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A statistical damage model for bone tissue based on distinct compressive and tensile cracks

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Abstract

Osteoporosis leads to bone fragility and represents a major health problem in our aging societies. Bone is a quasi-brittle hierarchical composite that exhibits damage with distinct crack morphologies in compression and tension when overloaded. A recent study reported the complex damage response of bovine compact bone under four different cyclic overloading experiments combining compression and tension. The aim of the present work is to propose a mechanistic model by which cracking bone accumulates residual strain and reduces elastic modulus in distinct compressive and tensile overloading modes. A simple rheological unit of bone with two types of cracks is formulated in the framework of continuum damage mechanics. A statistics of these rheological units is then assembled in parallel to compute the response of a macroscopic bone sample in which compressive and tensile cracks are opened, closed or propagated towards failure. The resulting constitutive model reproduces the key macroscopic features of bone tissue damage and delivers an excellent agreement with the four cyclic overloading experiments. The remarkable predictions of the model

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support the presence of 1) friction between the crack surfaces producing hystereses, 2) an incomplete closure of cracks leading to residual strains, 3) a bridging mechanism of collagen fibrils which failure reduces elastic modulus, and 4) two distinct classes of cracks where compressive cracks have a strong influence on tensile damage and tensile cracks have a limited impact on compressive damage. This work is expected to help improve our understanding of the bone damage mechanisms contributing to skeletal fragility and to foster a proper generalization of this damage behavior in 3D for computational analysis of bone and bone-implant systems.

Number of words: abstract=268 and article=3388 (without equations, references and captions)

Keywords: Bone, Constitutive Model, Damage, Micro-cracks, Residual Strain, Rheological Model, Strength

1 Introduction

In our aging societies, the growing incidence of osteoporotic bone fractures motivates a refined exploration not only of bone mechanical properties as a function of age and disease, but also in bone loading during physiological activities or accidental situation such as falls.

Bone is a hierarchical, heterogeneous and anisotropic composite that exhibits a quasi-brittle damage mode at the macro-scale that consists of accumulation of residual strains and a reduction in elastic modulus due to micro-cracks (Fondrk et al., 1999; Taylor et al., 2007; Garcia et al., 2009). Despite organizational differences at the bone structural unit level, a similar damage behavior is

11 observed in trabecular and compact bone tissue, which points to the same un-
12 derlying micro-cracking mechanism of the extracellular matrix (Keaveny et al.,
13 1994; Fazzalari et al., 1998; Lambers et al., 2013). At the nanoscale, deforma-
14 tion of bone is determined by mineralized collagen fibrils interacting through
15 a thin interfibrillar glue layer (Fratzl and Weinkamer, 2007), which represents
16 an ideal interface for initiation of residual shear strains, progressive sliding of
17 mineralized fibril bundles, formation of diffuse damage (Zioupos et al., 1994;
18 Poundarik et al., 2012), and coalescing into micro-cracks (Frost, 1960; Lee et al.,
19 2003).

20 Micro-damage is partly repaired by self-healing mechanisms (Seref-Ferlengez et al.,
21 2014) and the cell-orchestrated remodeling process (Burr et al., 1985), but is be-
22 lieved to be at least partially responsible for the reduced toughness of bone with
23 age (Zimmermann et al., 2011). From a biomechanical perspective, increasing
24 attention is therefore devoted to elucidate the role of micro-damage on the var-
25 ious post-yield properties of bone tissue.

26 Rate-independent rheological models to describe the reduction of elastic mod-
27 ulus and accumulation of residual strains of trabecular bone were proposed
28 and even generalized to 3D in the framework of continuum damage mechanics
29 (Zysset and Curnier, 1996; Garcia et al., 2009). Two distinct dissipation pro-
30 cesses are responsible for the friction between crack surfaces producing residual
31 strains and the growth of cracks reducing elastic modulus. Rate-dependent
32 constitutive models were also proposed for compact bone (Garcia et al., 2010;
33 Fondrk et al., 1999), but remain to a large extent phenomenological and were
34 not able to describe the interaction between compressive and tensile damage.

35 In a pioneering work based on a parallel arrangement of linear elastic spring
36 elements undergoing brittle failure beyond a given ultimate strain, Krajcinovic
37 et al. (1987) described successfully the reduction of elastic modulus as well

38 as stress softening of cortical bone in tension. This model was motivated by
39 the parallel arrangement of osteons in the bone microstructure and the overall
40 compact bone strength emerged from a uniform statistics of element strengths.
41 Although in a compressive loading mode, two recent experimental studies on dry
42 micro-samples could confirm that the strength of the bone extracellular matrix
43 is substantially higher than the strength of macroscopic osteonal bone samples
44 (Schwiedrzik et al., 2014; Luczynski et al., 2015). Moreover, Schwiedrzik et al.
45 (1987) reported large residual strains and no damage in their micro-samples.
46 Applying the statistical distribution idea of Krajcinovic et al. (1987) to elasto-
47 plastic rheological elements, they could not only predict the observed scale effect
48 in longitudinal bone strength but also the apparition of damage and stress soft-
49 ening at the macro-scale.

50 Motivated by the different loading modes of osteoporotic bones in physiologi-
51 cal versus traumatic conditions, a recent experimental study investigated the
52 impact of damage accumulated in tension and compression on the other defor-
53 mation mode in bovine osteonal bone (Mirzaali et al., 2015). Histological ex-
54 amination confirmed that tensile damage consists mostly of diffuse cracks that
55 were oriented perpendicular to the osteonal loading axis, while compression re-
56 sulted in more contrasted cross-hatched cracks that were oriented at 45° with
57 respect to the osteonal axis (Reilly and Currey, 1999). After tensile damage,
58 compressive loading closes the perpendicular cracks (Sun et al., 2010) and their
59 influence on the subsequent elastic and post-yield behavior in compression was
60 found to be limited. In contrast, the shear damage accumulated under uniaxial
61 compression had a significant influence on the elastic and post-yield behavior in
62 tension. This interaction between compressive and tensile loading cannot be ex-
63 plained by the statistical model of (Schwiedrzik et al., 2014) devoted exclusively
64 to compression.

65 Accordingly, the aim of the present work was to generalize our previous rheo-
66 logical model to include the damage behavior produced under tension that is
67 able to describe qualitatively and quantitatively the asymmetric coupling effects
68 observed in the cyclic tests of Mirzaali et al. (2015). The paper is divided in
69 sections describing the proposed rheological unit, the assembly of a statistics of
70 unit models, the identification of the material constants combined with the re-
71 sults and a discussion. The actual numerical algorithm to compute the model's
72 response is provided in the appendix. The study of such constitutive models is
73 expected to contribute to the understanding of the damage mechanisms under-
74 lying bone fragility and to inspire the development of improved 3D constitutive
75 models for FE analysis of bone and bone-implant systems.

76 **2. Rheological model**

77 In this section, a single unit model is developed that will then be assembled in
78 section 3 into a statistics of units with specific distributions of material proper-
79 ties. The single unit model (Fig. 1) consists of a linear elastic spring representing
80 the intact bone extracellular matrix in series with two crack elements represent-
81 ing each a distinct compression and tension crack that can open and close but
82 not beyond a given closure strain illustrated by a stop. Each crack is modeled
83 by a rate-independent slider in parallel with a spring representing the collagen
84 fibrils bridging the crack. The tension and compression sliders have high fric-
85 tional stress thresholds in the opening mode and much lower thresholds in the
86 closing mode. The collagen springs fail beyond a given ultimate strain. Beyond
87 this ultimate strain, the frictional behavior of the sliders vanishes as well and,
88 due to its perpendicular configuration, only the stop of the closing tensile crack
89 remains effective. The series arrangement of the model leads to an additive

90 decomposition of the elastic and crack strains:

$$E = E^e + E^c + E^t \quad (1)$$

91 where the compressive crack strain E^c and the tensile crack strain E^t are inde-
92 pendent internal variables. The same crack strains E^c and E^t are relevant for
93 stretching of the bridging collagen fibrils. The series arrangement implies also
94 that the total stress is identical in the matrix and in the crack.

95 Bone tissue is made of bundles of mineralized collagen fibrils arranged in series
96 and parallel and can therefore be represented by a statistics of such rheological
97 models. The post-yield behavior in compression and tension is then driven by
98 two families of cracks that generate residual strains, reduce elastic modulus by
99 failing of the bridging collagen fibrils and dissipate energy in both processes.

100 In the standard generalized materials framework, a free energy ψ provides the
101 stresses associated with the reversible springs and a dual dissipation potential ϕ^*
102 delivers the evolution or flow rules of the internal variables, namely the residual
103 strains and the damage variables responsible for failure of the bridging collagen
104 fibrils and vanishing of the frictional forces. These two functions and the stresses
105 are presented in the next subsections followed by the resulting flow rules.

