

Constitutive Modeling of Damage Mechanisms in Arterial Walls and Related Experimental Studies

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Abstract

In this paper we present a continuum damage model for collagenous soft tissues such as arterial walls, wherein damage evolution is governed by statistical distributions of microscopic quantities. The damage model is an extension to the constitutive framework published in [1] and includes specific damage functions that arise from considerations related to the collagen fiber's microstructure. In detail, statistical distributions of proteoglycan orientations, fibril length parameters and ultimate proteoglycan stretch are taken into account. The damage evolution resulting from each different considered statistical distribution is compared with respect to its capability of capturing uniaxial experimental data of a human carotid artery in the supra-physiological loading domain.

1 Introduction

During clinical interventions soft collagenous tissues such as arterial walls are often subjected to supra-physiological states, where microscopic damage is induced. Thus, there is an arising interest of understanding the mechanics of damaged tissue regions. Usually, formulations within the framework of continuum damage mechanics are derived, that make use of the concept of internal variables, cf. SIMO [17]. These models have been initially developed to describe the softening behavior in elastomers, and were then extended to biological fibrous tissues. In addition, phenomenological approaches according to the concept of pseudo-elasticity, as introduced by OGDEN & ROXBURG [12], have been applied to collagenous soft tissues, see, e.g., WEISBECKER *et al.* [18]. For the modeling of anisotropic damage using scalar-valued variables instead of damage tensors a practical approach is given in SCHRÖDER *et al.* [14]. Remanent strains after overstretch are described in GASSER & HOLZAPFEL [9] within a finite plasticity framework assuming remaining deformations in the fibers. EHRET & ITSKOV [7] also consider remanent deformations of the fibers based on continuum damage mechanics. In BALZANI *et al.* [1] a construction principle for damage models is given that can describe remanent strains after unloading and which ensures polyconvexity in the undamaged (physiological) regime. Damage in the fiber direction and in the ground-matrix material is, for instance, considered in NATALI *et al.* [11] and CALVO *et al.* [4].

To some extent the models mentioned above allow a micromechanical interpretation. However, damage is still described by phenomenological functions. As a micromechanical approach RODRÍGUEZ *et al.* [13] propose a model that considers a subsequent rupture of individual fibers according to a stochastic distribution of their waviness to be the main driver of damage evolution. GASSER [8] introduced a phenomenological damage model that is even motivated on a lower length scale, namely on the interfibrillar level, which will be

referred to as the microscopic level in this paper. The latter damage formulation takes into account a sliding filament model (see SCOTT [16]), which states that the interconnecting proteoglycans (PGs) on the fibril level can store reversible displacements only in a certain domain. Therefore, remanent sliding of PGs, when exceeding a sustainable PG stretch, is expected to be the main contributor to remanent sliding observed on a macrosopic level. In this contribution, we assume statistical distributions of microscopic parameters to be responsible for damage evolution. Therefore, different damage functions are derived that are associated to the regime of failed PGs and embedded in the constitutive framework [1]. The different damage functions that are obtained by assuming different distributions of microscopic parameters are combined with several undamaged (effective) transversely-isotropic functions for the physiological regime, and a parameter adjustment to experimental data is performed.

2 Framework for Damage Modeling

Consider a continuum body $\mathcal{B} \subset \mathbb{R}^3$ in the undeformed reference configuration. A motion function $\varphi_t : \mathcal{B} \rightarrow \mathcal{S}$ at time $t \in \mathbb{R}_+$ maps this configuration to the current configuration \mathcal{S} . Thereby, a point $\mathbf{X} \in \mathcal{B}$ is transformed to a point $\mathbf{x} \in \mathcal{S}$. The deformation gradient \mathbf{F} and the right Cauchy–Green deformation tensor \mathbf{C} are defined as $\mathbf{F}(\mathbf{X}) := \nabla \varphi_t(\mathbf{X})$ and $\mathbf{C} := \mathbf{F}^T \mathbf{F}$, with the Jacobian $J := \det \mathbf{F} > 0$. We postulate the existence of a strain-energy function Ψ , defined per unit reference volume. Due to the directional dependence on the deformation, we define the function Ψ to be dependent on the right Cauchy–Green tensor \mathbf{C} as well as on structural tensors $\mathbf{M}_{(a)} := \mathbf{A}_{(a)} \otimes \mathbf{A}_{(a)}$ with $\mathbf{A}_{(a)}$ denoting the orientation vector of fiber family a . Specifically, we consider the following decoupled form of the strain-energy function

