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PHENOMENOLOGICAL DESCRIPTION OF THE DISLOCATION MECHANISM OF FORMATION OF NUCLEATED DEFECTS IN PLASTIC DEFORMATION

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The phenomenological approach to the problem of describing the process of fracture consists of introducing damage parameters and kinetic equations [1] or functionals [2] which give their change during loading. This approach was first used in [3, 4] to study damage accumulation during plastic deformation. The system of phenomenological description may not correspond to the micromechanism of the processes taking place, but the presence of a physical interpretation makes such a description more reliable. Here we attempt to construct a phenomenological model of damage accumulation with the well-known dislocation mechanism of growth of nucleated defects.

In accordance with [5], dislocations moving during deformation encounter such obstacles as grain boundaries, subgrains, cells, particles of a secondary phase, etc., and accumulate at these sites, forming small regions with a high density of one type of dislocation. When the number of dislocations in the pileup exceeds a certain critical value, they combine: the pileup disappears and a nucleated defect takes its place. Thus, the process by which dislocations participate in the formation of defects can be tentatively subdivided into two stages: accumulation of dislocations at barriers; combination (disappearance) of the dislocations with the formation of a nucleated defect.

For the mechanism of defect formation proposed in [6] – connected with slip lines overcoming grain boundaries – the first stage is the accumulation of dislocations on corresponding boundaries characterized by a difference in the Burgers vector. The second stage is the formation of the defect (with the disappearance of the Burgers vector difference). For

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the rotational mechanism of defect formation [7] involving uniaxial particulate disclination dipoles based on superdislocations, the first stage consists of rotation of the dipole through an angle (or, equivalently, establishment of the Burgers vector of the superdislocation); the dipole and superdislocation disappear in the second stage and are replaced by the new defect.

1. A set of dislocations passing a given point of a continuum per unit of time can be characterized by the dislocation flux tensor J_{ij} [8], where $\delta u_{ij} = -J_{ij}\delta t$. Here, t is time; u_{ij}, plastic strain tensor. The magnitude of the plastic strain $p_{ij} = -\int_0^t (J_{ij} + J_{ji})\,dt$ being the integral of the symmetrical part of the dislocation flux tensor over time, is taken at a fixed point of the medium and characterizes the set of dislocations passing through the microscopic volume being examined during the time t.

Let an obstacle be located in the path of the dislocation flow. Then the tensor p_{ij} will in some way characterize the set of dislocations which have reached the obstacle during the same period of time. Let $\dot{p}_{ij} = \dot{p}_{ij}^{1} + \dot{p}_{ij}^{2}$, where $\dot{p}_{ij}^{1}$ is that part of the flux $\dot{p}_{ij}$ which forms the dislocation pileups at the obstacles; $\dot{p}_{ij}^{2}$ is that part embraced by the substructure and serving to increase dislocation density in the subboundaries. In the first approximation we assume that the subboundaries envelop a fixed part of the dislocation flow. Then $\dot{p}_{ij}^{1} = k_1\dot{p}_{ij}$ $(0 < k_1 \leq 1)$.

Dislocations not only pile up at obstacles, they also disappear in them and are used in the nucleation of defects. Let a set of dislocations accumulated at obstacles be characterized by the mean tensor ω_{ij}. This tensor has the same structure as p_{ij}.

The equation of dislocation balance at the obstacles can be written in the form

$$\dot{\omega}_{ij} = k_1\dot{p}_{ij} - \dot{p}_{ij}^{0}. \tag{1.1}$$

Here the term $\dot{p}_{ij}^{0}$ characterizes the rate of reduction in the number of dislocations at obstacles due to their participation in defect formation. The tensor $\dot{p}_{ij}^{0}$ formally defined by Eq. (1.1) gives the flow of dislocations which form defects. This flow should repeat the external flow determined by the tensor $k_1\dot{p}_{ij}$ but with a certain lag. The lag is connected with the accumulation (delaying) of the dislocations at the obstacles:

$$\dot{p}_{ij}^{0} = J\left(\dot{p}_{ij}\big|_0^t\right), \tag{1.2}$$

where the lag principle [9] should be observed for the functional J. The simplest suggestion for construction of relation (1.2) is

$$\dot{p}_{ij}^{0} = f(\omega_{ij}, \dot{p}_{ij}) \tag{1.3}$$

(the rate of dislocation loss due to their participation in defect formation is unambiguously determined by the instantaneous state of the dislocation pileups and the external flow). Equations (1.1) and (1.3) yield the following kinetic equation for ω_{ij}:

$$\dot{\omega}_{ij} = k_1\dot{p}_{ij} - f(\omega_{ij}, \dot{p}_{ij}). \tag{1.4}$$

In the special case when

$$f(\omega_{ij}, \dot{p}_{ij}) = k_2\omega_{ij} \tag{1.5}$$

(it is the dislocations which predominate in the pileups that disappear), the equation for ω_{ij} becomes a linear-tensorial equation.

