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### ► To cite this version:

Aline Brunon, Michel Coret, Karine Bruyere-Garnier, Alain Combescure. Comparison of two homogenization methods using a damage model for a fibrous membrane, based on the fibers' fracture process at the microscale.. European Journal of Mechanics - A/Solids, 2013, 39, pp.1-10. 10.1016/j.euromechsol.2012.10.006 . hal-00789316v1

**HAL Id: hal-00789316**

**<https://hal.science/hal-00789316v1>**

Submitted on 19 Feb 2013 (v1), last revised 29 May 2018 (v2)

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# Comparaison of two homogenization methods using a damage model for a fibrous membrane, based on the fibers' fracture process at the microscale.

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## Abstract

The objective of this paper is to evaluate two existing homogenization methods using a simple damage model for fibrous membrane and compare them. A macroscopic damage model of a fibrous membrane tissue based on the knowledge of the behavior and fracture process of a single fiber is constructed. Under some simplifying assumptions (linear behavior of the fibers, brittle fibers, constant angular distribution of the fibers), the evolution of fiber fracture is described by two macroscopic scalar variables; this enables the tissue's damage to be modeled without requiring a second discretization at the microscale. For the homogenization of the fibers contribution, an energetic method and a kinematic method are adapted and compared. Both lead to similar macroscopic behavior in the elastic phase, but the behavior differs in the fracture

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phase of the homogenized material. In the case of biological materials, the proposed law enables taking into account the often observed phenomena of crimping and damage of the fibers.

*Key words:* fibrous membrane tissue, damage, microstructure, homogenization, biaxial tension

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## 1. Introduction

The question of the fracture modeling of fibrous tissues arises in many domains, including composites, papers and biological tissues. In the latter case, the large presence of fibrous tissues of different natures in the human body lead to an general research effort to explain how the macroscopic behavior of those tissues is related to their microstructure, especially in the case of non-homogeneous fiber distributions. In terms of modeling, two aspects have to be considered : 1) the fibrous nature of the tissue and 2) the evolution of damage in the tissue. Several options are proposed in the literature to assess the first point. Using invariants to take into account the isotropic or anisotropic nature of the fibrous contribution is a first option, introduced in (Holzapfel, 2000). This method does not require any further homogenization as the invariants already are a macroscopic representation of the fiber orientations and assumes that the fibers are perfectly aligned along one or several directions. If the fibers are distributed around one or several preferred directions, a statistical distribution of the fibers is generally introduced. From there, two options are suggested in the literature: either a structure tensor (Gasser et al., 2006) can be used to provide a macroscopic representation of the microstructure (the *kinematic method*) or the individual energetic con-

20 tributions of the fibers are summed up (Lanir, 1983) (the *energetic method*).  
21 A more recent option is the microsphere-based approach (Menzel and Waf-  
22 fenschmidt, 2009; Sáez et al., 2012) which uses spherical units in which the  
23 homogenization process is conducted. The anisotropy of the tissue is then  
24 described by associating different weights to the fiber directions. Using a  
25 limited number of integration directions was proved to be enough to provide  
26 good accuracy as well as a low computational cost to the two-scale simulation  
27 of 3D tissues. Another recent feature is the macromolecular network model  
28 proposed by (Kuhl et al., 2005), based on eight-chain unit cells and which  
29 can handle fiber reorientation.

30 In this paper, the attention is focused on how the fiber distribution is  
31 taken into account: both the kinematic and energetic methods are adapted  
32 to the same fibrous tissue model to assess whether and in which domain they  
33 are equivalent.

34 The second point in modeling the fracture of fibrous tissue is the de-  
35 scription of the damage. In the field of biomechanics, the problem of the  
36 fracture of fibrous tissues has received only limited attention. Some authors  
37 model damage continuously using an internal damage variable which is to  
38 be identified for 1D (Calvo et al., 2007) or 2D tissues (Balzani et al., 2006).  
39 (Rodríguez et al., 2006) introduces a 3D model which also describes dam-  
40 age continuously, but uses two independent variables for the matrix and the  
41 fibers. Other papers describe damage in the tissue as the result of fiber  
42 fracture on the lower scale: (Hurschler et al., 1997) proposes a tendon-and-  
43 ligament model involving three scales (the fibrils', the fibers' and the tis-  
44 sue's) and studies the behavior and damage of the tissue in the case of fibers

45 aligned with a single direction. (Liao and Belkoff, 1999) also proposes a  
46 fracture model of a 1D tissue, but takes into account the initial crimping of  
47 the fibers. (Gasser and Holzapfel, 2006) models the fracture of arteries by  
48 dissection, *i.e.* splitting through the thickness, which constitutes a different  
49 fracture mode than surface tearing. (Cacho et al., 2007) proposes a damage  
50 model for a fibrous tissue made of initially crimped fibers. More recently,  
51 (Sáez et al., 2012) introduced damage in a microsphere model of the blood  
52 vessel taking into account its initial anisotropy.

53 The fibrous tissue modeling in the present paper is similar to that from  
54 (Cacho et al., 2007). The idea of our model is to describe the evolution of  
55 the macroscopic damage in a fibrous membrane and therefore of the tissue  
56 anisotropy during a biaxial strain loading; under simplifying assumptions, it  
57 will be shown that two scalar variables are sufficient for this purpose. This  
58 simple model, including some extensions related to biological tissues, is used  
59 to illustrate the comparison of the two homogenization methods.

60 The outline of this paper is the following: the first part presents the  
61 two homogenization methods adapted to a fibrous membrane model; the  
62 second part is focused on the description of the simple damage model that we  
63 propose; the third part illustrates the comparison of the two homogenization  
64 methods using our damage model; the fourth part presents extensions of the  
65 damage model to biological phenomena such as damageable fibers and fiber  
66 crimp, as well as the consequences on the homogenization methods.