$$\Psi(\mathbf{C}, \mathbf{M}_{(1)}, \mathbf{M}_{(2)}) = \Psi^{\text{pen}}(\det \mathbf{C}) + \Psi^{\text{iso}}(\mathbf{C}) + \sum_{a=1}^2 \Psi_{(a)}^{\text{ti}}(\mathbf{C}, \mathbf{M}_{(a)}), \quad (1)$$

where Ψ^{pen} denotes a penalty term accounting for the incompressibility constraint, Ψ^{iso} denotes the contribution of an isotropic ground-matrix material, and $\Psi_{(a)}^{\text{ti}}$ denotes the transversely isotropic contribution of a superimposed fiber family. For a coordinate-invariant representation the invariants $I_1 := \text{tr} \mathbf{C}$, $I_2 := \text{tr}[\text{Cof} \mathbf{C}]$, and $I_3 := \det \mathbf{C}$ of the right Cauchy–Green tensor \mathbf{C} as well as the mixed invariants

$$J_4^{(a)} := \text{tr}[\mathbf{C} \mathbf{M}_{(a)}], \quad J_5^{(a)} := \text{tr}[\mathbf{C}^2 \mathbf{M}_{(a)}], \quad (2)$$

which characterize the constitutive response of the fibers, have to be taken into account. Alternative polyconvex functions, as introduced by SCHRÖDER & NEFF [15], i.e.

$$\begin{aligned} K_1^{(a)} &:= \text{tr}[\text{Cof} \mathbf{C} \mathbf{M}_{(a)}] = J_5^{(a)} - I_1 J_4^{(a)} + I_2, \\ K_2^{(a)} &:= \text{tr}[\mathbf{C}(\mathbf{I} - \mathbf{M}_{(a)})] = I_1 - J_4^{(a)}, \\ K_3^{(a)} &:= \text{tr}[\text{Cof} \mathbf{C}(\mathbf{I} - \mathbf{M}_{(a)})] = I_1 J_4^{(a)} - J_5^{(a)} \end{aligned} \quad (3)$$

can be used. To introduce a damage mechanism, we incorporate the $(1 - D)$ -approach into the transversely isotropic parts of the strain-energy function, as in BALZANI *et al.* [1]. Thus,

$$\Psi_{(a)}^{\text{ti}} := m(P_{(a)}(\mathbf{C}, D_{(a)})) \quad \text{with} \quad P_{(a)} = (1 - D_{(a)}) \Psi_{(a)}^{\text{ti},0} - c, \quad (4)$$

where m is a monotonically increasing convex function, $D_{(a)} \in [0, 1]$ denotes the damage variable in the direction of the fiber family a , $\Psi^{\text{ti},0}$ denotes an undamaged (effective) transversely isotropic function, and c equals the amount of the latter function in the reference configuration.

Based on the Clausius-Duhem inequality for isothermal conditions the second Piola-Kirchhoff stress \mathbf{S} can be derived as

$$\mathbf{S} = 2 \frac{\partial \Psi}{\partial \mathbf{C}} = \mathbf{S}^{\text{pen}} + \mathbf{S}^{\text{iso}} + \sum_{a=1}^2 \mathbf{S}_{(a)}^{\text{ti}}, \quad \mathbf{S}^{\text{pen}} = 2 \frac{\partial \Psi^{\text{pen}}}{\partial \mathbf{C}}, \quad \mathbf{S}^{\text{iso}} = 2 \frac{\partial \Psi^{\text{iso}}}{\partial \mathbf{C}}, \quad (5)$$

and the transversely isotropic part

$$\mathbf{S}_{(a)}^{\text{ti}} = (1 - D_{(a)}) \mathbf{S}_{(a)}^{\text{ti},0}, \quad \mathbf{S}_{(a)}^{\text{ti},0} = 2m' \frac{\partial \Psi_{(a)}^{\text{ti},0}}{\partial \mathbf{C}}. \quad (6)$$

Herein, the abbreviation $m' := \partial_P m$ has been introduced. The physical Cauchy stresses can be computed as $\sigma = J^{-1} \mathbf{F} \mathbf{S} \mathbf{F}^T$.

