The Mechanical Behaviour of Continuous Media with stochastic damage

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THE MECHANICAL BEHAVIOUR OF CONTINUOUS MEDIA WITH STOCHASTIC DAMAGE

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Abstract—A model to describe the damage state of materials is developed within the framework of Continuum Damage Mechanics. The damage variable is considered as a random variable. To predict the damage state in a body, knowledge of its intrinsic damage distribution is required. After dividing the body into a number of elementary cells, the intrinsic damage distribution can be determined utilizing statistics of extremes. Fitting this distribution to experimental data the best-fit model parameters in the intrinsic damage distribution are obtained. The model is applied to fatigue damage evolution in brittle elastic materials.

1. INTRODUCTION

DURING the last 20 years the development of heart valve prostheses has been an active field of research. At the present basically two different types of heart valve prostheses can be distinguished. Firstly the mechanical valves, in which the blood flow is regulated by a tilting disc. Secondly the leaflet valve prostheses, which consist of three flexible leaflets that are attached to a frame. The leaflet materials are biological tissue structures or synthetic materials. Leaflet valve prostheses have the advantage of a pseudo-physiological, central flow and a reduced incidence of thromboembolic complications. However, compared to mechanical valves, the durability of the leaflet valves gives rise to concern. Due to an unfavourable design in comparison with the natural valves, large repetitive loadings in combination with calcium deposition locally initiate microcracks, which may grow to form a macrocrack leading to complete leaflet rupture[1]. The objective of our research is directed towards the development of an artificial heart valve with fibre reinforced leaflets[2]. To increase the durability of the leaflets knowledge of the failure mechanisms is required.

A model for predicting the effect of microstructural damage on the constitutive behaviour is presented in this paper. The model is based on Continuum Damage Mechanics (CDM), which is a branch of continuum mechanics characterized by the introduction of an internal field variable, representing in an appropriate sense the distribution of microcracks locally[3]. The constitutive theory can be developed rigorously on the basis of thermodynamics with internal state variables[4].

Damage evolution is mostly treated as a deterministic process[3, 5–7]. However, an important feature of damage is its probabilistic nature. For example in case of fatigue loading different lifetimes will result for identical bodies under identical loading conditions. An attempt to connect CDM and the statistical strength theories was made by Krajcinovic and Silva[8]. Their work focused on the establishment of simple statistical models for the uni-axial response of gradually damaging structures.

In this paper a model is derived, which considers the damage state at a material point in a body as a stochastic variable. Since the model must be confronted with experimental data, a physical meaning will be attributed to the damage variable. Therefore, a relationship is proposed between the damage variable and an effective defect size. To predict the local damage state in a body, its intrinsic damage distribution is required. In order to compute this distribution the body is divided into a number of cells. Utilizing statistics of extremes[9], the proposed distribution of the damage in a cell is transformed to the distribution of the maximum defect size in the body. The cell dimensions as well as the parameters in the intrinsic damage distribution are unknown in advance. By fitting the theoretical distribution of the maximum defect sizes to an experimental distribution the best-fit model parameters can be computed.

The model can be used for computing the damage state of a loaded structure if the damage evolution law and the intrinsic damage distribution are known. If the intrinsic damage distribution and the lifetime distribution are available, the theory can be applied for computing the damage

evolution law in high cycle fatigue. Moreover, the scale at which micrographic damage measurements have to be done is obtained naturally from a minimization problem.

The model is applied to fatigue damage evolution in brittle elastic materials. Utilizing experimental data from [10] the fatigue lifetime distribution of an elastomeric biomaterial is derived. The theoretical lifetime distribution is compared with fatigue experiments and the results will be discussed.

The damage state in three-dimensional structures can be described by the model. However, if the damage and elastic mechanisms are coupled, implementation of the model in a finite element code may lead to excessive computing times for large structures with geometrically and physically nonlinear behaviour. Choosing a special form of the stress relation, the elastic and inelastic mechanisms are uncoupled. The results of this choice are compared with the results of the coupled equations and with experiments.