## 67 2. Modeling the tissue fracture

68 Let us consider a fibrous membrane and suppose that we know the in-  
69 fluence of a biaxial tension loading on the fibers breakage. We can proceed  
70 to homogenize the behavior. Several homogenization methods are available  
71 in the literature; the most commonly used are the ones described in (Lanir,  
72 1983) and (Gasser et al., 2006). The first one consists in an energetic ho-  
73 mogenization of the fibers while the second is based on a kinematic homoge-  
74 nization. In this paper, these two methods are adapted to the fibrous tissue  
75 damage problem and compared.

### 76 2.1. General framework

77 This section takes up the same general framework as in (Gasser et al.,  
78 2006). We consider a plane tissue consisting of a matrix and fibers. The  
79 free energy  $\psi$  of the tissue is the sum of the free energy of the matrix  $\psi_m$   
80 and the free energy of the fibers  $\psi_f$ . A common way to enforce compress-  
81 ibility/incompressibility conditions is to split the strain energy of the matrix  
82 into a volumetric part  $U_m$  and an isochoric part  $\bar{\psi}_m$ . It is usually assumed  
83 that the incompressibility of biological tissues is due to the water content  
84 of the ground matrix and that the fibers' contribution is **negligible** in the  
85 volumetric part of the free energy. Therefore, it is not necessary to split the  
86 fibers' contribution into volumetric and isochoric parts.

87 Hence the free energy of the tissue can be written as:

$$\psi = \psi_m + \psi_f = U_m + \bar{\psi}_m + \psi_f \quad (1)$$

88 First we will address the matrix free energy expression; then, we will  
 89 examine two homogenization methods for the contribution of the fibers.

90 *2.2. Contribution of the matrix*

The volumetric/isochoric decomposition is applied to the gradient of transformation  $\mathbf{F}$ , *i.e.* to the right Cauchy-Green strain tensor  $\mathbf{C}$  :

$$\mathbf{F} = J^{\frac{1}{3}} \bar{\mathbf{F}} \quad (2)$$

$$\mathbf{C} = J^{\frac{2}{3}} \bar{\mathbf{C}} \quad (3)$$

91

with  $J = \det(\mathbf{F})$ . The cumulative energy decomposition enables the second Piola-Kirchhoff stress tensor (PK2) to be expressed as:

$$\mathbf{S}_m = 2 \frac{\partial \psi_m}{\partial \mathbf{C}} = 2 \left( \frac{\partial U_m}{\partial \mathbf{C}} + \frac{\partial \bar{\psi}_m}{\partial \mathbf{C}} \right) = \mathbf{S}_m^{vol} + \bar{\mathbf{S}}_m \quad (4)$$

92

We set  $p = \partial U_m / \partial J$ ; we also assume that the matrix follows an isotropic, neo-Hookean behavior expressed by  $\bar{\psi}_m = c (\text{tr} \bar{\mathbf{C}} - 3) / 2$ , where  $c$  is a material parameter. Then:

$$\mathbf{S}_m^{vol} = 2 \frac{\partial U_m}{\partial \mathbf{C}} = 2 \frac{\partial U_m}{\partial J} \frac{\partial J}{\partial \mathbf{C}} = p J \mathbf{C}^{-1} \quad (5)$$

$$\bar{\mathbf{S}}_m = 2 \frac{\partial \bar{\psi}_m}{\partial \mathbf{C}} = 2 \frac{\partial \bar{\psi}_m}{\partial \bar{\mathbf{C}}} : \frac{\partial \bar{\mathbf{C}}}{\partial \mathbf{C}} = J^{-\frac{2}{3}} \mathbb{P} : c \mathbf{I} \quad (6)$$

93 with the fourth-order operators  $\mathbb{P} = \mathbb{I} - \frac{1}{3} \mathbf{C}^{-1} \otimes \mathbf{C}$  and  $I_{ijkl} = (\delta_{ik} \delta_{jl} + \delta_{il} \delta_{jk}) / 2$ ,  
 94 where  $\delta_{ij}$  is Kronecker's symbol.

95 Thus, the contribution of the matrix to PK2 tensor becomes:

$$\mathbf{S}_m = p J \mathbf{C}^{-1} + J^{-\frac{2}{3}} \mathbb{P} : c \mathbf{I} \quad (7)$$

96 *2.3. Contribution of the fibers*

97 *2.3.1. The concept of fiber density function*

98 In order to describe the strain energy in the tissue, we introduce the  
99 concept of angular fiber density, denoted  $\rho(\xi)$ . This function defines the  
100 number of the fibers whose orientation belongs to the interval  $[\xi, \xi + d\xi]$ .  
101 Before damage occurs, this function is normalized, *i.e.*:

$$\frac{1}{\pi} \int_{-\frac{\pi}{2}}^{\frac{\pi}{2}} \rho(\xi) d\xi = \frac{1}{\pi} \int_{A_0} \rho(\xi) d\xi = 1 \quad (8)$$

102 In the case of a homogeneous distribution, *i.e.*  $\rho(\xi) = \text{constant}$ ,  $\rho(\xi) =$   
103  $1 \forall \xi \in A_0 = [-\pi/2, \pi/2]$ .

104 Once damage occurs, the density is a function of damage state  $\mathbf{D}$ . The  
105 integration domain evolves and is then denoted  $A(\mathbf{D})$ . Given this description,  
106 we can write :

$$\int_{A_0} \rho(\xi, \mathbf{D}) d\xi = \int_{A(\mathbf{D})} \rho(\xi) d\xi \quad (9)$$

107  $A(\mathbf{D})$  represents the domain where the fibers are not broken.

108 *2.3.2. The Energetic (E) homogenization method*

109 In this method, the strain energy of the fibrous part of the tissue is  
110 assumed to be the sum of the strain energies of the strained, but undamaged,  
111 fibers. A fiber subjected to a Green-Lagrange strain field  $\mathbf{E}$  is strained only  
112 along its longitudinal axis  $\mathbf{n}(\xi)$  and its strain energy is  $\phi_f = \phi_f(\varepsilon_f)$  *i.e.*



113  $\phi_f(\xi, \mathbf{E})$ , whose expression depends on the constitutive relation of the fiber.