3 Micromechanically-Based Damage Function

A micromechanically-based stretch-induced damage function is here derived and embedded in the constitutive framework proposed by BALZANI *et al.* [1], where a phenomenological function for D is used. The evolution of damage is assumed to be associated to a subsequent loss of interfibrillar proteoglycan (PG) bridges, cf. GASSER [8], induced by statistical distributions of microscopic quantities. This approach is based on a sliding filament model [16], which states that PG bridges store reversible displacements only in a certain domain. This results from their composition of two neighboring aonic glycosaminoglycan (AGAG) chains, which can bond differently and, therefore, enable sliding of the collagen fibrils when subjected to stress.

We consider the structure shown in Fig. 1 as a simplified fibril microstructure that is composed of two half collagen fibrils and interconnecting PG bridges. The orientation of an individual PG bridge is given by an initial PG angle α . The initial distance between collagen fibrils, their length and initial overlap are denoted by d_0 , L_{cf} and L_{ov} , respectively. A macroscopic fiber stretch λ_{fib} yields an extension of the fibrils and of the PG bridges. Since incompressibility of the tissue is assumed the stretch in the direction transverse to the fiber orientation can be computed by $1/\sqrt{\lambda_{\text{fib}}}$ and thus the distance of collagen fibrils in the deformed configuration is

$$d = \frac{d_0}{\sqrt{\lambda_{\text{fib}}}}. \quad (7)$$

Note that the fiber stretch can be expressed in terms of the fourth invariant from eq. (2) as $\lambda_{\text{fib}} = \sqrt{J_4}$. The resulting relative displacement u of collagen fibrils is given by $u = (\lambda_{\text{fib}} - 1)(L_{cf} - L_{ov})$.

The stretch of a PG bridge, say λ_{pg} , is evaluated by means of standard trigonometric arguments as the ratio of lengths in the undeformed ($L_{\text{pg},0}$) and the deformed (L_{pg}) setting, i.e.

$$\lambda_{\text{pg}} = \frac{L_{\text{pg}}}{L_{\text{pg},0}} = \sqrt{[\cos \alpha - L(\lambda_{\text{fib}} - 1) \sin \alpha]^2 + \frac{\sin^2 \alpha}{\lambda_{\text{fib}}}}, \quad (8)$$

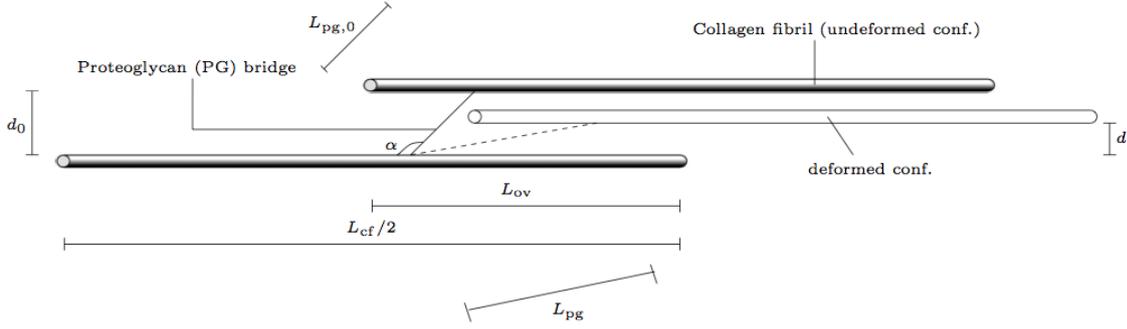


Figure 1: Definition of geometric quantities defining the fibril-proteoglycan microstructure.