106 *2.1. Free energy*

107 The free energy of the model represents the sum of the recoverable energy stored
108 in the extracellular matrix and in the collagen fibers bridging the two cracks:

$$\psi = \psi^e(E, E^c, E^t) + \psi^c(E^c, D^c) + \psi^t(E^t, D^t) \quad (2)$$

109 The free energy of the intact matrix is

$$\psi^e(E, E^c, E^t) = \frac{1}{2}\epsilon(E - E^c - E^t)^2 \quad (3)$$

110 where ϵ is Young's modulus.

111 The free energy related to the bridging of collagen fibrils in the compression
112 crack is

$$\psi^c(E^c, D^c) = \begin{cases} \frac{1}{2}(1 - D^c)\chi^c E^{c2} + I_{]-\infty, 0]}(E^c) & \text{if } D^c < 1 \\ 0 & \text{if } D^c = 1 \end{cases} \quad (4)$$

113 where χ^c is the hardening modulus and $D^c \in [0, 1]$ is a damage variable that re-
114 duces the modulus of the spring (Lemaitre and Chaboche, 1990). The indicatrix
115 function is defined by

$$I_{\{.\}}(X) = \begin{cases} 0 & \text{if } X \in \{.\} \\ +\infty & \text{otherwise} \end{cases}$$

116 The latter function ensures that strain of the compression crack remains positive
117 until failure ($D^c < 1$). However, as shown in Fig. 1, this restriction vanishes at
118 failure and the strain of the compression crack can become positive when $D^c = 1$.

119

120 The free energy related to the the bridging of collagen fibrils (ligaments) in the
121 tensile crack is

$$\psi^t(E^t, D^t) = \frac{1}{2}(1 - D^t)\chi^t E^{t2} + I_{[E^{t, clo}, +\infty[}(E^t) \quad (5)$$

where χ^t is the hardening modulus, $D^t \in [0, 1]$ is a damage variable that breaks
the tensile spring and $E^{t, clo} \geq 0$ is a positive closure strain of the tensile crack

that grows linearly with maximal crack opening:

$$E^{t, clo}(t) = \gamma \text{Max}_{\tau \in [0, t]} \{E^t(\tau)\} \geq 0 \quad (6)$$

122 where $\gamma > 0$ is the proportionality factor.

123 Unlike sliding of compression cracks, closing of tensile cracks remains limited
 124 by $E^{t, clo}$ when $D^t = 1$. This is justified by the perpendicular configuration of
 125 tensile cracks that close and lock in compression even when the collagen spring
 126 is failed and no friction can occur any more.

127 The total stress S is given by the derivative of the free energy with respect to
 128 the total strain

$$S = \frac{\partial \psi}{\partial E} = \epsilon(E - E^c - E^t) \quad (7)$$

129 The frictional stress S^c in the compression crack slider also derives from the free
 130 energy with respect to the compressive crack strain,

$$S^c \in -\partial_{E^c} \psi \quad (8)$$

$$\in \begin{cases} \left\{ \begin{array}{ll} \{\epsilon(E - E^c - E^t) - (1 - D^c)\chi^c E^c\} & \text{if } E^c < 0 \\ [\epsilon(E - E^t), +\infty[& \text{if } E^c = 0 \quad \text{if } D^c < 1 \\ \emptyset & \text{if } E^c > 0 \end{array} \right. \\ \left\{ \{0\} \right. & \text{if } D^c = 1 \end{cases}$$

131 where $\partial_x \psi$ is the sub-differential of ψ with respect to the variable x that gen-
 132 eralizes the derivative to non-differentiable (C^0) functions (Rockafellar, 1970).

133 The symbol \emptyset is the empty set.

134 The second frictional stress S^t of the tensile slider derives from the free energy

135 with respect to the tensile crack strain,

$$S^t \in \begin{cases} -\partial_{E^t} \psi & \\ \emptyset & \text{if } E^t < E^{t, clo} \\] -\infty, \epsilon(E - E^c - E^{t, clo})] & \text{if } E^t = E^{t, clo} \\ \{\epsilon(E - E^c - E^t) - (1 - D^t)\chi^t E^t\} & \text{if } E^t > E^{t, clo} \end{cases} \quad (9)$$

136 Similarly, the conjugate energies to the two damage variables are obtained from
137 derivation of the free energy:

$$W^c = -\frac{\partial \psi}{\partial D^c} = \frac{1}{2} \chi^c E^{c2} \quad (10)$$

$$W^t = -\frac{\partial \psi}{\partial D^t} = \frac{1}{2} \chi^t E^{t2} \quad (11)$$

138 2.2. Dual dissipation potential

139 In the framework of standard generalized materials, the flow rules of the in-
140 ternal variables, here the residual strains and damage variables, derive from a
141 dual dissipation potential ϕ^* depending on the conjugate variables. For a rate-
142 independent process, the dissipation potential is the indicatrix of the convex
143 set representing the flow criterion (Germain, 1973). Due to the independence
144 of the internal variables and their conjugates, the dissipation potential can be
145 expressed as the sum of independent contributions:

$$\phi^* = \phi^{*, E^c}(S^c; E^c, D^c) + \phi^{*, E^t}(S^t; E^t) + \phi^{*, D^c}(W^c) + \phi^{*, D^t}(W^t)$$

146 where the variables listed after the semi-colon are considered as parameters.

147 The compression part responsible for crack opening and closing is

$$\begin{aligned} \phi^{*,E^c} &= \\ &= \begin{cases} \begin{cases} I_{[-\sigma^{c-}, +\sigma^{c+}]}(S^c) & \text{if } E^c < 0 \\ I_{[-\sigma^{c-}, +\infty]}(S^c) & \text{if } E^c = 0 \quad \text{if } D^c < 1 \\ \emptyset & \text{if } E^c > 0 \end{cases} \\ \{0\} & \quad \text{if } D^c = 1 \end{cases} \end{aligned} \quad (12)$$

148 where σ^{c-} and σ^{c+} are the absolute values of the stress thresholds for opening

149 and closing of the compression crack. The tensile part is

$$\phi^{*,E^t} = \begin{cases} \emptyset & \text{if } E^t < E^{t,clo} \\ I_{]-\infty, +(1-D^t)\sigma^{t+}]}(S^t) & \text{if } E^t = E^{t,clo} \\ I_{[-(1-D^t)\sigma^{t-}, +(1-D^t)\sigma^{t+}]}(S^t) & \text{if } E^t > E^{t,clo} \end{cases} \quad (13)$$

150 where σ^{t-} and σ^{t+} are the stress thresholds for closing and opening of the tensile

151 cracks. The compressive and tensile crack failure parts are

$$\phi^{*,D^c} = I_{[0, W^{c,ult}]}(W^c) \quad (14)$$

$$\phi^{*,D^t} = I_{[0, W^{t,ult}]}(W^t) \quad (15)$$

153 The flow rule resulting for the compression crack strain is

$$\dot{E}^c \in \partial_{S^c} \phi^{*, E^c} = \begin{cases} \left\{ \begin{array}{ll} \emptyset & \text{if } S^c < -\sigma^{c-} \\] -\infty, 0] & \text{if } S^c = -\sigma^{c-} \\ \{0\} & \text{if } S^c \in] -\sigma^{c-}, +\sigma^{c+}[\quad \text{if } E^c < 0 \\ [0, +\infty[& \text{if } S^c = +\sigma^{c+} \\ \emptyset & \text{if } S^c > +\sigma^{c+} \end{array} \right. & (16) \\ \left\{ \begin{array}{ll} \emptyset & \text{if } S^c < -\sigma^{c-} \\] -\infty, 0] & \text{if } S^c = -\sigma^{c-} \\ \{0\} & \text{if } S^c \in] -\sigma^{c-}, +\infty[\end{array} \right. & \text{if } E^c = 0 \end{cases}$$

154 For the strain variable related to the tensile crack

$$\dot{E}^t \in \partial_{S^t} \phi^{*, E^t} = \begin{cases} \left\{ \begin{array}{ll} \emptyset & \text{if } S^t < -(1-D^t)\sigma^{t-} \\] -\infty, 0] & \text{if } S^t = -(1-D^t)\sigma^{t-} \\ \{0\} & \text{if } S^t \in] -(1-D^t)\sigma^{t-}; (1-D^t)\sigma^{t+}[\quad \text{if } E^t > E^{t, clo} \\ [0, +\infty[& \text{if } S^t = (1-D^t)\sigma^{t+} \\ \emptyset & \text{if } S^t > (1-D^t)\sigma^{t+} \end{array} \right. \\ \left\{ \begin{array}{ll} \{0\} & \text{if } S^t \in] -\infty; (1-D^t)\sigma^{t+}[\\ [0, +\infty[& \text{if } S^t = (1-D^t)\sigma^{t+} \\ \emptyset & \text{if } S^t > (1-D^t)\sigma^{t+} \end{array} \right. & \text{if } E^t = E^{t, clo} \end{cases} \quad (17)$$