114 Therefore, on the tissue's scale, the free energy  $\psi_f^E$  of the fibers is:

$$\psi_f^E(\mathbf{E}, \mathbf{D}) = \frac{1}{\pi} \int_{A_0} \rho(\xi, \mathbf{D}) \phi_f(\xi, \mathbf{E}) \, d\xi \quad (10)$$

115 The expression of stress tensor PK2 is:

$$\mathbf{S}_f^E = \frac{\partial \psi_f^E(\mathbf{E}, \mathbf{D})}{\partial \mathbf{E}} = \frac{1}{\pi} \int_{A_0} \rho(\xi, \mathbf{D}) \frac{\partial \phi_f(\xi, \mathbf{E})}{\partial \mathbf{E}} \, d\xi \quad (11)$$

116 Now let us consider the particular case where the fibers behave linearly  
117 before damage. The total free energy of the fibrous part of the tissue is:

$$\psi_f^E(\mathbf{E}, \mathbf{D}) = \frac{1}{\pi} \int_{A_0} \rho(\xi, \mathbf{D}) \frac{1}{2} E (\mathbf{M} : \mathbf{E})^2 \, d\xi \quad (12)$$

118 where  $E$  is the Young's modulus of the fiber's material and  $\mathbf{M} = \mathbf{n}(\xi) \otimes$   
119  $\mathbf{n}(\xi)$  the orientation tensor (see Section 3). Thus, the expression of stress  
120 tensor PK2 is:

$$\mathbf{S}_f^E = \frac{E}{\pi} \int_{A_0} \rho(\xi, \mathbf{D}) (\mathbf{M} : \mathbf{E}) \mathbf{M} \, d\xi \quad (13)$$

121 Eq. (11) is valid if we suppose that the variation of the integral of the  
122 density function  $\int_{A_0} \rho(\xi, D)$  with strain is negligible compared to the vari-  
123 ation of  $\phi_f(\xi, \mathbf{E})$ , which is the case for the simple model considered in this  
124 approach. Therefore only the inner member of the integral is differentiated  
125 with respect to  $\mathbf{E}$ .

126 *2.3.3. The Kinematic (K) method*

127 The *kinematic* method is derived from works described in (Gasser et al.,  
 128 2006), among others. We introduce a generalized second-order structure  
 129 tensor  $\mathbf{H}$  defined by Eq.(14). This tensor is used as a macroscopic projector  
 130 of the strain tensor onto the structure of the undamaged fibers.

$$\mathbf{H} = \frac{1}{\pi} \int_{A_0} \rho(\xi, D) \mathbf{n}(\xi) \otimes \mathbf{n}(\xi) d\xi \quad (14)$$

131  $H_{33}$  equals zero because the fibers are oriented only perpendicularly to  
 132 Direction 3.

133 Thus, the constitutive law is applied to the tissue rather than to its  
 134 constituent fibers, taking the scalar  $E_h = \mathbf{H} : \mathbf{E}$  as the strain value to  
 135 express the macroscopic strain energy  $\psi_f^K$  here in the linear case (Eq.15).

$$\psi_f^K = \frac{1}{2} E_h E_h^2 = \frac{1}{2} E (\mathbf{H} : \mathbf{E})^2 \quad (15)$$

From that expression, we deduce the PK2 tensor corresponding to the *K*  
 method:

$$\mathbf{S}_f^K = \frac{\partial \psi_f^K}{\partial \mathbf{E}} = E (\mathbf{H} : \mathbf{E}) \mathbf{H} \quad (16)$$

136 Once again, the damage variable  $\mathbf{D}$  and the Green-Lagrange strain tensor  $\mathbf{E}$   
 137 are independent variables. So  $\mathbf{H}$  is not differentiated with respect to  $\mathbf{E}$ .

138 *2.4. Association of contributions from the matrix and the fibers*

139 Let us recall that the total energy of the tissue can be divided into three  
 140 parts (Eq.1) including two from the matrix contribution (Eq.7).

141 Thus, depending on the homogenization method, tensor PK2 has either  
 142 of the two forms:

$$\mathbf{S}^E = pJ\mathbf{C}^{-1} + J^{-\frac{2}{3}}\mathbb{P} : c\mathbf{I} + \frac{2E}{\pi} \int_{A_0} \rho(\xi, \mathbf{D}) (\mathbf{M} : \mathbf{E}) \mathbf{M} d\xi \quad (17)$$

$$\mathbf{S}^K = pJ\mathbf{C}^{-1} + J^{-\frac{2}{3}}\mathbb{P} : c\mathbf{I} + E (\mathbf{H} : \mathbf{E}) \mathbf{H} \quad (18)$$

143 Since the objective is to implement the model into a finite element calcu-  
 144 lation code, the tissue is modeled in 3D and plane stresses; in order to apply  
 145 this condition, we define the Cauchy stress tensor  $\mathbf{T}$ :

$$\mathbf{T} = J^{-1}\mathbf{F}.\mathbf{S}.\mathbf{F}^T \quad (19)$$

The components derived from the matrix contribution are:

$$\mathbf{T}_m^{vol} = p\mathbf{I} \quad (20)$$

$$\bar{\mathbf{T}}_m = J^{-\frac{5}{3}}c \left( \mathbf{B} - \frac{1}{3}tr(\mathbf{C}^{-1})\mathbf{I} \right) \quad (21)$$

146 where  $\mathbf{B} = \mathbf{F}.\mathbf{F}^T$  is the left Cauchy-Green tensor. Concerning the contri-  
 147 bution of the fibers, we distinguish the two homogenization methods:

$$\mathbf{T}_f^E = J^{-1}\mathbf{F}.\mathbf{S}_f^E.\mathbf{F}^T = \frac{2E}{J\pi} \mathbf{F} \cdot \int_{A_0} \rho(\xi, \mathbf{D}) (\mathbf{M} : \mathbf{E}) \mathbf{M} d\xi \cdot \mathbf{F}^T \quad (22)$$

$$\mathbf{T}_f^K = J^{-1}\mathbf{F}.\mathbf{S}_f^K.\mathbf{F}^T = \frac{E}{J} (\mathbf{H} : \mathbf{E}) \mathbf{F}.\mathbf{H}.\mathbf{F}^T \quad (23)$$

148 We can notice that the fibrous contribution to the overall tissue does  
 149 not create a Cauchy stress components out of the plane; this is due to the  
 150 formulation of the fiber density function and to the fact that we do not  
 151 split the fibrous contribution into a volumetric component and an isochoric  
 152 component.