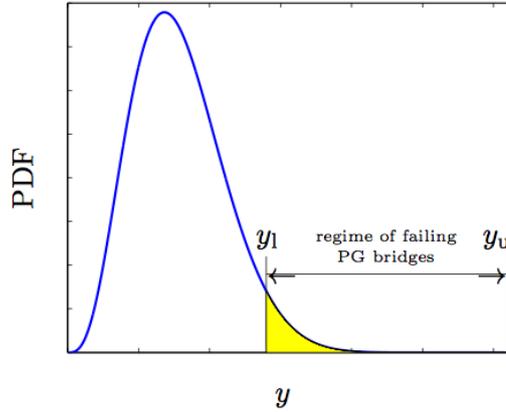


Figure 2: Evaluation of the damage variable as the integral of the associated probability density function (PDF) over y_l and y_u .

wherein $L = (L_{cf} - L_{ov})/d_0$ represents a dimensionless internal length parameter. Motivated by the statement in SCOTT [16] that PG stretch is only reversible as long as the overlap between two neighboring AGAG chains is above a critical value, the parameter λ_{pg}^{sust} is introduced, which describes the maximum sustainable PG stretch. For the microstructural properties α and L , as well as for the ultimate PG stretch λ_{pg}^{sust} statistical distributions are assumed in the sequel. A regime of failed PG bridges is evaluated for which $\lambda_{pg} - \lambda_{pg}^{sust} \geq 0$ holds. Depending on the random microscopic quantity y for which a statistical distribution is assumed either the lower regime bound y_l , the upper regime bound y_u , or both are solutions of

$$\lambda_{pg} - \lambda_{pg}^{sust} = 0. \quad (9)$$

The damage variable D is defined as the fraction of failed PG bridges. According to this definition D can be evaluated by integration of the respective probability density function (PDF) of y over y_l and y_u

$$D(\lambda_{fib}) = \int_{y_l(\lambda_{fib})}^{y_u(\lambda_{fib})} \vartheta(y) dy = \hat{\vartheta}(y_u) - \hat{\vartheta}(y_l) \quad \text{with} \quad \int_y \vartheta(y) dy = 1. \quad (10)$$

Herein, $\hat{\vartheta}$ denotes the cumulative distribution function (CDF). Due to the above definition $0 \leq D \leq 1$ is satisfied. In the sequel, Gaussian as well as beta distributions are

considered yielding the CDFs

$$\hat{\vartheta}_{\text{Gauss}}(y) = \frac{1}{2} + \frac{1}{\sqrt{\pi}} \int_0^{\frac{y-\mu}{\sqrt{2}\sigma_D}} e^{-\tilde{y}^2} d\tilde{y}, \quad \text{and} \quad (11)$$

$$\hat{\vartheta}_{\text{beta}}(y) = \frac{1}{B(a, b)} \int_0^y \tilde{y}^{a-1} (1 - \tilde{y})^{b-1} d\tilde{y}, \quad \text{with } 0 < y < 1. \quad (12)$$

The mean value and standard deviation of the Gaussian distribution are denoted by μ and σ_D , respectively. The shape parameters of the beta distribution are denoted by a and b , and the beta function is denoted by B .

4 Comparison with Experiments

In this section the damage model presented above is incorporated into different strain-energy functions and the resulting stress response is adjusted to uniaxial cyclic tension tests of the media of human carotid artery specimens, cf. [1]. Specifically, in this contribution, the following function is considered to represent the fiber response

$$\Psi_{(\text{BNSH}_{1,e})}^{\text{ti}} = \frac{k_1}{2k_2} \left\{ \exp \left[k_2 \left\langle (1 - D_{(a)}) K_3^{(a)} - 2 \right\rangle^2 \right] - 1 \right\}, \quad (13)$$

as introduced in BALZANI *et al.* [2]. Within the latter strain energy the damage variable $D_{(a)}$ is computed according to (10), whereby three different cases of statistically distributed microscopic quantities are considered. The initial PG angle $\alpha \in]0, \pi[$ as well as the ultimate PG stretch $\lambda_{\text{pg}}^{\text{sust}} \in]1, 2[$ are assumed to follow a beta distribution, because the respective distribution bounds are well-defined. Furthermore, a Gaussian distributed internal length parameter $L \in]0, +\infty[$ is considered, since for this quantity the domain size is not known. Subsequently, the complete strain-energy function is denoted as $\Psi_{(\text{BNSH}_{1,e})}$. Therein, the material restrictions $k_1 > 0$, $k_2 > 0$ have to hold in order to guarantee polyconvexity in the undamaged regime. Hereby, k_1 is a stress-like parameter, whereas k_2 is dimensionless.