2. BASIC EQUATIONS AND CONSTITUTIVE THEORY

Under assumptions of sufficient smoothness the balance laws can be written as differential field equations[4]. The balance of linear momentum neglecting inertial effects and body forces is given by

$$\nabla \cdot \boldsymbol{\sigma} = \mathbf{0} \quad (1)$$

where ∇ is the gradient operator with respect to the deformed configuration and $\boldsymbol{\sigma}$ is the Cauchy stress tensor. The law of balance of angular momentum is expressed by the symmetry of the Cauchy stress tensor

$$\boldsymbol{\sigma} = \boldsymbol{\sigma}^c. \quad (2)$$

The condition for balance of energy is

$$\rho \dot{\epsilon} = \rho r + \boldsymbol{\sigma} : \mathbf{D} + \nabla \cdot \mathbf{q} \quad (3)$$

where $\dot{\cdot}$ is the material time derivative, ρ is the density, ϵ is the specific internal energy, r is the extrinsic heat supply, $\mathbf{D}$ is the rate of strain tensor and $\mathbf{q}$ is the heat flux vector. In addition to these equations it is required that the second law of thermodynamics

$$-\rho \dot{\psi} - \rho \eta \dot{\theta} + \boldsymbol{\sigma} : \mathbf{D} - (1/\theta) \mathbf{q} \cdot \nabla \theta \geq 0 \quad (4)$$

is satisfied, where θ is the absolute temperature, η is the specific entropy and ψ is the specific Helmholtz free energy defined as $\psi = \epsilon - \eta \theta$.

In the following we restrict ourselves to isothermal conditions. Let the state variables be given by the deformation gradient $\mathbf{F} = (\nabla_0 \mathbf{x})^c$, where $\mathbf{x}(\mathbf{x}_0, t)$ is the position vector and ∇_0 is the gradient operator with respect to the undeformed configuration, and by a set of internal state variables $\boldsymbol{\beta}$, representing dissipative mechanisms. The material is characterized by response functions specifying the dependence of the Cauchy stress tensor and the rate of damage growth on the state variables. In accordance with the principle of equipresence we demand that each response function is a function of all state variables. Considerations of the principle of objectivity of material properties and employment of the second law of thermodynamics yield

$$\mathbf{P} = \rho_0 \frac{\partial \psi(\mathbf{E}, \boldsymbol{\beta})}{\partial \mathbf{E}} \quad (5)$$

$$\dot{\boldsymbol{\beta}} = \mathbf{f}(\mathbf{E}, \boldsymbol{\beta}) \quad (6)$$

$$\frac{\partial \psi(\mathbf{E}, \boldsymbol{\beta})}{\partial \beta_i} \beta_i < 0 \quad (7)$$

where

$$\mathbf{P} = J \mathbf{F}^{-1} \cdot \boldsymbol{\sigma} \cdot \mathbf{F}^{-c} \quad \text{second Piola-Kirchhoff stress tensor}$$

$$\mathbf{E} = 1/2(\mathbf{F}^c \cdot \mathbf{F} - \mathbf{I}) \quad \text{Green-Lagrange strain tensor.}$$

Fig. 1. Damaged volume element.

3. MECHANICAL DESCRIPTION OF THE DAMAGE STATE

3.1. Damage definitions

Degradation of material properties is the result of initiation, growth and coalescence of microcracks or microvoids. Within the context of CDM one may model this process by introducing an internal field variable defining the state of damage locally and recording its accumulation. In the following damage is considered as the only dissipative mechanism.