153 The plane stress condition is expressed by:

$$T_{33} = p + J^{-\frac{5}{3}}c \left( \lambda_3^2 - \frac{1}{3} (\lambda_1^2 + \lambda_2^2 + \lambda_3^2) \right) = 0 \quad (24)$$

154 We can observe that this equation involves  $p$ , the material characteristics  
 155 of the matrix and the components  $\lambda_i$  of the deformation gradient tensor.  
 156 Here, as is commonly assumed for soft biological tissues, the matrix is con-  
 157 sidered to be incompressible. In this case, the strain component across the  
 158 thickness is defined by Eq.(25) and  $p$  is a Lagrange multiplier determined by  
 159 the plane stress condition (Eq.26).

$$J = 1 \Leftrightarrow \lambda_3 = \frac{1}{\lambda_1 \lambda_2} \quad (25)$$

$$p = -\frac{c}{3} \left( \frac{2}{\lambda_1^2 \lambda_2^2} - \lambda_1^2 - \lambda_2^2 \right) \quad (26)$$

160 The two homogenization methods have been described; the next section  
 161 presents the damage model that was constructed to compare them.

### 162 **3. A simple kinematic fracturing fiber model**

163 In this section, we propose a simple model to describe the evolution of  
 164 the macroscopic damage of a plane fibrous tissue subjected to biaxial tension

165 loading, which is assumed to be the result of fiber breakage on the microscale.  
166 The two objectives of this section are first, **to build a damage evolution law**  
167 **for the macroscale damage, taking into account its anisotropic nature due**  
168 **to the microscale phenomena**; second, to compare the two homogenization  
169 methods presented in the previous section.

170 The underlying assumptions of this section are the following:

- 171 • the fibers are rectilinear, linear elastic, and brittle;
- 172 • prior to damage, the angular distribution function of the fibers is  
173 known, continuous and nonzero;
- 174 • the phenomenon of fiber reorientation during loading is neglected ;
- 175 • the *principal* directions of the biaxial strain loading do not change.

176 The **third** assumption relies on the conclusions of Sacks and Gloeckner  
177 (1999), which observed that the closer the loading to equibiaxiality, the lesser  
178 the reorientation of the fibers. Therefore, in the framework of the present  
179 study which focuses on biaxial loading, it appears acceptable to ignore fiber  
180 reorientation.

181 This model is academic. It is designed to produce clear conclusions when  
182 the two homogenization methods proposed in the previous section are com-  
183 pared. **It can be extended using a two scale approach to more realistic situ-**  
184 **ations as uncrimping, damage, non isotropic fiber orientations: the price to**  
185 **pay to these extensions is a larger number of internal variables to describe**  
186 **the small scale state.**

187 *3.1. Parameterization of the problem*

188 Let us consider a plane fibrous tissue. In the material plane  $(\mathbf{X}_m, \mathbf{Y}_m, \mathbf{Z}_m)$ ,  
 189 the direction of a fiber is characterized by the angle  $\xi \in [-\pi/2, \pi/2]$  and its  
 190 initial direction vector  $\mathbf{n}$  defined by:

$$\mathbf{n} = \cos \xi \mathbf{X}_m + \sin \xi \mathbf{Y}_m \quad (27)$$

191 The tissue is subjected to a biaxial strain characterized by the macro-  
 192 scopic Green-Lagrange strain tensor  $\mathbf{E}$  described in Cartesian coordinates  
 193 by:

$$\mathbf{E} = \varepsilon_r k (\cos \varphi \mathbf{X}_m \otimes \mathbf{X}_m + \sin \varphi \mathbf{Y}_m \otimes \mathbf{Y}_m) \quad (28)$$

$$= E_1 \mathbf{X}_m \otimes \mathbf{X}_m + E_2 \mathbf{Y}_m \otimes \mathbf{Y}_m \quad (29)$$

194

195 where  $\varepsilon_r$  is the ultimate longitudinal strain of the fibers.  $\varphi$  is the loading  
 196 angle. From here on, we will assume that  $\varphi \in [0, \pi/2]$  and  $k \geq 0$ , which  
 197 implies strict biaxial tension.

198 The fibers constituting the tissue are uniaxial elements which can with-  
 199 stand only solicitations along their axis. The longitudinal **Green** strain  $\varepsilon_f$  of  
 200 a fiber oriented along an angle  $\xi$  is defined by:

$$\varepsilon_f = \mathbf{n}(\xi) \cdot \mathbf{E} \cdot \mathbf{n}(\xi) = k \varepsilon_r (\cos \varphi \cdot \cos^2 \xi + \sin \varphi \sin^2 \xi) \quad (30)$$

201 We can observe that for  $\varphi = \frac{\pi}{4}$  all the fibers are **solicited** equally; then,  
 202 their longitudinal strain is  $\varepsilon_f = \frac{k \varepsilon_r}{\sqrt{2}}$ . Also, differentiating  $\varphi$  with respect to

203  $\xi$  shows that the most highly loaded fibers are oriented along the principal  
 204 directions of the strain tensor, that is  $\xi = 0$  or  $\xi = \frac{\pi}{2}$ .