There is only rare information provided in the literature that allows for conclusions about the values of the microscopic parameters. Some specific information about PG orientation can be drawn from microscopic images as, for example, given in DELL'ORBO *et al.* [5] or DINGEMANS *et al.* [6]. Furthermore, only very little is known about the geometric properties of the collagen fibrils, see, e.g., GRAHAM *et al.* [10] or BIRK & ZYCBAND [3], as well as about the ultimate PG stretch. Hence, the microscopic parameters are also fitted to experimental data by the adjustment routine. They are contained within the vector of material parameters $\boldsymbol{\alpha}$ that enters the least-square function

$$\bar{r}(\boldsymbol{\alpha}) = \sum_{k=1}^{n_{\text{exp}}} \sqrt{\frac{1}{n_{\text{mp}}} \sum_{i=1}^{n_{\text{cyc}}} \sum_{j=1}^{n_{\text{mp},i}} r(\boldsymbol{\alpha})}, \quad r(\boldsymbol{\alpha}) = \left(\frac{\sigma^{\text{exp}}(\lambda^{(i,j)}) - \sigma^{\text{comp}}(\lambda^{(i,j)}, \boldsymbol{\alpha})}{\max_i(\sigma^{\text{exp}})} \right)^2, \quad (14)$$

wherein λ represents the stretch in the tension direction of the uniaxial tension tests. The overall error measure \bar{r} is minimized with respect to $\boldsymbol{\alpha}$. The vector $\boldsymbol{\alpha}$ includes the introduced parameters of the strain-energy function and the damage model, but also the fiber angle β_f , which describes the angle between the fiber orientation and the circumferential direction of the artery. The material parameters are adjusted to a cyclic uniaxial tension test in circumferential and in axial direction of the artery, thus $n_{\text{exp}} = 2$. A number of n_{cyc} cycles with $n_{\text{mp},i}$ measuring points in cycle i is considered. The maximum experimental

| Statistical Distribution | c_1 [kPa] | k_1 [kPa] | k_2 [-] | β_f [°] | α [-] | L [-] | λ_{pg}^{sust} [-] | $a \mu$ [-] | $b \sigma_D$ [-] | \bar{r} [-] |
|------------------------------|----------------|----------------|--------------|------------------|-----------------|------------|------------------------------|----------------|---------------------|------------------|
| α (beta) | 9.6 | 894.4 | 87.0 | 39.4 | – | 3.8 | 1.016 | 9.8 | 18.95 | 0.067 |
| L (Gauss) | 11.8 | 541.9 | 30.2 | 39.1 | 0.9 | – | 1.016 | 9.3 | 1.67 | 0.177 |
| λ_{pg}^{sust} (beta) | 13.1 | 541.3 | 115.9 | 39.2 | 2.4 | 3.6 | – | 1.7 | 0.96 | 0.135 |

Table 1: Material parameters and error measure \bar{r} of the model $\Psi_{(BNSH_{1,e})}$ for the media of a human carotid artery under consideration of different statistical distributions of microscopic quantities.

stress $\max(\sigma^{\text{exp}}) \neq 0$ of the corresponding cycle is used as a normalization factor for the difference between the experimental stress σ^{exp} and the computed stress σ^{comp} according to the constitutive model. A number of $n_{\text{cyc}} = 18$ cycles in axial direction and $n_{\text{cyc}} = 27$ in circumferential direction is taken into account. The overall number of measuring points is $n_{\text{mp}} = 1271$ for the axial and $n_{\text{mp}} = 1793$ for the circumferential direction. Within the optimization routine an iterative procedure adjusts for the uniaxial tension conditions under the assumption of incompressibility. Thus, the penalty term in (1) does not apply here. In Fig. 3 the (a) experimentally observed stress response as well as the response of the model $\Psi_{(BNSH_{1,e})}$ under assumption of (b) a beta distributed PG angle, (c) a Gaussian distributed internal length parameter, and (d) a beta distributed ultimate PG stretch is depicted, respectively. The associated material parameters are given in Table 1. As it turns out, good correlations with the experimental data are obtained with beta distributed PG angles and ultimate PG stretches, respectively, whereas no adequate reproduction of the experiment is found under the assumption of a Gaussian distributed internal length parameter. In the latter case no pronounced softening hystereses is induced since the occurring damage is too small. In the cases of statistically distributed PG angles and ultimate PG stretches qualitatively and quantitatively comparable softening hysteresis to the one in the experiment are obtained, whereas an underlying distribution of the PG angle yields slightly better results.