Consider a damaged volume element δV (Fig. 1). The normal $\mathbf{n}$ defines a surface of intersection δS . Due to the formation of microscopic defects in the form of distributed microcracks and microvoids, the effective load-carrying area is decreased from δS to δS_e . The damage variable associated with the direction of the normal $\mathbf{n}$ is defined as:

$$D(\mathbf{n}) = 1 - \delta S_e / \delta S. \quad (8)$$

When cracks and voids are regularly oriented, D is a function of the direction of the normal $\mathbf{n}$, which implies that an adequate representation of damage would require a continuum tensor field [11–14]. In the theory to be discussed it is assumed that the damage state is isotropic. Hence the damage variable $D_{(\mathbf{n})}$ is no longer dependent on the direction of the normal $\mathbf{n}$ and can be characterized by a scalar quantity $D = D(\mathbf{x}, t)$, for which $D = D(\mathbf{x}, 0) = D_0 \geq 0$ corresponds to the intrinsically damaged state and $D = D(\mathbf{x}, t_c) = D_c \leq 1$ corresponds to complete local rupture. For isotropic damage the effective Cauchy stress tensor $\hat{\sigma}$ associated with the effective surface δS_e is related to the Cauchy stress tensor σ by

$$\hat{\sigma} = \sigma \delta S / \delta S_e = \frac{\sigma}{1 - D}. \quad (9)$$

In addition the hypothesis of strain equivalence [5–6] is introduced, which states that the constitutive equation for a damaged material ($D \neq 0$) can be derived from the same Helmholtz free energy potential as for a virgin material ($D = 0$) by replacing the stress tensor with the effective stress tensor. Mathematically, in the absence of other dissipative effects such as plastic flow, this assumption amounts to postulating a constitutive relation in effective stress space of the form

$$\hat{\sigma} = 1/JF \cdot \frac{\partial \psi^0(\mathbf{E})}{\partial \mathbf{E}} \cdot \mathbf{F}^c \quad (10)$$

where ψ^0 is the free energy potential of the undamaged material. Using eqs (9) and (10) the resulting constitutive equations are written as

$$\mathbf{P} = (1 - D) \rho_0 \frac{\partial \psi^0(\mathbf{E})}{\partial \mathbf{E}} \quad (11)$$

$$\dot{D} = f(\mathbf{E}, D) \geq 0. \quad (12)$$

In the constitutive equations a natural coupling between the elastic mechanisms and the damage mechanisms is obtained. If it is assumed that the damage growth has no influence on the stress–strain relation until a critical damage level is reached,

$$\mathbf{P} = [1 - D_c H(D - D_c)] \rho_0 \frac{\partial \psi^0(\mathbf{E})}{\partial \mathbf{E}} \quad (13)$$

where H is the Heaviside step function, the elastic and dissipative mechanisms are uncoupled. The consequences of this assumption will be discussed in Section 4.3.

3.2. Stochastic nature of damage

Different damage states will be observed for a given population of testing specimens under identical loading conditions. This phenomenon can only be explained by assuming that the specimens possessed different intrinsic damage states. Thus for predicting the damage state of a body under loading its intrinsic damage distribution is required locally. In the following a model, which is based on the statistics of extremes[9] is proposed for determining this distribution.

Since the parameters in the intrinsic damage distribution are unknown in advance, the model must be confronted with experimental data. Therefore, a physical meaning is attributed to the damage variable D . In eq. (8) damage is interpreted as the effective area reduction caused by distributed defects. If the damage mainly consists of microcracks, then eq. (8) needs some adaptation. For a small density of randomly oriented cracks the stiffness reduction can be described with a crack density parameter[15], which will be used as a damage variable

$$D = c^2/\delta S; \quad c = \|(l_1, \dots, l_n)\| \quad (14)$$

where for convenience an effective defect size c is introduced, which must be thought of as the defect size that has the same effect as the smaller interacting defects with size l_i in the surface element δS . Combining classical fracture mechanics and damage mechanics, Lemaitre[16] derived eq. (14) as an approximate expression for isotropic damage in a volume element.