155 Similarly, the flow rule for the corresponding damage variables become

$$\dot{D}^c \in \partial_{W^c} \phi^{*,D^c} = \begin{cases} \emptyset & \text{if } W^c < 0 \\ \{0\} & \text{if } W^c \in [0; W^{c,ult}[\\ [0, +\infty[& \text{if } W^c = W^{c,ult} \\ \emptyset & \text{if } W^c > W^{c,ult} \end{cases} \quad (18)$$

156 and

$$\dot{D}^t \in \partial_{W^t} \phi^{*,D^t} = \begin{cases} \emptyset & \text{if } W^t < 0 \\ 0 & \text{if } W^t \in [0; W^{t,ult}[\\ [0, +\infty[& \text{if } W^t = W^{t,ult} \\ \emptyset & \text{if } W^t > W^{t,ult} \end{cases} \quad (19)$$

157 Since the damage criteria are constant values of an elastic energy function W
 158 that increases monotonically with strain, the failure of the crack elements be-
 159 comes fully brittle. The definition of the conjugate energies implies that their
 160 ultimate values correspond to ultimate crack strains:

$$W^{c,ult} = \frac{1}{2} \chi^c (E^{c,ult})^2 \Rightarrow E^{c,ult} = -\sqrt{\frac{2W^{c,ult}}{\chi^c}} \quad (20)$$

$$W^{t,ult} = \frac{1}{2} \chi^t (E^{t,ult})^2 \Rightarrow E^{t,ult} = \sqrt{\frac{2W^{t,ult}}{\chi^t}}. \quad (21)$$

161 The latter flow rules determine entirely the mechanical behavior of the rheo-
 162 logical model and the numerical implementation of these flow rules is presented
 163 in the appendix. The power dissipated by the model is the sum of the specific
 164 dissipative processes

$$\Phi = S^c \dot{E}^c + S^t \dot{E}^t + W^c \dot{D}^c + W^t \dot{D}^t. \quad (22)$$

165 The first two terms relate to friction associated with residual strains in compres-
166 sion and tension, while the two last terms are related to failure of the collagen
167 springs and the associated annealing of the frictional resistance.

168 *2.4. Response of the single element model*

169 The response of the developed single element model under cyclic loading is il-
170 lustrated in Fig. 2. The dissipative loops in compression and tension correspond
171 to opening and closing of the respective compression and tensile cracks. Failure
172 of the tensile crack ($D^t = 1$) still allows for compressive stresses, but failure
173 of the compression crack ($D^c = 1$) brings the total stress of the model to zero
174 everywhere. An animation of the rheological model's response is provided in
175 the supplementary material.

176 **3. Statistics of elements**

177 The behavior of bone tissue is then represented by a statistics of rheological
178 elements arranged in parallel. Following our previous work on micro-pillar
179 compression (Schwiedrzik et al., 2014), distinct statistical distributions were
180 assigned to the conservative and dissipative material properties. The elastic
181 properties of the extracellular matrix and the bridging springs follow a normal
182 distribution with a relative standard deviation of 8%, while the yield stresses
183 and ultimate crack strains follow a uniform distribution up to a maximal value
184 (Fig. 3). Given the strain formulation adopted in the above theory, computing
185 the response of the model requires only total stress for a strain based experi-
186 ment, but requires also the total tangent operator necessary in the numerical
187 implementation for a stress driven experiment.

188 Due to the parallel configuration of the rheological elements, the converged

189 stresses and the tangent operators are simply added:

$$S_{i+1}^{tot} = \sum_{e=1}^{nele} S_{i+1}^e \quad (23)$$

$$\frac{dS_{i+1}^{tot}}{dE_{i+1}} = \sum_{e=1}^{nele} \frac{dS_{i+1}^e}{dE_{i+1}} \quad (24)$$

190 where $nele$ is the number of elements.

191 4. Identification of material constants and results

192 The material constants were determined from the different experimental curves
193 for bovine bone obtained by Mirzaali et al. (Table ??). The monotonic ex-
194 periments delivered directly the crack opening stresses, hardening slopes and
195 ultimate strains in compression and tension, while a trial and error process with
196 the cyclic experiments provided the closing stresses as well as the residual clo-
197 sure strain in tension. The number of elements was set to 240 as a compromise
198 between smoothness of the response and computing time. A convergence anal-
199 ysis up to 1920 elements for monotonic tension and compression was conducted
200 that confirmed that beyond 240 elements, the mean stress errors over the entire
201 stress-strain curves remained below 1% (Fig. 4). The response of the model to
202 the monotonic experiments in compression and tension is displayed in Fig. 4.
203 As expected, the opening stress amplitude and the ultimate strain of tensile
204 cracks is significantly lower than the ones of compression cracks, which leads to
205 the well known difference in tensile and compressive ultimate strength of bone.
206 In compression, progressive failure of the elements lead to a slight reduction
207 of stress (stress softening) beyond a maximal value. In tension, the post-yield
208 response is almost constant up to 1.5% strain due to the higher hardening slope
209 of the bridging spring.

210 The loading schedule, stress response, numerical and experimental stress-strain
211 curves of a cyclic experiment in compression is shown in Fig. 5. The statistical
212 model captures the key features of the compressive damage process, namely the
213 residual strains, the reduction in stiffness due to failure of the bridging collagen
214 fibrils and the hysteresis produced by opening and closing of the cracks. Clearly,
215 the damage accumulated in compression becomes visible in the response to the
216 interrogation cycles in tension where stiffness is progressively reduced.

217 The same illustration is shown in Fig. 6 for a cyclic experiment in tension. Again,
218 the residual strains, stiffness reduction and hysteresis are properly reproduced
219 by the numerical simulation. In contrast to the previous cyclic experiment, the
220 response to the interrogation cycles in compression do not reveal the presence of
221 any reduction in compressive stiffness. A small shift of the tensile crack closure
222 strains $E^{t, clo}$ improves the qualitative correspondence with the experimental
223 curves.

224 The loading schedule, stress response, numerical and experimental stress-strain
225 curves of a further cyclic experiment in compression followed by a monotonic
226 loading in tension is shown in Fig. 7. Beyond the proper account of the dam-
227 age process in compression, the model predicts the induced reduction in tensile
228 strength.

229 The same illustration is shown in Fig. 8 for the cyclic experiment in tension
230 followed by a monotonic loading in compression. The post-yield behavior in
231 compression remains essentially unchanged after the overloading in tension.

232 Finally, the obtained material constants fulfill the equations 1 (Appendix), and
233 suggest that closure of tensile cracks require proportionally more stress than
234 closure of compression cracks. Animations of the cyclic load cases are provided
235 in the supplementary material.

236 5. Discussion

237 The aim of this work was to formulate, program and evaluate a novel consti-
238 tutive model to describe the influence of compressive and tensile crack families
239 on the cyclic response of bone tissue at the macroscopic level. A minimal set of
240 10 material constants was determined with six types of experimental curves in
241 monotonic compression, tension and multiple combinations of loading cycles.

242 In monotonic compression and tension, the distinct opening stresses and hard-
243 ening slopes are sufficient to reproduce the experimental stress-strain curves
244 both qualitatively and quantitatively. Interestingly, the post-yield material con-
245 stants obtained here by fitting the macroscopic monotonic compression curves
246 for bovine compact bone ($\sigma^{c-} = 456$ MPa and $E^{c,ult} = 0.07$) are in the same
247 range as the ones found for micro-pillar compression experiments for a single
248 crack in ovine bone ($\sigma^{c-} = 521$ MPa and $E^{c,ult} = 0.08$) (Schwiedrzik et al.,
249 2014).

250 In cyclic compressive overload, the model predicts also the macroscopic residual
251 strain, the reduction in elastic modulus and the growing hysteresis in a repeated
252 loading/unloading cycle (Fig. 5). The tensile damage is clearly visible with the
253 reduction in elastic modulus and the ultimate strength in tension is strongly
254 degraded (Fig. 7).

255 In cyclic tensile overload, the model predicts the macroscopic residual strain,
256 the reduced modulus and the progressing hysteresis (Fig. 6). In the absence of
257 substantial compressive loading, compressive damage remains low as only very
258 few elements yield and fail in compression. The ultimate strength in compression
259 remains essentially unaffected (Fig. 8).

260 The remarkable correspondence of the experimental and simulation stress-strain
261 curves (Fig. 5 - Fig. 8) suggests that the hypotheses of the model are consistent

262 with the actual bone damage process at the micro-scale.