5 Conclusion

In this contribution a statistical approach for the modeling of microscopic damage in soft collagenous tissues was presented. Statistical distributions of proteoglycan orientation, ultimate proteoglycan stretch and an internal length parameter of the collagen fibrils were investigated in terms of the capability to be responsible for the softening hysteresis as, for example, observed when subjecting arterial walls to cyclic supra-physiological loading. To account for statistically distributed microscopic quantities a new damage function was derived and embedded in the constitutive framework proposed in [1]. The obtained constitutive model was adjusted to cyclic experimental data of human carotid artery specimens by means of a least square fit, cf. [1]. Due to the adjustment results we may conclude that a distributed PG angle may have the largest influence on the softening hysteresis as observed in experiments among all investigated microscopic quantities.

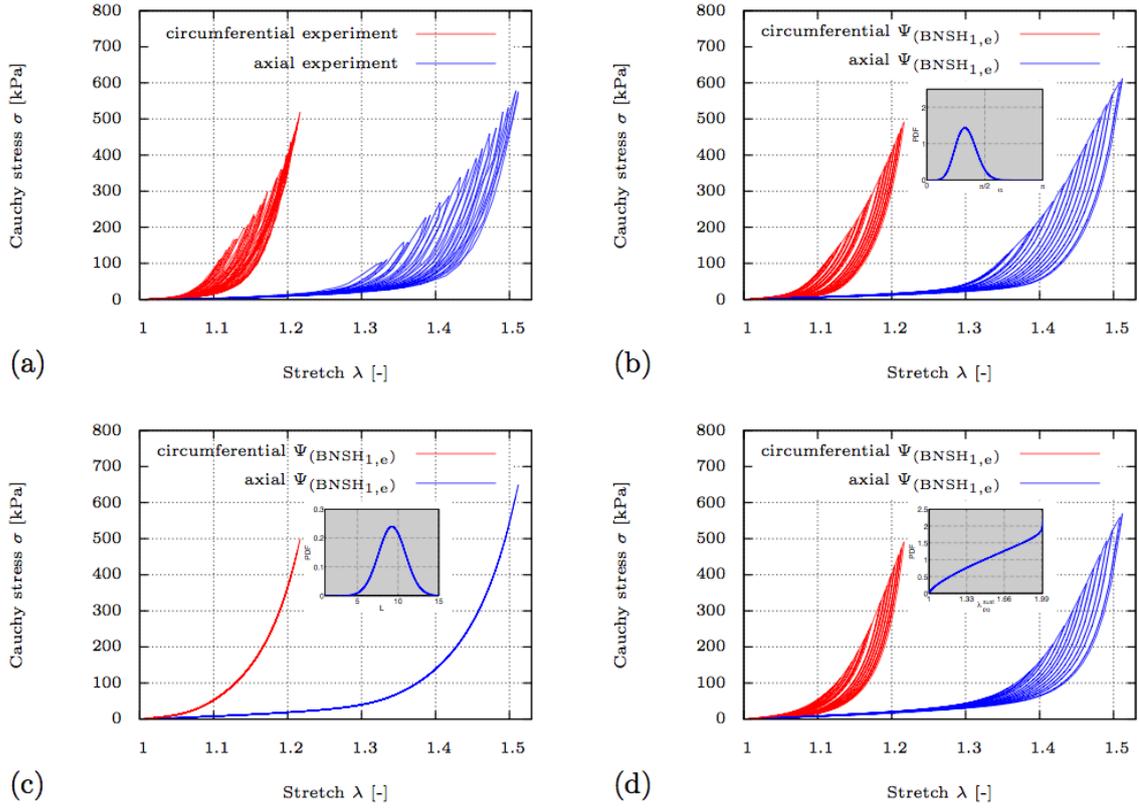


Figure 3: Cyclic uniaxial tension tests of the media of human carotid artery specimens in circumferential and axial directions. The stress-strain responses are depicted for (a) the experiment and for the model $\Psi_{(BNSH_{1,e})}$ under consideration of (b) a beta distributed PG angle, (c) a Gaussian distributed internal length, and (d) a beta distributed ultimate PG stretch, respectively. The associated material parameters and error measure are given in Table 1.

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