Consider a body with intrinsic damage in the form of microcracks. Let the body be divided into n cells with surface δS_i ($i = 1, 2, \dots, n$). In order to use the concepts of continuum mechanics, it is noted that the ratio of the linear cell sizes must be of the order 1. The damage in cell i is expressed by eq. (14). The damage state and consequently the effective defect size are stochastic variables. The model will be employed in terms of the effective defect sizes c_i , where the underscore denotes that c_i is a stochastic variable. In a globally homogeneous stress field failure of the specimen is initiated in the cell with the largest intrinsic defect size. This maximum defect size is also a stochastic variable, denoted with c_m . The cumulative distribution function (CDF) of the maximum defect size is expressed in terms of those of the c_i by realizing that

$$F_{c_m}(c) \equiv P(c_m \leq c) = P\{(c_1 \leq c) \cap (c_2 \leq c) \dots \cap (c_n \leq c)\}. \quad (15)$$

If the defect sizes are independent, eq. (15) becomes

$$F_{c_m}(c) = P(c_1 \leq c)P(c_2 \leq c) \dots P(c_n \leq c) = \prod_{i=1}^n F_{c_i}. \quad (16)$$

If all the c_i are identically distributed, we may write

$$F_{c_i}(c) = F_c(c; \delta S_i) = \int_0^c f_c(x; \delta S_i) dx \quad (17)$$

where the cell area δS_i acts as a parameter. If the body consists of n equally sized cells, eq. (16) reduces to

$$F_{c_m}(c) = [F_c(c)]^n. \quad (18)$$

In this expression F_c is referred to as the parent distribution and F_{c_m} is the maximum extreme-value distribution. The probability distribution function (PDF) of c_m is obtained by differentiating eq. (18) with respect to c

$$f_{c_m}(c) = n[F_c(c)]^{n-1}f_c(c) \quad (19)$$

where f_c is the PDF of each of the c_i . The PDF of the damage parameter D is determined by

$$f_D(D) = f_c(c) \left| \frac{dc}{dD} \right|. \quad (20)$$

Combining eqs (19) and (20) the PDF of the maximum damage in a body of n equally sized cells

can be expressed as

$$f_{D_m}(D) = n[F_\xi(c)]^{n-1} f_\xi(c) \left| \frac{dc}{dD} \right|. \quad (21)$$

Let an experimental frequency distribution of the maximum defect sizes in a body and a model for the PDF of the damage in a cell be given. The PDF of the maximum defect size distribution in the body can be determined using eqs (19) and (20). This PDF contains a set of unknown model parameters. By minimizing a merit function

$$J = \sum_{i=1}^n |f_{\xi_m}(c_i) - \hat{f}_{\xi_m}(c_i)|^2 \quad (22)$$

that measures the agreement between the experimentally frequency distribution $\hat{f}$ and the theoretical PDF f with a particular choice of parameters, the best-fit model parameters are found. Among others the minimization yields the best-fit parameter for the cell area. Henceforth this cell will be called an elementary cell (EC). Due to the assumption of locally isotropic damage, the experimental distribution of the maximum defect size in a population of testing specimens can be derived from static uni-axial tensile tests.

The EC can be considered as equivalent to a representative volume element (RVE), which is defined as the smallest volume to represent the discrete mechanisms of damage by a continuous variable[16]. Up to the present the size of a RVE was a subjective value, which could only be guessed for after careful micrographic examination of material structures. With the proposed theory the cell dimensions are obtained from a minimization problem.

4. APPLICATION OF THE THEORY ON A TENSILE FATIGUE TEST

4.1. Constitutive equations

A class of materials of practical importance is that of rubbers, which are highly flexible crosslinked networks. In common with other crosslinked polymers rubbers fracture in a brittle manner in the sense that any plastic flow is very local, but this is accompanied by large elastic deformations. Hence the damage growth is considered as the only inelastic mechanism during deformation. In this section the proposed theory is employed on an elastomeric biomaterial in a tensile fatigue test. The one-dimensional stress-strain relation for the undamaged material is given by

