffness followed by an increase with respect to the axial strain (Fig. 3, 0.05%/s). If, on the other hand, the collagen fibrils stiffen quickly with the flow-dependent lateral expansion, the transient stiffness hence grows quickly with the strain if the compression rate is high so that the fluid has little time to dissipate, approaching the instantaneous modulus (if within the elastic limit). Thus the actual pattern of the transient stiffness versus the axial strain depends on the combination of the fluid dissipation speed and the rate of fibril stiffening. The stiffness increases monotonically with the axial strain only if the compression rate is sufficiently high (Fig. 3a, 5%/s). For the same reason, the transient response is load-history dependent and therefore, the transient stiffness and strain would be altered should the compression protocol be changed.

The interplay between fluid kinetics and fibril stiffening can be further illustrated by the cases of reduced material nonlinearity. When the permeability is constant (\( M = 0 \)) with other conditions remaining unchanged, the transient stiffness still increases quickly and monotonically at high compression rates (Fig. 5, 5%/s). However, even if the permeability varies with the matrix dilatation (\( M = 10 \)), but the fibrils do not stiffen with its strain (\( E_i \approx 20 \) MPa, a typical value for medium strains), the transient modulus does not increase much with the strain even for the relatively high compression rate (Fig. 7, 5%/s). Moreover it has been found that a higher constant \( E_i \) (50 MPa) does not result in a greater increase (not shown). This indicates the overwhelming ability of fibrillar matrix stiffening to produce an increasing transient stiffness at higher axial strains. The actual role of the fibrillar matrix is however determined by fluid dissipation that in turn is affected by compression rates and amplitudes (in addition to the matrix elasticity and permeability), as well as loading history. The current study does not consider the fibril viscoelasticity that might play a role in the strain-rate dependence of cartilage unconfined stiffness.

The instantaneous stiffness (which is the maximum elastic stiffness) is certainly of interest in clinical studies. It is unique and determines the upper elastic limit of loadings at a given strain (The lower limit corresponds to the equilibrium stiffness). The instantaneous stress of the nonfibrillar matrix is dependent only on the shear modulus of the matrix when the deformation is small (A-1). This could indicate the failure type of cartilage should the nonfibrillar matrix be overloaded. Incidentally, under an impact loading cartilage disks were found to crack at a plane approximately 45° to the articular surface [3]. The flow independent responses derived in the appendix for small deformations can be obtained analytically for large deformations too. Then finite element computations are not required if only the upper and lower limits are desired. Similar studies have been done previously for poroelastic beams, columns and plates with linear material properties. In those cases, if the deflection is small, the solutions for the instantaneous responses can be obtained by the corresponding equilibrium solutions after the Young’s modulus of the drained material is multiplied by a dimensionless constant [22].

The material parameters used as reference in the present work were originally based on fitting the data from multi-step ramp compression tests, for which the nominal axial strain was not greater than 10% and the strain rates were around 0.1%/s [14,15]. We had to adopt smaller \( M \) and \( E_i \) in order to fit the transient responses for different compression rates when the axial strain was as large as 20% [18]. This is because they are not so sensitive in the case of small deformations and low strain rates. The fibrillar modulus \( E_i \), currently taken as a linear function of the strain \( e_i \), might not be accurate enough for rather large deformations. Should \( E_i (e_i) \) be a convex curve [while \( \sigma_i (e_i) \) is concave], a smaller \( E_i \) for the case of large deformation and a larger \( E_i \) for the case of small deformation must be taken in order to improve fittings when \( E_i \) is approximately replaced by a secant. Further work is required to test this hypothesis. Probably a hyperelastic constitutive law should be employed when the axial strain is close to 20%. However, we do not expect much change in the general patterns of the transient stiffness when the representation of fibrillar stiffness is revised.

Alternative material parameters were also considered to examine their influence on cartilage stiffness. In some cases, \( E_m \) (2.14 MPa) might be slightly too large to represent articular cartilage. However, the purpose to use large \( E_m \) was to demonstrate its insignificance in the transient stiffness when the compression rate is high. For the same reason, a large range of \( \nu_m \) was considered. Another concern is whether the relevant stress is within the strength of the bulk material (water saturated). The answer would be positive at least for all the cases with limited compression rates, for which the maximum stress is less than 8 MPa (Fig. 3a). The maximum instantaneous stress is 14.5 MPa (derived from Fig. 2b). More evidence is required to know if cartilage fails before reaching this stress, but the instantaneous stiffness presented should be justified for a slightly smaller strain (e.g. 18% axial). Kerin et al. [4] found that the disks failed at a nominal stress

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between 14 and 59 MPa, while Repo and Finlay [6] reported the failure at 25 MPa. On the other hand, the current work has not considered intrinsic property changes due to repeated loadings. While such changes do occur, they are not so significant within a load (or stress) threshold [8,17].

The present study has explored the mechanism of the strain rate/amplitude dependent cartilage response to external loadings and demonstrated the distinct mechanical functions of the three major constituents of cartilage. An impact load is primarily balanced by the fluid pressure restrained by the fibrillar matrix; it is essential that the fibrils be strong in tension and provide extra stiffness whenever a fibril strain increment is produced as a result of rising fluid pressure. The fibrillar stress is then greatly relaxed by water flow, facilitating the physiological activities within cartilage. The stress of the nonfibrillar matrix is relatively invariable under a given strain, a mechanism protecting the matrix from damage, as long as the collagen fibrils have not yet reached their own limit. On the other hand, if the fibrils fail to function properly due to collagen degradation, the cartilage will be easily destroyed by an even small impact load. In fact, the importance of the tensile strength of the fibrils in keeping cartilage integrity has been observed experimentally [23]. For normal cartilage, the current model suggests that mechanical damage would not occur as long as the loading path (strain-varying) falls within the upper limit, regardless of the loading rate and history.