263 First, friction between the crack surfaces leads to hystereses in the cyclic stress-
264 strain diagrams and represents an important dissipative mechanism that tends
265 to increase with growing damage as more cracks are open. The distinct level of
266 friction in the opening and closing modes may well be the rheological manifesta-
267 tion of the sacrificial molecular bonds observed in AFM studies (Fantner et al.,
268 2005). Second, the residual strains observed at the macroscopic level in the ab-
269 sence of load emerge from the incomplete closure of the cracks in both compres-
270 sion and tension mode. Moreover, the tensile crack strain do not return to zero
271 even after a compression cycle indicating that potentially some debris pushed
272 between the crack surfaces may prevent the tensile cracks to close completely.

273 Third, propagation of a crack through the ECM unit and the subsequent failure
274 of the bridging collagen fibrils is responsible for the reduced elastic modulus ob-
275 served in cyclic experiments and recovers the original idea of (Krajcinovic et al.,
276 1987). Fourth, compression and tensile cracks have indeed a distinct mechani-
277 cal behavior related to their orientation in the bone microstructure with respect
278 to the loading direction. The yield stress is three times higher in compression
279 than in tension, which is partially compensated by an order of magnitude higher
280 hardening slope in tension to achieve a macroscopic ultimate stress that is 85%
281 higher in compression than in tension. The higher hardening slope induces also
282 a more monotonic post-yield curve in tension (Fig. 4). The different nature of
283 the stop in compression and tension cracks explains not only the reduction of
284 the modulus in tension after accumulation of damage in compression, but also
285 the insensitivity of the modulus in compression after damage accumulated in
286 tension.

287 Despite the successful qualitative and quantitative predictions of the uniaxial
288 bone response for rather complex loading sequences, the model has some limi-

289 tations. First, the response of the bridging collagen fibrils was assumed to be
290 linear and neither viscoelasticity, nor viscous damage was included. In principle,
291 linear or non-linear dampers could be added in the ECM and in parallel to each
292 crack, but a proper identification would require a large number of experiments
293 performed at different strain rates on similar samples. More importantly, this
294 1D model is limited to uniaxial loading along the osteonal axis and was not
295 identified along other grain directions that are likely to deliver substantially dif-
296 ferent material constants since toughness was shown to be substantially lower
297 for cracks along the osteonal axis (Peterlik et al., 2006). Also, no shear load-
298 ing was investigated. Finally, the presented model is based on a continuum
299 approach of damage and cannot describe the propagation of the failing crack
300 treated in fracture mechanics.

301 The initial motivation for this model was to assess the potential weakening of
302 the proximal femur in a side fall configuration due to damage accumulation in
303 a physiological loading mode associated with normal activities. Femoral bone
304 tissue loaded in tension or compression during a fall may indeed be loaded in
305 the opposite mode during gait. What we learn from our findings is that the
306 compressive damage accumulated in the medial cortex during stance will in-
307 deed be detrimental to the overloading in tension caused by a fall on the side
308 (Nawathe et al., 2014). In contrast, the tensile damage that may accumulate in
309 the supero-lateral cortex would hardly affect the mechanical resistance against
310 overloading in compression due to a fall. This qualitative deduction calls obvi-
311 ously for further research as the amount and distribution of micro-damage in
312 the human osteoporotic proximal femur is widely unknown.

313 Nevertheless, the main benefits of such models are their relative simplicity and
314 their capacity to explain macroscopic behavior from a statistics of mechanical
315 prototypes (Bazant, 2004).The proposed model including crack opening and

316 closing is well-suited to extend quasi-static damage to fatigue damage and this
317 will be the object of future developments. Full generalization of this model
318 to 3D including anisotropy may prove to become difficult, but may be of high
319 interest for realistic computational analysis of bones and bone-implant systems
320 subjected to cyclic loading. In the light of the identified cracking mechanisms,
321 it becomes clear that the widely used elasto-plastic models are not appropriate
322 for bone as soon as unloading histories are involved. Finally, the understanding
323 of damage accumulation history in a human skeleton's life and its repair by
324 self-healing and bone remodeling remains a major challenge in contemporary
325 bone research.

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330 ical tests.

331 **Conflicts of interest statement**

332 The authors have no conflicts of interest to disclose.

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417 **6. Appendix: Numerical algorithm**

418 This appendix provides the detailed algorithm to reprogram the proposed single
419 element rheological model. Knowing the internal variables at the previous time
420 step n , the algorithm of a strain based method aims at calculating the total
421 stress S_{n+1} and the update of the internal variables for a given new total strain
422 E_{n+1} . In the alternative case of a stress based approach, an iterative method
423 is used to estimate a new strain E_{i+1} that requires additionally the tangent
424 operator dS_{i+1}/dE_{i+1} . At convergence of the iterative method, $E_{n+1} = E_{i+1}$
425 and the strain variables are updated accordingly. For a detailed insight in these
426 classical concepts, see for instance (Curnier, 1994).

427

428 *6.1. Hypothesis*

429 We first assume here that 1) the crack opening stress in compression is strictly
 430 lower than the crack closing stress in tension, and 2) the crack opening stress
 431 in tension is strictly higher than the crack closing stress in compression:

$$-\sigma^{c-} < -\sigma^{t-} \quad \sigma^{t+} > \sigma^{c+} \quad (1)$$

432 where all material constants σ are positive real numbers. These conditions
 433 are consistent with the experimental observations at the macroscopic scale and
 434 ensure that only one crack strain evolves at a given time in the model. Both
 435 conditions are maintained with respect to a change in the crack closure strains
 436 $E^{t,clo} > 0$.

437 *6.2. Failed compression crack*

438 If the damage variable $D_n^c = 1$ then the total stress

$$S_{i+1} = 0 \quad (2)$$

439 and the tangent stiffness

$$\frac{dS_{i+1}}{dE_{i+1}} = 0 \quad (3)$$

440 The algorithm stops here for that element and the variables $D_{n+1}^c = 1$, $E_{n+1}^c =$
 441 E_{n+1} and $E_{n+1}^t = 0$ are updated at convergence.

442 *6.3. Trial stresses*

443 If the damage variable $D_n^c = 0$, trial stresses $S^{c,trial}$ and $S^{t,trial}$ are computed
 444 that depend on the crack strains E_n^c , E_n^t and tensile damage D_n^t of the previous

445 time step:

$$S_{i+1}^{c,trial} = \epsilon(E_{i+1} - E_n^c - E_n^t) - \chi^c E_n^c \quad (4)$$

$$S_{i+1}^{t,trial} = \epsilon(E_{i+1} - E_n^c - E_n^t) - (1 - D_n^t)\chi^t E_n^t \quad (5)$$

446 *6.3.1. Elastic case*

447 In case no yield criterion is active

$$S_{i+1}^{c,trial} > -\sigma^{c-} \quad \text{and} \quad S_{i+1}^{c,trial} < \sigma^{c+} \quad (6)$$

$$S_{i+1}^{t,trial} < (1 - D_n^t)\sigma^{t+} \quad \text{and} \quad S_{i+1}^{t,trial} > -(1 - D_n^t)\sigma^{t-} \quad (7)$$

448 The response is elastic

$$S_{i+1} = \epsilon(E_{i+1} - E_n^c - E_n^t) \quad (8)$$

449 and the tangent is

$$\frac{dS_{i+1}}{dE_{i+1}} = \epsilon \quad (9)$$

450 Since no flow occurs, all internal variables are updated with their value at the

451 previous time step.

452 *6.3.2. Compression crack opening*

453 If $S_{i+1}^{c,trial} \leq -\sigma^{c-}$, the following projection is performed:

$$E_{i+1}^c = \frac{\epsilon(E_{i+1} - E_n^t) + \sigma^{c-}}{\epsilon + \chi^c} \quad (10)$$

454 The resulting stress remains

$$S_{i+1} = \epsilon(E_{i+1} - E_{i+1}^c - E_n^t) \quad (11)$$

455 The corresponding tangent is

$$\frac{dS_{i+1}}{dE_{i+1}} = \frac{\epsilon\chi^c}{\epsilon + \chi^c} \quad (12)$$

456 The update will enforce $E_{n+1}^c = E_{i+1}^c$ and $E_{n+1}^t = E_n^t$.

457 In case the failure threshold is attained,

$$E_{n+1}^c \leq E^{c,ult} \Rightarrow D_{n+1}^c = 1 \quad (13)$$

458 *6.3.3. Compression crack closing*

459 If $S_{i+1}^{c,trial} \geq +\sigma^{c+}$, the following projection is performed:

$$E_{i+1}^c = \text{Min}\left(0, \frac{\epsilon(E_{i+1} - E_n^t) - \sigma^{c+}}{\epsilon + \chi^c}\right) \quad (14)$$

460 The minimum ensures that the compression crack does not close beyond the

461 zero strain.