### 205 3.2. Initial elasticity range

206 The elasticity range  $\mathcal{D}$  of a fiber is defined in the strain space by:

$$\mathcal{D} = \{\varepsilon_f / F(\varepsilon_f) = \varepsilon_f - \varepsilon_r < 0\} \quad (31)$$

207 The corresponding elasticity range of the tissue, denoted  $\mathcal{S}$ , is:

$$\mathcal{S} = \{\mathbf{E} / \forall \xi, \mathbf{n}(\xi) \cdot \mathbf{E} \cdot \mathbf{n}(\xi) - \varepsilon_r < 0\} \quad (32)$$

The shape of  $\mathcal{S}$  corresponds to the resolution of the equation  $\varepsilon_f - \varepsilon_r < 0$   
 and is described by:

$$\begin{cases} k < \frac{1}{\cos\varphi} & \forall \varphi \in [0, \frac{\pi}{4}] \\ k < \frac{1}{\sin\varphi} & \forall \varphi \in [\frac{\pi}{4}, \frac{\pi}{2}] \end{cases} \quad (33)$$

208 At the boundary of  $\mathcal{S}$ , at least one fiber breaks as the non-rupture crite-  
 209 rion is not respected anymore (Eq.31). The first fiber to break is always the  
 210 one oriented along  $\xi = 0$  if  $\varphi \leq \frac{\pi}{4}$  or the one oriented along  $\xi = \frac{\pi}{2}$  if  $\varphi \geq \frac{\pi}{4}$ .  
 211 The next section describes the damage process of the fibrous tissue.

### 212 3.3. Damage evolution

213 We now consider different loading cases to study the evolution of the  
 214 damage state of this model at the micro scale. Different cases are treated to  
 215 better understand the potentialities of the model.

216 *Case of a proportional loading.* We assume that the tissue is subjected to  
 217 a proportional strain loading (*i.e.* with  $\varphi$  constant) so as to reach a point  
 218 defined by  $(k, \varphi)$  such that:

$$\begin{cases} k > \frac{1}{\cos\varphi} & \text{if } \varphi \in \left[0, \frac{\pi}{4}\right] \\ k > \frac{1}{\sin\varphi} & \text{if } \varphi \in \left[\frac{\pi}{4}, \frac{\pi}{2}\right] \end{cases}$$

219 The damaged state at the micro scale is defined by two subsets: the  
 220 subset of healthy fibers and the set of broken ones. These sets are simply  
 221 defined by two angles  $\xi_1$  and  $\xi_2$ . From here on, the vector of the two damage  
 222 variables  $\xi_1$  and  $\xi_2$  will be denoted  $\mathbf{D}$  which characterizes the damaged tissue  
 223 state. These angles are obtained by the solution of equation  $\varepsilon_f(\xi) - \varepsilon_r > 0$   
 224 (which is detailed in Appendix A). A proportional loading with an intensity  
 225  $k$  greater than the bounds defined in Eq.(33) leads to the fracture of the  
 226 fibers as follows:

$$\varphi \in \left[0, \frac{\pi}{4}\right] : \begin{cases} \text{all fibers are broken } \forall \xi \in [-\xi_1, \xi_1] \\ \xi_1 = \arccos \sqrt{\frac{1 - k \sin\varphi}{k(\cos\varphi - \sin\varphi)}} \end{cases} \quad (34)$$

$$\varphi = \frac{\pi}{4} : \text{all fibers break simultaneously at } k = \sqrt{2} \text{ (Eq.30)} \quad (35)$$

$$\varphi \in \left[\frac{\pi}{4}, \frac{\pi}{2}\right] : \begin{cases} \text{all fibers are broken } \forall \xi \in \left[-\frac{\pi}{2}, -\xi_2\right] \cup \left[\xi_2, \frac{\pi}{2}\right] \\ \xi_2 = \arccos \sqrt{\frac{1 - k \sin\varphi}{k(\cos\varphi - \sin\varphi)}} \end{cases} \quad (36)$$

227 Micro scale damage state and the angles  $\xi_1$  and  $\xi_2$  are illustrated in Fig.1  
 228 for two typical cases:  $\{\varphi = \pi/6, k = 1.7\}$  and  $\{\varphi = \pi/2.7, k = 1.2\}$ . Fig.2  
 229 displays the evolution of  $\xi_1$  with increasing  $k$  values for different angles  $\varphi$ .



Figure 1: Examples of fiber damage states: if the normalized strain  $\tilde{\varepsilon}_f = \frac{\varepsilon_f}{\varepsilon_r}$  is greater than 1 (the red-hatched zones), the fibers break.

Figure 2: Examples of evolution of the damage variable  $\xi_1$  for different values of  $\varphi$

230 *Loading case of a constant-amplitude strain but rotating angle  $\varphi$ .* It is inter-  
 231 esting to treat the case of a constant-amplitude strain ( $k = cte$ ) separately  
 232 because it corresponds to an evolution of damage that is not monotonic. If  
 233  $k < \sqrt{2}$  and  $\varphi$  evolves between 0 and  $\pi/2$ , fiber fracture occurs near  $\xi = 0$   
 234 and  $\xi = \pi/2$ , but the fibers near  $\pi/4$  are unaffected (**Fig.3 illustrates the**  
 235 **evolution of the two angles when  $\varphi$  increases from 0 to  $\frac{\pi}{2}$** ). If  $k \geq \sqrt{2}$ , all the  
 236 fibers break at  $\varphi = \pi/4$ .

Figure 3: Evolution of the damage variables  $\xi_1$  and  $\xi_2$  as functions of  $\varphi$  in the case of a circular loading (an increasing  $\varphi$  with  $k < \sqrt{2}$  constant).  $\xi_1^*$  and  $\xi_2^*$  are the virtual damage variables due to the current loading without taking the loading history into account.

237 *Case of an arbitrary loading.* We now consider that both the characteris-  
 238 tic angle  $\varphi$  and the amplitude of the loading vary. This corresponds to the  
 239 general complex case. The solution of evolution of damage state can be pre-  
 240 dicted with an incremental approach. A special attention has to be paid to  
 241 the vicinity of singular point  $\{\sqrt{2}, \frac{\pi}{4}\}$  at which all the fibers break simulta-  
 242 neously. Therefore, it is necessary to describe the path of the loading point  
 243 precisely, especially in the vicinity of this point. Detailed explanation on the  
 244 numerical strategy can be found in Brunon (2011).