Acknowledgments

The first author received the industrial research fellowships from the NSERC-Canada.

Appendix A: The Instantaneous and Static Responses of Cartilage With Small Deformation

First, consider the instantaneous response of the disk, when the load is trapped. Since all constituents of the material are incompressible, no volume change is possible. Thus the stress increments are

\[
\Delta \sigma_r = 2 \mu_m \Delta e_r - \Delta p_f
\]

\[
\Delta \sigma_f = (2 \mu_m + E_f) \Delta e_f - \Delta p_f
\]

where \( \mu_m \) is the Lamé constant, i.e. the shear modulus of the nonfibrillar matrix. The other Lamé constant is absent due to zero volume increment. The stress increment for the fibrillar matrix is represented by \( E_f \Delta e_f \), for which \( E_f = E_f^0 + E_f^0 \varepsilon_f \). It has been assumed that the axial fibrils are not stressed in compression.

The radial stress increment is required to be zero. Thus the pore pressure increment is determined as

\[
\Delta p_f = (2 \mu_m + E_f) \Delta e_f
\]

Noting that the zero volume increment condition requires \( \Delta e_r = -\Delta e_f / 2 \), substituting (A-2) into the first equation of (A-1) yields

\[
\Delta \sigma_r = \frac{3 \mu_m + \frac{1}{2} E_f}{E_f^0} \Delta e_f
\]

The radial strain cannot generally be expressed as a simple function of the axial strain. However, if there was no previous loading before the current instantaneous compression, we have \( e_f = -e_r / 2 \). Therefore the effective modulus (\( \Delta \sigma_r / \Delta e_r \)) for instantaneous response is presented as axial strain dependent

\[
E_{inst} = \frac{E_f}{2(1 + \nu_m)} + \frac{1}{2} E_f^0 - \frac{1}{4} E_f^0 \varepsilon_r
\]

In this case, the axial stress is extracted by \( \int E_{inst} \Delta e \)

\[
\sigma_r = \left[ \frac{3 E_f}{2(1 + \nu_m)} + \frac{1}{2} E_f^0 - \frac{1}{4} E_f^0 \varepsilon_r \right] \varepsilon_r
\]

Similarly integrating (A-2) results in pore pressure

\[
p_f = -\left[ \frac{E_m}{2(1 + \nu_m)} + \frac{1}{2} E_f^0 - \frac{1}{8} E_f^0 \varepsilon_r \right] \varepsilon_r
\]

It is observed that in the radial direction the pore pressure described by the first term of (A-6) is balanced by the nonfibrillar matrix stress and the difference is balanced by the fibrillar matrix stress.

Now consider the static response when the pore pressure is nil. For the nonfibrillar matrix,

\[
\Delta \sigma_r = \frac{1}{E_m} \left[ \Delta \sigma_r - 2 \nu_m \Delta \sigma_r^m \right]
\]

\[
\Delta \sigma_f = \frac{1}{E_f} \left[ (1 - \nu_m) \Delta \sigma_f^m - \nu_m \Delta \sigma_r \right]
\]

Here the matrix stress increments \( \Delta \sigma_r^m \) and \( \Delta \sigma_f^m \) are identical due to uniform stresses at equilibrium. The first equation of (A-7) gives

\[
\Delta \sigma_r^m = \frac{1}{2 \nu_m} (\Delta \sigma_r - \nu_m \Delta \sigma_r^m)
\]

The total stress increment in the radial direction must be zero

\[
\frac{1}{2 \nu_m} (\Delta \sigma_r - \nu_m \Delta \sigma_r^m) + \Delta \sigma_r^m = 0
\]

where \( \nu_m \) is Poisson’s ratio of the nonfibrillar matrix. Hence, the effective modulus \( E_m \) and Poisson's ratio \( \nu_m \) are

\[
E_m = \frac{E_m \left[ E_m + E_f \left( 1 - \nu_m \right) \right]}{E_m + E_f \left( 1 - \nu_m \right)} \nu_m = \frac{E_m \nu_m}{E_m + E_f \left( 1 - \nu_m \right)}
\]

The initial strains are not considered in the present study, i.e. \( e_f = 0 \) when \( e_r = 0 \). Then by using \( \varepsilon_f = \varepsilon_r / 2 \), the axial strain is expressed as a second order polynomial of the radial strain

\[
\varepsilon_f = -\frac{1}{E_m \nu_m} \left( E_m + E_f \varepsilon_f \right)^{-1} \frac{E_m + E_f \varepsilon_f}{E_m + E_f} \varepsilon_f
\]

The positive root of the above algebraic equation gives the radial strain if \( E_f > 0 \)

\[
\varepsilon_f = -\frac{1}{E_m \nu_m} \left( E_f \varepsilon_f \right) \left( \frac{E_m + E_f \varepsilon_f}{E_f} \right) \varepsilon_f
\]

Then the axial stress at equilibrium can be obtained by integrating (A-9)

\[
\sigma_f = E_f \varepsilon_f - \nu_m (\Delta \sigma_f^m + E_f \varepsilon_f) \varepsilon_f
\]

References