462 The resulting stress is

$$S_{i+1} = \epsilon(E_{i+1} - E_{i+1}^c - E_n^t) \quad (15)$$

463 If $E_{i+1}^c = 0$, the tangent is

$$\frac{dS_{i+1}}{dE_{i+1}} = \epsilon \quad (16)$$

464 else

$$\frac{dS_{i+1}}{dE_{i+1}} = \frac{\epsilon\chi^c}{\epsilon + \chi^c} \quad (17)$$

465 The update will enforce $E_{n+1}^c = E_{i+1}^c$ and $E_{n+1}^t = E_n^t$.

466

467 6.3.4. Tension crack opening

468 Then, if $S_{i+1}^{t,trial} \geq (1 - D_n^t)\sigma^{t+}$, the other projection is performed:

$$E_{i+1}^t = \frac{\epsilon(E_{i+1} - E_n^c) - (1 - D_n^t)\sigma^{t+}}{\epsilon + (1 - D_n^t)\chi^t} \quad (18)$$

469 The total stress remains

$$S_{i+1} = \epsilon(E_{i+1} - E_n^c - E_{i+1}^t) \quad (19)$$

470 The tangent is

$$\frac{dS_{i+1}}{dE_{i+1}} = \frac{\epsilon(1 - D_n^t)\chi^t}{\epsilon + (1 - D_n^t)\chi^t} \quad (20)$$

471 The update will enforce $E_{n+1}^t = E_{i+1}^t$ and $E_{n+1}^c = E_n^c$.

472 Finally, if the residual strain at convergence exceeds the failure threshold

$$E_{n+1}^t \geq E^{t,ult} \Rightarrow D_{n+1}^t = 1 \quad (21)$$

473 6.3.5. Tension crack closing

474 Then, if $S_{i+1}^{t,trial} \leq -(1 - D_n^t)\sigma^{t-}$, the alternative projection is performed:

$$E_{i+1}^t = \text{Max}(E^{t,clo}, \frac{\epsilon(E_{i+1} - E_n^c) + (1 - D_n^t)\sigma^{t-}}{\epsilon + (1 - D_n^t)\chi^t}) \quad (22)$$

475 The maximum ensures that the crack does not close beyond the limit strain

476 $E^{t,clo}$.

477 The total stress is

$$S_{i+1} = \epsilon(E_{i+1} - E_n^c - E_{i+1}^t) \quad (23)$$

478 If $E_{i+1}^t = E^{t, clo}$

$$\frac{dS_{i+1}}{dE_{i+1}} = \epsilon \quad (24)$$

479 else

$$\frac{dS_{i+1}}{dE_{i+1}} = \frac{\epsilon(1 - D_n^t)\chi^t}{\epsilon + (1 - D_n^t)\chi^t} \quad (25)$$

480 The update will enforce $E_{n+1}^t = E_{i+1}^t$ and $E_{n+1}^c = E_n^c$.

Exp./ Samples	Variables Units	ϵ [MPa]	χ [MPa]	σ^- [MPa]	σ^+ [MPa]	E^{ult} [-]	γ [-]
Fig. 2	ECM	21'700	—	—	—	—	—
Fig. 2	Comp. crack	—	543	456	64	0.070	—
Fig. 2	Tension crack	—	5'425	76	152	0.035	0.12
Fig. 4 L	ECM	24'000	—	—	—	—	—
Fig. 4 L	Comp. crack	—	600	432	43	0.085	—
Fig. 4 L	Tension crack	—	6'000	84	168	0.035	0.12
Fig. 4 R	ECM	22'500	—	—	—	—	—
Fig. 4 R	Comp. crack	—	600	473	47	0.070	—
Fig. 4 R	Tension crack	—	6'000	85	169	0.035	0.12
Fig. 5	ECM	30'000	—	—	—	—	—
Fig. 5	Comp. crack	—	7'500	270	49	0.020	—
Fig. 5	Tension interface	—	7'500	105	210	0.035	0.12
Fig. 6	ECM	26'000	—	—	—	—	—
Fig. 6	Comp. crack	—	650	546	98	0.070	—
Fig. 6	Tension crack	—	6'500	68	195	0.030	0.30
Fig. 7	ECM	21'700	—	—	—	—	—
Fig. 7	Comp. crack	—	543	260	26	0.050	—
Fig. 7	Tension crack	—	5'425	38	76	0.040	0.12
Fig. 8	ECM	17'000	—	—	—	—	—
Fig. 8	Comp. crack	—	425	405	20	0.046	—
Fig. 8	Tension crack	—	4'250	61	122	0.028	0.12

Table 1: Material constants for the three components of the rheological model used in Fig. 2 and the six experiments/samples shown in Fig. 4-8. The letters "L" and "R" stand for left and right.

Figure 1: Top: Rheological model of a linear elastic bone matrix (spring) with compression and tension cracks that can open and close (sliders) with bridging collagen fibrils (kinematic hardening) and locking mechanisms (stops) in the opposite loading mode. When the sliders reach the open end of the support, the crack elements reach an ultimate strain and undergo brittle failure. Bottom: Stress-strain behavior of the three rheological elements for two loading cycles with increasing amplitudes. The spring (E^e) shows linear elasticity, while the compression crack (E^c) and the tension crack (E^t) exhibit plasticity with kinematic hardening followed by brittle failure beyond the ultimate strains ($E^{c,ult}$ and $E^{t,ult}$). The crack models exhibit distinct flow stresses in compression ($\sigma^{c\pm}$) and tension ($\sigma^{t\pm}$) but undergo locking in the opposite loading mode as long as they do not fail. The position of the tensile stop increases with the maximal extension strain. Upon brittle failure (dotted lines), the compression crack does not carry stress in any mode ($D^c = 1$), while the tensile crack can still sustain compressive stress ($D^t = 1$).

Figure 2: Response of the single element model to cyclic loading with increasing amplitudes. The tensile crack shows a strong hardening slope, while opening of the compression crack requires a higher stress amplitude. Failure is only partial in extension as it can still sustain load in shortening but the failure becomes complete beyond a given shortening threshold. An animation of the loading and stress-strain curves is available in the supplementary material.

Figure 3: Parallel arrangement of rheological elements with statistical distribution of parameters. The elastic parameters of the ECM and the collagen fibrils are distributed normally, while the yield and failure parameters are assigned a uniform distribution.

Figure 4: Response of the model to monotonic compression (left) and monotonic tension (right). In black, the experimental curves from (Mirzaali et al., 2015) and in blue with increasing intensity, the simulation curves with 120, 240, 480, 960 and 1920 elements. Mean changes in stress remain below 1% beyond $n=240$. In compression, the ultimate stress is about 190 MPa and the ultimate strain approximately 0.015. In tension, the yield stress is approximately 90 MPa and no ultimate strain can be defined. The enhanced ductility in tension is due to the higher hardening slope. The material constants were adapted to the two experimentally tested bovine bone samples (see Table 1) and kept identical for the convergence study.

Figure 5: Response of the model to cyclic compression. In the upper left graph, the applied strain schedule and below the resulting stress. In the upper right graph, the experimental stress-strain curve from (Mirzaali et al., 2015) and below the simulation curve in dark blue superimposed to the experimental curve in light gray. The overloading in compression produces substantial damage in tension. The material constants were adapted to the tested bone sample and provided in Table 1.

Figure 6: Response of the model to cyclic tension. In the upper left graph, the applied strain schedule and below the resulting stress. In the upper right graph, the experimental stress-strain curve from(Mirzaali et al., 2015) and below the simulation curve in dark blue superimposed to the experimental curve in light gray. The overloading in tension produces essentially no damage in compression, but the cracks do not close completely. The material constants were adapted to the tested bone sample and provided in Table 1.

Figure 7: Response of the model to cyclic compression followed by monotonic tension. In the upper left graph, the applied strain schedule and below the resulting stress. In the upper right graph, the experimental stress-strain curve from(Mirzaali et al., 2015) and below the simulation curve in dark blue superimposed to the experimental curve in light gray. The ultimate stress in tension is substantially reduced by damage in compression. The material constants were adapted to the tested bone sample and provided in Table 1.

Figure 8: Response of the model to cyclic tension followed by monotonic compression. In the upper left graph, the applied strain schedule and below the resulting stress. In the upper right graph, the experimental stress-strain curve from(Mirzaali et al., 2015) and below the simulation curve in dark blue superimposed to the experimental curve in light gray. The ultimate stress in compression is not influenced by damage in tension. The material constants were adapted to the tested bone sample and provided in Table 1.