$$\sigma = e\epsilon^m \quad (23)$$

where ϵ is the logarithmic strain, and e and m are material constants. Using the concept of effective stress and the hypothesis of strain equivalence, the constitutive equation for the damaged material is expressed as

$$\sigma = (1 - D)e\epsilon^m. \quad (24)$$

For cumulative damage growth, where damage depends on the number of cycles N , we adapt the empirical Paris law for fatigue crack growth[17]

$$\frac{dc}{dN} = a \Delta T^b \quad T_0 \leq \Delta T \leq T_c \quad (25)$$

where a and b are crack growth constants and ΔT is the range of the tearing energy in a cycle. For convenience the minimum tearing energy is taken zero. For a crack in a sheet specimen in simple extension it is found that[18]

$$T = 2Kc\psi^0(\epsilon) \quad (26)$$

where ψ^0 is the potential of the undamaged material and K is a function, which varies weakly with strain. The major influence here is the shortening of a crack due to large strains. Using eq. (14) and (26) the crack growth law can be transformed to the following damage evolution law

$$\frac{dD}{dN} = p\psi^0(\epsilon)^b D^r \quad u_0 \leq D\psi^0(\epsilon) \leq u_c \quad (27)$$

where $p = 2^{b+1}aK^b\sqrt{\delta}S^{b-1}$ and $r = (b + 1)/2$.

Fig. 2. Tensile specimen, which contains n cells with area δS .

Substitution of eq. (24) in the damage evolution law yields the following ordinary differential equation for D

$$\frac{dD}{dN} = \alpha(1 - D)^\beta D^r \quad \hat{u}_0 \leq D(1 - D)^{\beta/b} \leq \hat{u}_c \quad (28)$$

where

$$\alpha = p \left[\frac{e}{m+1} \right]^b \left[\frac{\sigma}{e} \right]^\beta \quad \text{and} \quad \beta = -\frac{b(m+1)}{m}.$$

If the stress-strain relation is given by

$$\sigma = [1 - D_c H(D - D_c)] e \epsilon^m \quad (29)$$

analytical solution of eq. (27) yields

$$D(N) = ((1 - r)p\psi^0(\epsilon)^b N + D_0)^{1/(1-r)} \quad \hat{u}_0 \leq D \leq \hat{u}_c. \quad (30)$$

4.2. Derivation of the intrinsic damage distribution

Consider the tensile specimen in Fig. 2, which is loaded sinusoidally in tension at a fixed frequency level. Let the specimen surface A be built up of n identical cells. The area δS of a cell is unknown *a priori*. Let the damage in a cell be distributed exponentially

$$f_D(D; \lambda) = \lambda \exp(-\lambda D). \quad (31)$$

Using eq. (14) and the transformation law for probabilities (20) we find

$$f_c(c; \lambda, \delta S) = (2c\lambda/\delta S) \exp(-\lambda c^2/\delta S). \quad (32)$$

From eq. (17) it follows that

$$F_c(c; \lambda, \delta S) = 1 - \exp(-\lambda c^2/\delta S). \quad (33)$$

After substitution of eqs (32) and (33) in (19) the PDF for the maximum defect size distribution in a specimen consisting of n cells is given by

$$f_{c_m}(c; \lambda, \delta S) = 2nc\lambda\delta S^{-1} \exp(-\lambda c^2/\delta S) [1 - \exp(-\lambda c^2/\delta S)]^{n-1}. \quad n = A/\delta S \quad (34)$$

Fitting eq. (34) with the adjustable parameters λ and δS to a set of observations, the best-fit parameters $\hat{\lambda}$ and A_{ec} are obtained.

The basic idea behind the determination of the experimental distribution function of the maximum defect size is that for any particular material the breaking strength is controlled by the size of the defects present in the structure. The strength of a body can be increased by reducing the size of these defects. The reduction of the size of artificially induced cracks clearly also causes an increase in the strength of materials. When the crack size is reduced below a critical level, the breaking strength becomes independent of crack size. The materials therefore behave as if they contain natural defects of these critical sizes.