245 3.4. *Macroscopic evolution of the elasticity range: distortion*

The fracture surface at the macroscopic level evolves as damage increases. We denote  $A$  the set of healthy fibers angles. The fiber density function becomes:

$$\rho(\xi, \mathbf{D}) = \begin{cases} 0 & \text{if } \xi \notin A \\ 1 & \text{if } \xi \in A \end{cases} \quad (37)$$

246 The fiber density function can be observed for a particular case in Fig.4.

Figure 4: Shape of the density function for a particular damage case ( $k = 1.5, \varphi = \pi/10$  and  $\pi/3$ ).

The elasticity range becomes:

$$\mathcal{S} = \{\mathbf{E} / \forall \xi \in A, \mathbf{n}(\xi) \cdot \mathbf{E}(k, \varphi) \cdot \mathbf{n}(\xi) - \varepsilon_r < 0\} \quad (38)$$

247 The distortion of the surface is simply the change of the set  $A$ . More  
248 details for the model are described in Brunon (2011).

249 **4. Influence of the homogenization method on the shape of the**  
250 **stress strain curves**

251 This section compares the properties of the two homogenization methods  
252 when they are applied to the simple fiber breaking damage model defined in  
253 the preceding section. All the graphs of this part correspond to the biaxial  
254 loading described on Fig.7(a).

255 *4.1. Macroscopic structure tensor properties*

256 Let us observe some properties of the resulting macroscopic structure  
 257 tensor  $\mathbf{H}$ . We can immediately observe that  $H_{12} = H_{21} = 0$  because the  
 258 function  $\cos * \sin$  is odd. Indeed, since the integration intervals are always  
 259 symmetrical with respect to 0, these two components are always zero, even  
 260 when damage occurs. Besides, damage affects the components of  $\mathbf{H}$  directly.  
 261 The even nature of functions  $\cos^2$  and  $\sin^2$  leads to:

$$\mathbf{H} = \frac{2}{\pi} \int_0^{\frac{\pi}{2}} \rho(\xi, \mathbf{D}) \mathbf{n}(\xi) \otimes \mathbf{n}(\xi) d\xi \quad (39)$$

262 An example of the evolution of the nonzero components of  $\mathbf{H}$  for a loading  
 263 up to rupture and for a uniform angular distribution prior to damage, is given  
 in Fig.5.

Figure 5: An example of the evolution of the components of the structure tensor ( $\varepsilon_r = 0.5$ ,  
 $\varphi = \pi/10$ ).

264

265 *4.2. Homogenization methods comparison*

266 A plot of the fibers' contribution to the strain energy for each method  
 267 (Fig.6) shows that under the current assumptions of uniform angular distri-  
 268 bution prior to damage and brittle linear fibers, the two macroscopic models  
 269 behave differently when damage occurs. This difference can be observed by  
 270 expanding the expressions of these energies (Eq.40,41): it comes down to  
 271 the difference between the integral of a square and the square of an integral.  
 272 Indeed, for  $\xi_1$  and  $\xi_2$  constant (especially prior to damage), the ratio of  $\psi_f^K$

273 to  $\psi_f^E$  is constant throughout the loading and independent of the value of  
 274 the elastic parameter  $E$ .

$$\psi_f^E = \frac{E}{\pi} \int_{\xi_1}^{\xi_2} (\mathbf{M}(\xi) : \mathbf{E})^2 d\xi = \frac{E}{\pi} \int_{\xi_1}^{\xi_2} (E_1 \cos^2 \xi + E_2 \sin^2 \xi)^2 d\xi \quad (40)$$

$$\psi_f^K = \frac{E}{2} (\mathbf{H} : \mathbf{E})^2 = \frac{2E}{\pi^2} \left( \int_{\xi_1}^{\xi_2} (E_1 \cos^2 \xi + E_2 \sin^2 \xi) d\xi \right)^2 \quad (41)$$

Figure 6: Comparison of the macroscopic strain energies of the fibrous tissue for the two proposed homogenization methods.

275 Besides, the shape of the components of tensor PK2 (Fig.7, b) show that  
 276 the  $K$  method leads to the same value of the two nonzero components of  $\mathbf{S}_f^K$   
 277 prior to damage, whereas the tension applied to the tissue is not equibiaxial.  
 278 Conversely, with the  $E$  method, this unrealistic result is not obtained. **This**  
 279 **is probably an illustration of the possible non-physical results obtained when**  
 280 **the structure tensor theory is applied to a planar isotropic fiber distribu-**  
 281 **tion (Holzapfel et al, 2010). However for both homogenization methods, the**  
 282 **Cauchy stress components (Fig.7, c) are consistent with the components of**  
 283 **the strain tensor.** Finally, there is a clear difference between the two methods  
 284 concerning the concavity of the stress component corresponding to the least  
 285 solicited direction. The softening part of the constitutive relation is much  
 286 more anisotropic using the  $E$  method than using the  $K$  method.



$$E \text{ method: } \begin{cases} \phi_f = \frac{1}{2}E(1 - D(\mathbf{\Gamma}, \xi))\varepsilon_f^2 \\ \psi_f^E = \int_{A_0} \frac{1}{2}E(1 - D) (\mathbf{n} \cdot \mathbf{E} \cdot \mathbf{n})^2 d\xi \end{cases} \quad (42)$$

$$K \text{ method: } \begin{cases} \mathbf{H} = \frac{1}{\pi} \int_{A_0} \rho(\mathbf{\Gamma}, \xi) \mathbf{n} \otimes \mathbf{n} d\xi \\ \psi_f^K = \frac{1}{2}E (\mathbf{H} : \mathbf{E})^2 \end{cases} \quad (43)$$