Figure
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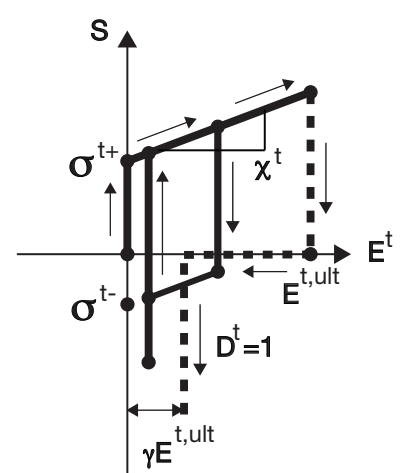
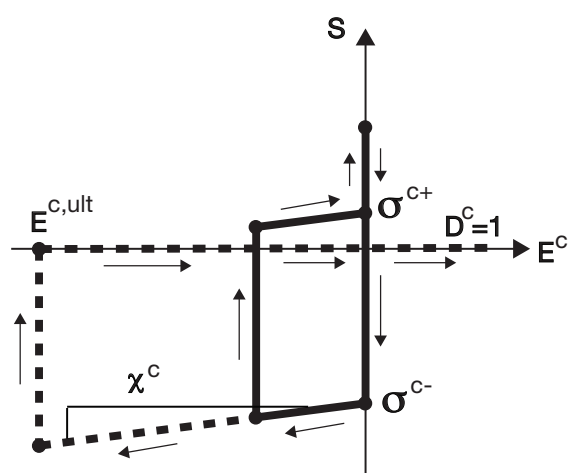
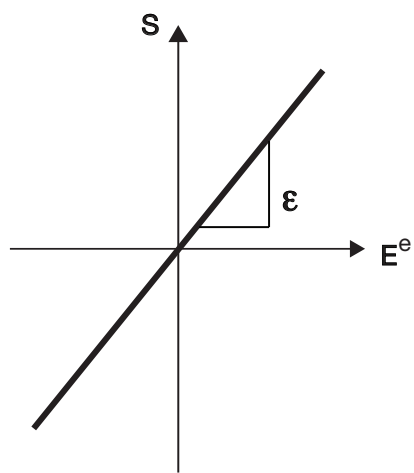
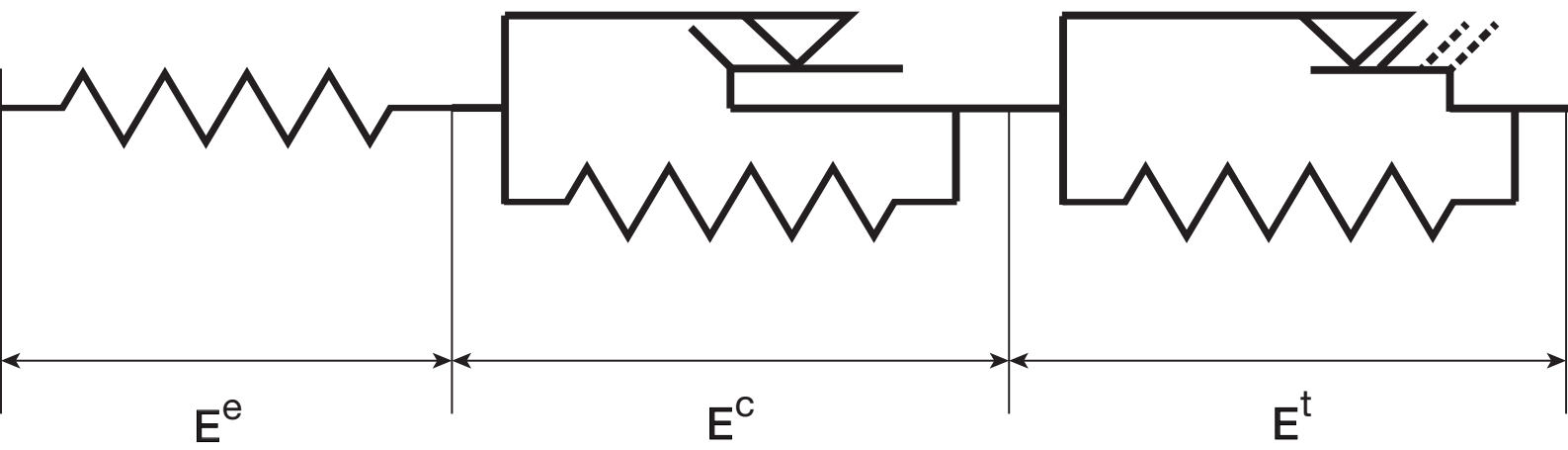


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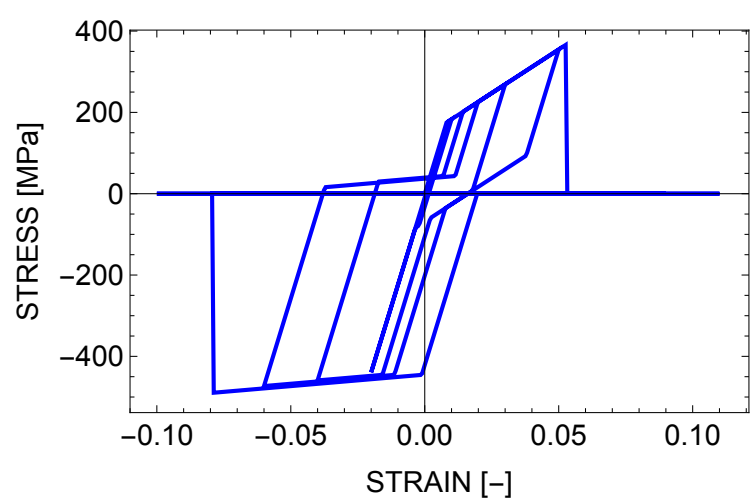
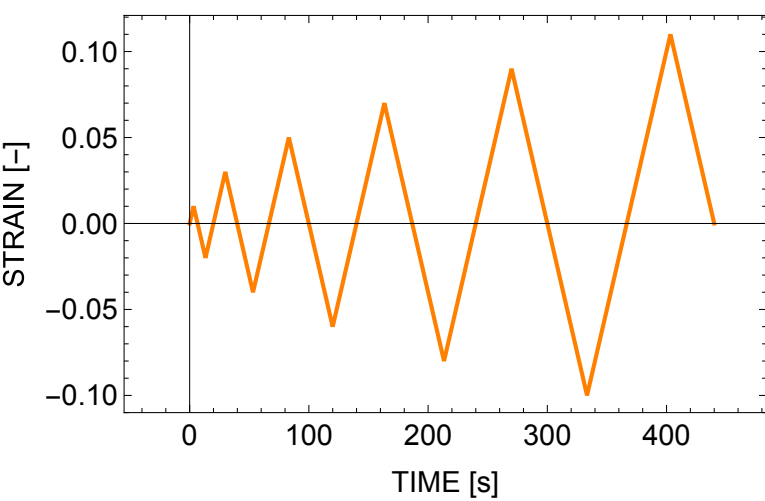
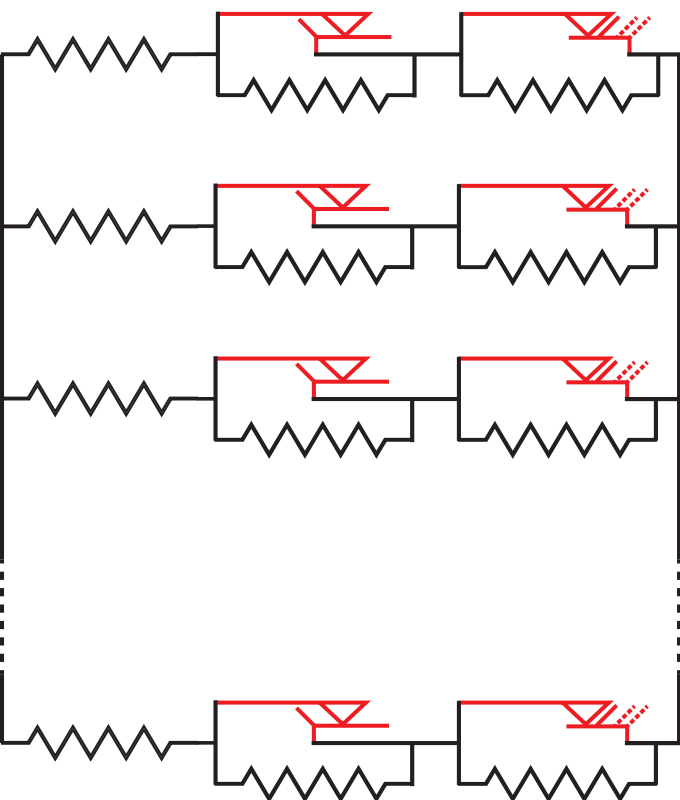
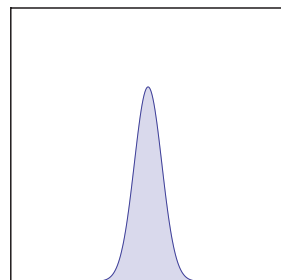