Data about intrinsic defect size distributions in an elastomeric biomaterial (Biomer) were acquired by Gadkaree and Kardos[10]. In static tensile tests they measured the distribution of breaking strength in a population of specimens without an induced cut. Furthermore the breaking strength of specimens with an induced cut of variable size was measured, which yielded a relationship between the breaking strength and the maximum defect size in a specimen (Fig. 3). After transformation of variables an expression for the experimental distribution of the maximum defect size in a specimen is obtained.

Fig. 3. Dependence of breaking strength upon crack length for Biomer[9].

Employing a least squares fit according to eq. (22) the best-fit model parameters turn out to be $\hat{\lambda} = 437.8$ and $A_{ec} = 0.385 \text{ mm}^2$. The parent distribution of the intrinsic defect size (eq. 32) and the fitted extreme-value distribution of the intrinsic defect size in the specimen are plotted in Fig. 4. Calculation of the mean of the parent distribution yields $\mu_c = 0.026 \text{ mm}$. This value agrees surprisingly well with the intrinsic defect sizes in natural rubbers, which amount to 0.025 mm [17].

It is noted that the linear size of an EC of Biomer lies within the scale for a representative volume element (RVE). For polymers this scale of local averaging of the microstructural defects ranges somewhere between 0.1 and 1 mm [16]. Hence, with the proposal theory the question of the scale of a RVE is reduced to a minimization problem.

4.3. Results

Two possible definitions of failure can be used.

- (1) The specimen fails if its most severely damaged EC fails. This approach yields a

Fig. 4. Extreme value distribution of the intrinsic defect size in the specimen (curve 2) and its parent distribution (curve 1).

Fig. 5. Intrinsic damage distribution according to definition (1) for different cell dimensions kA_{ec} ($k = 1, 2, \dots, n = A/A_{ec}$).

conservative estimate of the lifetime distribution. Using eq. (21) the PDF for the maximum intrinsic damage in a specimen is expressed as

$$f_{D_m}(D_0) = n\lambda \exp(-\lambda D_0)[1 - \exp(-\lambda D_0)]^{n-1} \quad (35)$$

where $n = A/A_{ec} = 436$. The effect of the cell area on the damage state is demonstrated in Fig. 5, where the PDF of the maximum damage in a cell with dimensions kA_{ec} ($k = 1, 2, \dots, n$) is plotted. Utilizing the data from [9] we obtain $K = 1.6$, $a = 0.0002 \text{ N}^{2.5} \text{ mm}^{3.5}$, $b = 2.5$, $e = 11.1 \text{ N mm}^2$, $m = 154$, $\sigma = 10.89 \text{ N mm}^2$. The coefficients in the damage evolution law (28) turn out to be $\alpha = 0.1$, $\beta = 4.123$ and $r = 1.75$.

(2) The specimen fails if a crack grows vertically until its size equals the specimen width w . In terms of CDM a strip with dimensions $A_s = w\sqrt{A_{ec}}$ (Fig. 2) must fail. Since this failure state corresponds to complete rupture a more realistic estimate of the lifetime distribution is obtained. The PDF of the maximum intrinsic damage is given by eq. (35) with $n = A/A_s = 40.96$. Employing a least squares fit with fixed $A_s = w\sqrt{A_{ec}}$ we find $\hat{\lambda} = 2920$. The PDF of the maximum intrinsic damage in a cell with dimensions kA_s ($k = 1, 2, \dots, n$) is plotted in Fig. 6. In this case the coefficients in the damage evolution law (28) are $\alpha = 0.768$, $\beta = 4.123$ and $r = 1.75$.