303 In these expressions, **tensor**  $\mathbf{\Gamma}$  represents the value of the strain tensor  $\mathbf{E}$   
 304 which led to the current macroscopic damage state of the tissue. Since this  
 305 state is characterized by two independent variables  $\xi_1$  and  $\xi_2$ , we distinguish  
 306  ${}^1\mathbf{\Gamma}$  and  ${}^2\mathbf{\Gamma}$  which led to the current values of  $\xi_1$  and  $\xi_2$  respectively. The  
 307 piecewise linear evolution of the variable  $D$  or  $\rho$  is given within the range  
 308  $[0, \pi/4]$  (Eq.44). Within the range  $[\pi/4, \pi/2]$ , the expression is similar, but  
 309 involves  ${}^2\mathbf{\Gamma}$ .

$$D = \begin{cases} 1 & \text{if } \xi \leq \xi_1 \\ \frac{\varepsilon_f^*(\xi) - \varepsilon_D}{\varepsilon_r - \varepsilon_D} = \frac{{}^1\Gamma_1 \cos^2 \xi + {}^1\Gamma_2 \sin^2 \xi - \varepsilon_D}{\varepsilon_r - \varepsilon_D} & \text{if } \xi_1 \leq \xi \leq \xi'_1 \\ 0 & \text{if } \xi'_1 \leq \xi \end{cases} \quad (44)$$

$$\rho(\xi, {}^1\mathbf{\Gamma}) = 1 - D(\xi, {}^1\mathbf{\Gamma}) \forall \xi \quad (45)$$

310 The shape of the strain energy for each method is given in Fig.9. We  
 311 obtain a smoothed peak for the evolution of the Cauchy stress in the tis-  
 312 sue, corresponding to a more progressive macroscopic damage of the tissue  
 313 (Fig.10).

a)

b)

Figure 8: Constitutive law of a damageable fiber and evolution of the damage variable  $D$  and density variable  $\rho$  throughout the loading (a); damage state of the tissue's fibers (b).

Figure 9: Strain energy during biaxial tension loading for the  $K$  and  $E$  methods in the case of brittle fibers or damageable fibers.

### 314 5.2. Introduction of fiber uncrimping

315 In many applications (biological tissues, elastomers, etc.) the loaded  
 316 fibers uncrimp before being stretched. To take this phenomenon into account,  
 317 we adapt the method described in (Cacho et al., 2007) and introduce the  
 318 strain value  $\varepsilon_t$  beyond which the fiber starts becoming stretched (Fig.11).

319 The function  $t(\xi)$  is introduced to describe the stretched or unstretched  
 320 state of the fibers:  $t(\xi) = 0$  if  $\varepsilon_f < \varepsilon_t$ ,  $t(\xi) = 1$  otherwise. Then, the  
 321 description of the two macroscopic models is that of Eq.(46,47).

$$E \text{ method: } \begin{cases} \phi_f(\mathbf{E}, \xi) = \frac{1}{2} E t(\xi) (\varepsilon_f(\xi) - \varepsilon_t)^2 = \frac{1}{2} E t(\xi) (\mathbf{n}(\xi) \cdot \mathbf{E} \cdot \mathbf{n}(\xi) - \varepsilon_t)^2 \\ \psi_f^E = \frac{1}{\pi} \int_{A_0} \rho(\xi) \phi_f(\mathbf{E}, \xi) d\xi \end{cases} \quad (46)$$

Figure 10: Components of the Cauchy stress tensor of the tissue for the  $K$  and  $E$  methods in the case of brittle fibers or damageable fibers, as functions of the corresponding components of  $\mathbf{E}$ .

Figure 11: Behavior of a fiber taking uncrimping into account.

$$K \text{ method: } \begin{cases} \mathbf{H} = \frac{1}{\pi} \int_{A_0} \rho(\xi) t(\xi) \mathbf{n} \otimes \mathbf{n} d\xi \\ \psi_f^K = \frac{1}{2} E (\mathbf{H} : \mathbf{E})^2 \end{cases} \quad (47)$$

322 Fig.12 shows the evolution of the components of the stress tensor: at the  
 323 beginning of the loading, they are null since no fiber is stretched. The strain  
 324 in the fibers gradually increases to  $\varepsilon_t$  and the components of  $\mathbf{H}$  increase to  
 325 their maximum values. Then, rupture including the uncrimping phenomenon  
 326 in our model leads to an unrealistic shape of the strain energy. Indeed, when  
 327 the fibers uncrimp, the strain energy increases because both the number of  
 328 stretched fibers and the amplitude of the loading increase. Then, once all the  
 329 fibers have uncrimped, the energy continues to increase only because of the  
 330 loading. Thus, the slope of the energy curve is more gentle once all the fibers  
 331 are stretched than when some are still crimped. When the fibers uncrimp, the  
 332 global behavior of the tissue actually "catches up" with the behavior without  
 333 taking uncrimping into account, leading to steeper slopes of the energy and  
 334 stress curves. This is a limitation of the model: if all the fibers uncrimp  
 335 before damage begins, it leads to an energy trend which is never observed  
 336 experimentally. In order to obtain the "toe region" which is often observed  
 337 experimentally and which is attributed to the uncrimping of the fibers, only  
 338 a fraction of the fibers must be stretched when damage begins; this could be  
 339 obtained using a random distribution of the fibers' crimping, which is not in  
 340 the scope of this paper as it requires a two-scale computation.



Figure 12: Evolution of the structure tensor, the strain energy and the components of the Cauchy tensor under biaxial tension for initially crimped fibers ( $\varepsilon_t = 0.15$ ,  $\varepsilon_r = 0.5$ ). The box emphasizes the area where the obtained behavior is not realistic.

## 341 6. Conclusions

342 The objective of this work was to compare the response of two homoge-  
343 nization methods, available in the literature, in the case of damaging fibers  
344 within a fibrous membrane. For that purpose we propose a very simple  
345 anisotropic damage model able to account for several phenomena observed  
346 experimentally.

347 The evolution of macroscopic damage based on fiber fracture at the mi-  
348 croscopic scale is described using two scalar variables. The explicit expression  
349 of these variables as functions of the material parameters and of the macro-  
350 scopic strain loading enables a rapid implementation of the fracture behavior  
351 law of the tissue. Besides, this paper does not deal with any phenomenon  
352 which would require reverting to the microscopic scale.