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ECM



Cracks

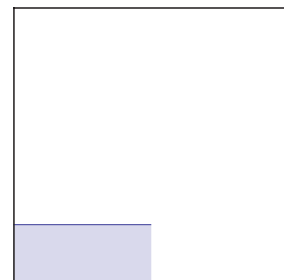


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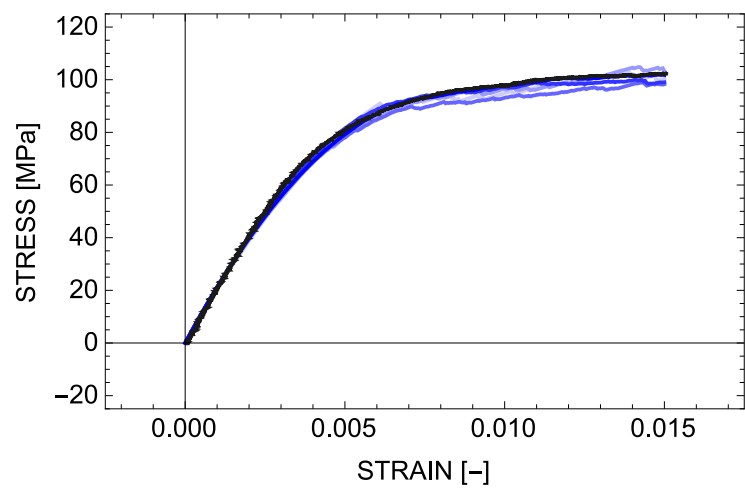
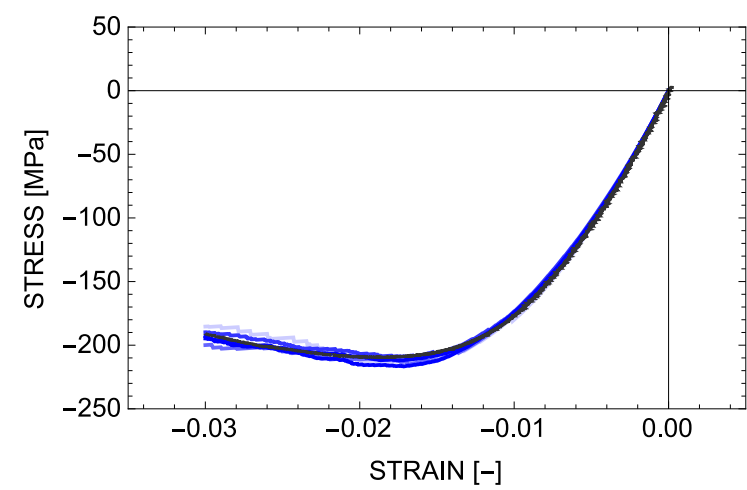


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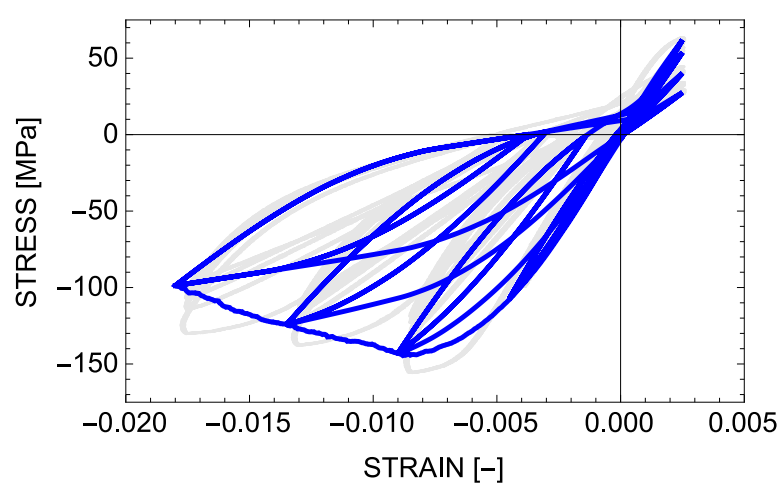
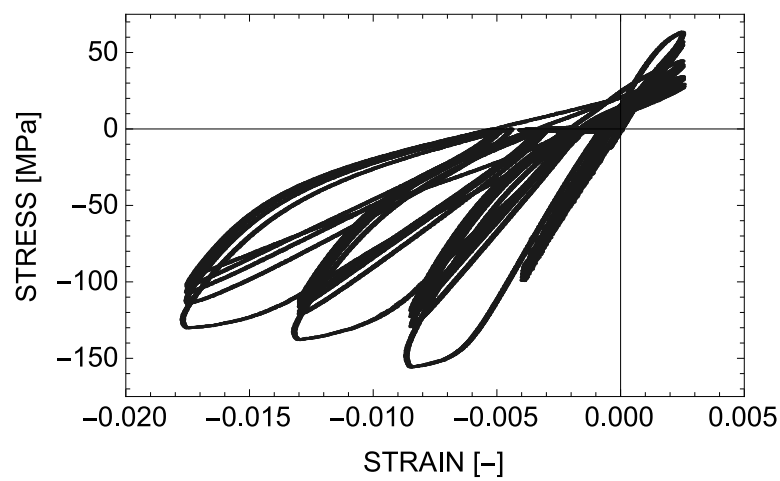
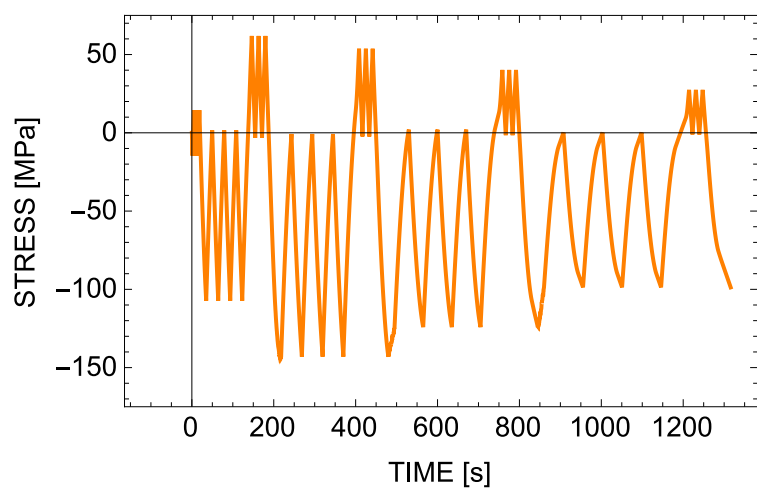
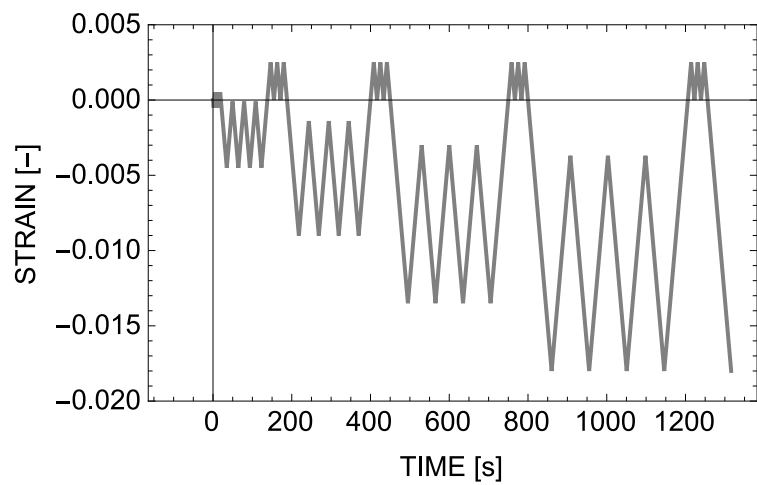