In general the probability that a specimen will fail after N cycles is

$$F_n(N) = P(\underline{D}_m > D_c; N) = \int_{D_c}^{\infty} f_{D_m}(\xi; N) d\xi. \quad (36)$$

If the damage evolution is expressed by eq. (28), the PDF of the maximum damage after N cycles cannot be derived analytically. Therefore, the lifetime distribution must be approached with simulation techniques. Simulation techniques essentially assume that the statistics of the input random quantities are known. The solution process then consists of performing n experiments. In each experiment a set of values of input quantities is chosen, performing a deterministic analysis and thus obtaining a set of response quantities. The response statistics are finally calculated from the sample of responses. Given the maximum intrinsic damage distribution in a specimen (eq. 35) the distribution of the number of cycles to failure is computed for both failure definitions with $n = 50$ experiments. For any particular realization of the intrinsic damage distribution the damage evolution law was solved numerically with a fourth order Runge–Kutta integration scheme with adaptive stepsize control[19]. The resulting lifetimes were fitted to a two-parameter Weibull distribution, which is commonly identified with structural failure applications

$$F_n(N) = 1 - \exp\left[-\left[\frac{N}{N_0}\right]^\phi\right]. \quad (37)$$

Fig. 6. Intrinsic damage distribution according to definition (2) for different cell dimensions kA_s , ($k = 1, 2, \dots, n = A/A_s$).

It was found that $\phi = 6.1$ and $N_0 = 193$ for definition (1) and $\phi = 4.9$ and $N_0 = 254$ for definition (2). When the mean of the maximum damage distribution is chosen as a particular realization of the maximum intrinsic damage in the specimen ($\mu_D = 1.46 \times 10^{-3}$), the damage evolution according to definition (2) is plotted in Fig. 7 for 3 different maximum stresses. Due to the instantaneous rise in the damage in high cycle fatigue problems, it is extremely difficult to determine the damage evolution in a body by measuring variations in the stress-strain relation. If the lifetime distribution as well as the maximum intrinsic damage distributions are available, the proposed theory can be applied to derive the parameters in the damage evolution law.

An analytical solution for the lifetime distribution can be derived if the damage evolution law and the stress-strain relation are uncoupled with eq. (29). In this case the damage after N cycles is given by eq. (30). An expression for the PDF of the maximum damage in a specimen is obtained

Fig. 7. Damage evolution for the coupled equations according to definition (2) for three different maximum tensile stresses, $\sigma = 5.45 \text{ MPa}$, $\sigma = 7.5 \text{ MPa}$ and $\sigma = 10.89 \text{ MPa}$; the mean intrinsic damage is $\mu_D = 1.46 \times 10^{-3}$.

Fig. 8. Experimental cumulative lifetime distribution (curve 1) and theoretical CDFs for both definitions, where the curves 2 and 3 correspond to the coupled equations for definition (1), respectively (2) and the curves 4 and 5 correspond to the uncoupled equations for definition (1), respectively (2).

by transforming the PDF of the maximum intrinsic damage (35) and using eq. (30). The CDF of the number of cycles to failure is obtained after integration of eq. (36).

$$F_n(n) = 1 - [1 - \exp(-\lambda D_0(D_c; N))]^n \quad (38)$$

$$D_0(D_c; N) = [D_c^{1-r} - p(1-r)\psi^0(\epsilon)^b N]^{1/(1-r)} \quad (39)$$

Tensile fatigue experiments were carried out by Gadkaree and Kardos[9] on a population of 10 uncut Biomer specimens at 10.89 MPa maximum sinusoidal stress. The resulting experimental CDF as well as the theoretical CDFs of the lifetimes computed with eq. (37) (coupled equation) and with eq. (38) (uncoupled equation), are plotted in Fig. 8. For failure according to definition (2) the CDF of the uncoupled constitutive equations shows only slight deviations from the CDF of the coupled constitutive equations, whereas for failure according to definition (1) larger deviations are observed. However in this case it appears that the conservative estimate of the lifetime distribution is improved by the uncoupling. Uncoupling the elastic mechanisms and the damage mechanisms has some very attractive features, such as reduction of computing times and improvement of the failure estimates.