353 The main limitation of this model is that it considers the tissue's damage  
354 to be borne by the fibers alone independently of the components of the tissue  
355 (namely the matrix) with the assumption that the fibers are isolated from  
356 one another. This leads to a drastic nature of the constitutive law, that could  
357 be tone down by introducing fiber interactions, a distribution of the fibers  
358 properties or a possible decohesion between the fibers and the matrix.

359 The implementation of the two homogenization methods enabled us to

360 compare them: in the case of an isotropic tissue, the difference between the  
361 two methods appears during damage and is due to the different formulation of  
362 the tissue's macroscopic strain energy. The mathematical expression of this  
363 difference makes us believe that a heterogeneous angular distribution of the  
364 fibers (for an initially anisotropic tissue) would reveal a disparity between the  
365 two methods right from the elastic zone. Another fundamental difference is  
366 that the  $K$  method cannot take into account the behavior on the fibers' scale  
367 because the constitutive law is applied directly to the macroscopic tissue.

368 Depending on the homogenization method, the principle of the identifi-  
369 cation of the damage model is different: indeed, the  $E$  method based on the  
370 strain energy of a fiber requires knowing the behavior and fracture strain  
371 of a fiber; these parameters can be determined independently of the fibrous  
372 tissue (see, for example, (Sasaki and Odajima, 1996; Svensson et al., 2010)  
373 for collagen fibers). Nevertheless, while the response of the fibrous tissue can  
374 be determined *a priori*, the response of the matrix is still to be identified.  
375 In the case of the  $K$  method, the constitutive law is applied to the tissue  
376 rather than to the fibers; consequently, the identification of the macroscopic  
377 parameters of the tissue is required both for the fibrous tissue and for the ma-  
378 trix; only the fracture strain of the fibers can be determined experimentally  
379 *a priori*. In practice, the identification of material parameters associated  
380 with damage requires being able to control the evolution of damage so that  
381 rupture does not to occur too rapidly. This constitutes the main difficulty of  
382 the experimental characterization of damage.

383 The model proposed in this study can be applied to initially isotropic  
384 membrane tissues. Therefore, the prospective applications of this study con-

cern the identification of the model's parameters in practical cases. In the  
 case of biological tissues, the fracture modeling of the liver capsule could en-  
 hance a model of the human body and improve the prediction of liver injuries  
 during an impact.

### Appendix A: Details about the description of damage

The objective of this development is to define, for a radial loading with  
 an amplitude greater than the bounds defined in Eq.(33), the angular sectors  
 in which the fibers break. This corresponds to the resolution of  $\varepsilon_f - \varepsilon_r > 0$ .  
 In the general case, since  $\sin^2\xi = 1 - \cos^2\xi$ :

$$\cos\varphi \cos^2\xi + \sin\varphi (1 - \cos^2\xi) > \frac{1}{k} \quad (48)$$

$$\cos^2\xi (\cos\varphi - \sin\varphi) > \frac{1 - k \sin\varphi}{k} \quad (49)$$

Two cases need to be distinguished:

$$\varphi \in \left[0, \frac{\pi}{4}\right] \Rightarrow \cos\varphi - \sin\varphi \geq 0 \Rightarrow \cos^2\xi > \frac{1 - k \sin\varphi}{k(\cos\varphi - \sin\varphi)} \quad (50)$$

$$\varphi \in \left[\frac{\pi}{4}, \frac{\pi}{2}\right] \Rightarrow \cos\varphi - \sin\varphi \leq 0 \Rightarrow \cos^2\xi < \frac{1 - k \sin\varphi}{k(\cos\varphi - \sin\varphi)} \quad (51)$$

In the following, only the case  $\varphi \in \left[0, \frac{\pi}{4}\right]$  is explained.

These equations hold provided that the right-hand side is positive, *i.e.*:

$$1 - k \sin\varphi \geq 0 \Leftrightarrow k \leq \frac{1}{\sin\varphi} \quad (52)$$

Therefore, one can write:

$$\cos\xi > \sqrt{\frac{1 - k \sin\varphi}{k(\cos\varphi - \sin\varphi)}} \quad \forall k \in \left[\frac{1}{\cos\varphi}, \frac{1}{\sin\varphi}\right] \quad (53)$$

395 This expression is valid if the second term (which is positive) is between  
 396 0 and 1, which is verified for  $k \geq \frac{1}{\cos \varphi}$ .

397 Finally, the damage in the tissue can be described as follows:

$$\forall k \in \left[ \frac{1}{\cos \varphi}, \frac{1}{\sin \varphi} \right] : \cos \xi \geq \sqrt{\frac{1 - k \sin \varphi}{k (\cos \varphi - \sin \varphi)}} \quad (54)$$

398 For  $\xi \in A$ , the solutions of inequality  $\varepsilon_f(\xi) - \varepsilon_r > 0$  are:

$$\varphi \in \left[ 0, \frac{\pi}{4} \right] : \begin{cases} \xi \leq \arccos \sqrt{\frac{1 - k \sin \varphi}{k (\cos \varphi - \sin \varphi)}} \\ ou \\ \xi \geq -\arccos \sqrt{\frac{1 - k \sin \varphi}{k (\cos \varphi - \sin \varphi)}} \end{cases} \quad (55)$$

$$\varphi \in \left[ \frac{\pi}{4}, \frac{\pi}{2} \right] : \begin{cases} \xi \geq \arccos \sqrt{\frac{1 - k \sin \varphi}{k (\cos \varphi - \sin \varphi)}} \\ ou \\ \xi \leq -\arccos \sqrt{\frac{1 - k \sin \varphi}{k (\cos \varphi - \sin \varphi)}} \end{cases} \quad (56)$$

399 This leads to the definition of the two angles  $\xi_1$  and  $\xi_2$  described in the  
 400 paper.

**Acknowledgement 1.** *The authors would like to thank the Region Rhône-Alpes for its financial support.*

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