Figure
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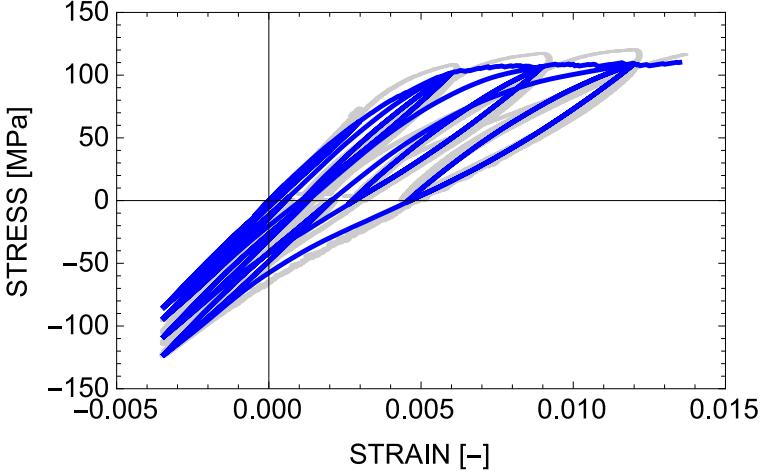
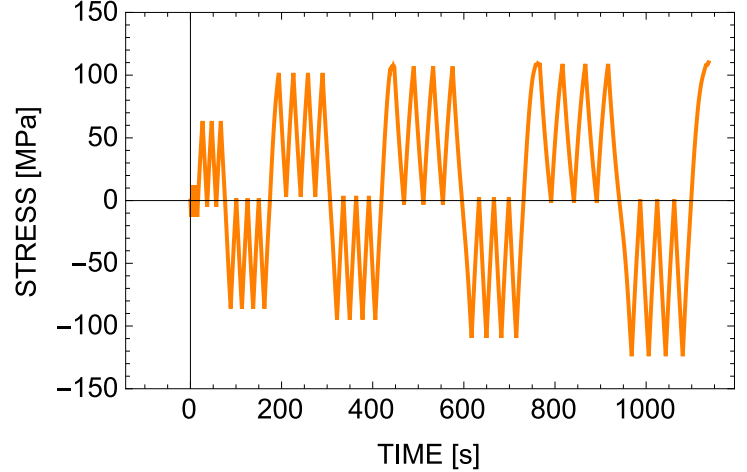
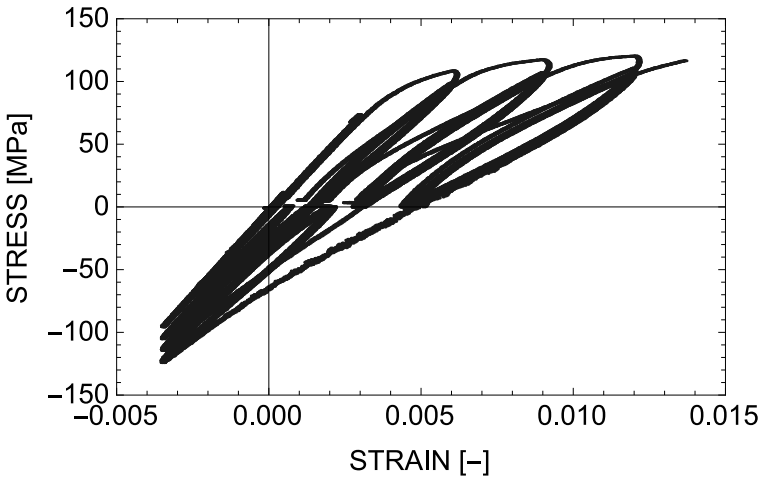
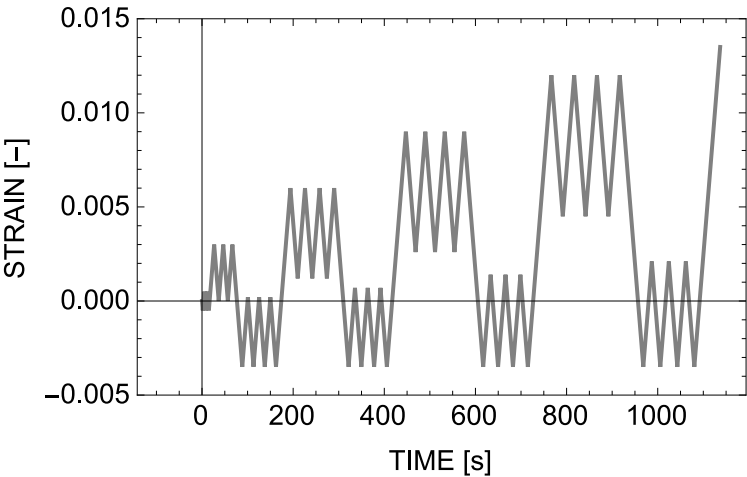


Figure
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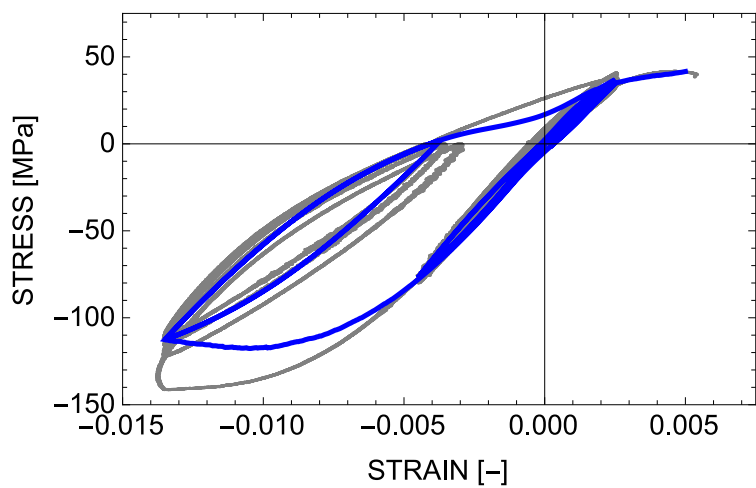
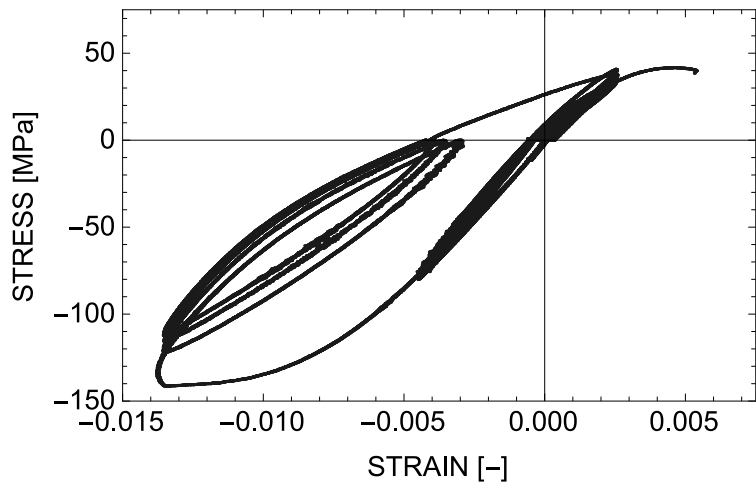
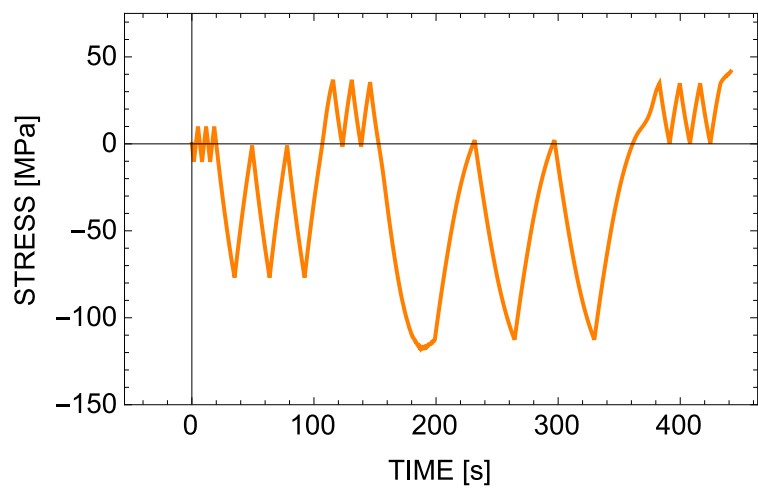
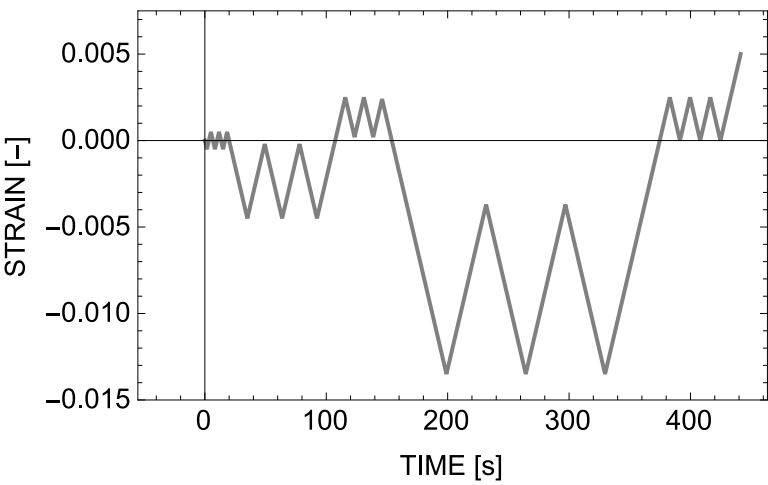


Figure
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