A Kolmogorov-Smirnov test of goodness of fit is used[9] to investigate whether the Weibull distribution (eq. 37) for failure according to definition (2) is able to predict the experimental distribution from [10]. The *K-S* test is applicable to ungrouped distributions that are functions of a single independent variable. To apply the test, the experimental CDF as well as the theoretical CDF must be evaluated for each distinct observation. The maximum value of the absolute deviation between the experimental CDF and the theoretical CDF is computed. As can be seen in Fig. 8 the maximum deviation Δ is 0.2 at $N = 279$. At $\Delta = 0.2$ and $n = 10$ the significance level of Δ is $P(\underline{\Delta} > 0.2) = 0.82$ (see Appendix). Since the significance level is sufficiently large, there is no reason to reject the hypothesis that eq. (37) does describe the lifetime distribution at a stress level of $\sigma = 10.89$ MPa.

5. SUMMARY AND CONCLUSIONS

A continuum theory was presented for the modeling of material damage. The theory takes the probabilistic nature of damage into account by treating the internal state variable, which represents the damage mechanisms, as a random variable. The intrinsic damage distribution as well as the dimensions of an elementary cell, which is equivalent to a representative volume element, are computed from a minimization problem.

The model can be used for predicting the damage state if the intrinsic damage distribution and the damage evolution law are known. Furthermore it can also be applied for determining the damage evolution law, especially in cases where damage measurements are extremely difficult. Moreover, the computed elementary cell dimension yields a value for the scale on which micrographic measurements have to be done.

The model was applied to fatigue damage evolution in brittle elastic materials and comparison with fatigue experiments showed promising results.

The proposed model shows a strong similarity with the finite element method with regard to the division of a body into a finite number of cells. Therefore the application of the model in combination with the finite element method provides a useful numerical tool for the failure analysis of loaded structures with stochastic damage. If damage and elasticity are uncoupled a considerable amount of computing time is saved with little loss in accuracy. In a subsequent paper the implementation of the theory in a finite element code will be discussed.

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APPENDIX

Kolmogorov–Smirnov test of fit

The Kolmogorov–Smirnov test is based on the deviate $\underline{\Delta}$

$$\underline{\Delta} = \max_x [F_n(x) - P_x(x)] \quad (\text{A1})$$

where $F_n(x)$ is the cumulative frequency distribution of a sample of size n . $F_n(x)$ is regarded as a discrete random variable whose possible values are $0, 1/n, 2/n, \dots, 1$. Kolmogorov's theorem offers the possibility to estimate the distribution function of $\underline{\Delta}$. In particular if the CDF of x , $P_x(x)$ is continuous, the theorem states that

$$\lim_{n \rightarrow \infty} P[\underline{\Delta} < y/\sqrt{n}] = Q(y) = \begin{cases} 0 & y \leq 0 \\ \sum_{k=-\infty}^{\infty} (-1)^k \exp(-2k^2 y^2) & y > 0 \end{cases} \quad (\text{A2})$$

Kolmogorov's theorem is useful for testing whether two distributions are different. Assume that a CDF $P_x(x)$ has been guessed for x and that $F_n(x)$ has been built up from an observed sample. Then compute

$$\Delta = \max_x |\hat{F}_n(x) - \hat{P}_x(x)| \quad (\text{A3})$$

i.e. the realization of the random variable corresponding to the observed sample. If the sample size is sufficiently large it is possible to calculate from eq. (A2) the significance level of an observed value of Δ .

$$P(\underline{\Delta} \geq \Delta) \cong 1 - Q(\sqrt{n} \Delta). \tag{A4}$$

Small values of P show that the CDF of $F_n(x)$ is significantly different from $P_x(x)$. It should then be concluded that $\hat{P}_x(x)$ cannot be used to describe the random variable $\underline{x}$. However if P is sufficiently large, the calculated deviate Δ is a value that is likely to be found in a single test. In this case the sample test is not in conflict with the hypothetical CDF $P_x(x)$.