We further assume that under certain conditions (normal temperatures, mean strain rates), the process of dislocation accumulation at obstacles, as the process of plastic deformation, is little sensitive to the time scale. If as this scale we take the length of the plastic-strain arc L $\int\sqrt{dp_{ij}dp_{ij}}$ then, in accordance with (1.4), (1.5), and (1.3),

$$d\omega_{ij} = k_1 dp_{ij} - k_2\omega_{ij}dL, \quad dp_{ij}^{0} = k_2\omega_{ij}dL. \tag{1.6}$$

The relation between the stresses and strains (case f) presented in [10] can be derived on the basis of an equation which is somewhat more general than (1.6). The proposed equation

connects the tensor of the residual microstresses and the plastic strains. It was used to formulate equations of the theory of plasticity and creep in [11] and in several other studies. It must be noted that, in accordance with the criteria in [3, 4], it is the components of the residual microstresses that determine the rate of damage accumulation. In [12], the relation (1.6) ($k_1 = 1$) was obtained on the basis of the hypothesis that the material can be adapted to the strain path by means of the tensor p_{ij}^0 in such a way that the least amount of damage will be incurred. The functional (1.2) was constructed in [13] by using the supposition that the components of the tensor p_{ij}^0 are coordinates of the center of gravity of the preceding stress–strain curve. In the case of variable density ρ for points on the strain path, we obtain

$$p_{ij}^0 = \frac{\int_0^L \rho(L, l)\, p_{ij}(l)\, dl}{\int_0^L \rho(L,l)\, dl}. \tag{1.7}$$

Equation (1.7) reduces to (1.6) if $\rho(L, \ell) = \rho_1(L)\rho_2(\ell)$ [for example, for exponential density $\rho(L, \ell) = e^{-k(L-\ell)}$]. Otherwise [for example, for $\rho(L, \ell) = (L - \ell)^{-m}$, $0 < m < 1$] the quantities p_{ij}^0 (1.7) cannot be solutions of (1.6).

It is clear that we should have $k_2 \geq 0$ in (1.6) (the capacity of the dislocation pileups does not increase, but instead decreases, due to the participation of dislocations in defect formation). Let $k_2 = \text{const}$. For a nonmonotonic change in the components which is proportional in the general case,

$$p_{ij} = \dot{p} p_{ij}^*, \quad p_{ij}^* = \text{const}, \quad p_{ij}^* p_{ij}^* = 1, \quad \left|\frac{dp}{dL}\right| = 1 \tag{1.8}$$

we will have $p_{ij}^0 = p^0 p_{ij}^*$, $\omega_{ij} = \omega\omega_{ij}^*$, where

$$\omega = k_1 \int_0^L e^{-k_2(L-\xi)} \operatorname{sign} \frac{dp}{d\xi}\, d\xi.$$

For monotonic deformation from the initial state

$$\omega = \frac{k_1}{k_2}\left(1 - e^{-k_2 L}\right). \tag{1.9}$$

In accordance with (1.9), for small $L(k_2 L \ll 1)$, $\omega \approx k_1 L$, $p^0 \approx (k_1 k_2/2)L^2 \ll \omega$, $d\omega/dL \approx k_1$, $dp^0/dL \approx k_1 k_2 L$, $dp^0/dL \ll d\omega/dL$. Physically, this means that a large number of dislocation pileups of the critical size has not yet been formed at the early stages of plastic deformation. The disappearance of dislocations takes place slowly, so that nearly all of the dislocations which have reached obstacles remain in pileups. With a further increase in L, the rate of increase in ω begins to lag behind the rate of increase in p by the amount $\dot{p}^0$, which increases monotonically. At $L \to \infty$, the value of ω asymptotically approaches $k_1/k_2 = \text{const}$. Here, conversely, $\dot{\omega} = k_1 e^{-k_2 L} \approx 0$, $\dot{p}^0 \approx k_1 \dot{p} = k_1$, which corresponds to the attainment of dynamic equilibrium between dislocations which have reached obstacles and dislocations disappearing with the formation of defects. Here, the capacity and configuration of the dislocation pileups does not change during deformation, while the amount of damage present increases.

If reversal takes place at a certain point on the monotonic strain path with $L = L_1$, then ω begins to decrease sharply ($d\omega/dL = -k_1(2 - e^{-k_2 L_1})e^{-k_2(L-L_1)}$). This describes the movement of dislocations away from obstacles with a change in the direction of deformation. If the reverse deformation is continued, then ω passes through zero. After this, $|\omega|$ increases for $\omega < 0$, which corresponds to the accumulation of dislocations of the opposite sign at obstacles (that is, dislocations which have recently been formed in the material or have traveled from a given subboundary and reached the opposite subboundary).

It can be shown [12] that in the case of cyclic deformation with the range $\Delta \ll 1/k_2$, a steady-state regime begins after a certain number of cycles of change in ω and $\dot{p}^0$. During this regime, ω changes in accordance with the symmetrical cycle of the range of $k_1\Delta$. This

statement is accurate to within infinitesimals of a higher order than $k_2\Delta$. Thus, the alternating movement of dislocations during cyclic loading is described [14]. This does not mean that the dislocations move cyclically within a subgrain during fatigue loading. Some of the dislocations which have reached the boundaries of subgrains take part in the formation of defects and disappear. Their place is taken by new dislocations which have been formed in the bulk of the material. Thus, we again obtain a unique dynamic equilibrium whereby during each half-cycle as many dislocations are nucleated as are consumed during the same period of time in the formation of defects.

Equation (1.6) may become more complicated if as k_2 we take not a constant but the function L and invariants ω_{ij} and dp_{ij}/dL. Thus, if we take $k_2 = k'\left(\frac{\omega_{ij}dp_{ij}/dL}{\omega_i} + 1\right)$, then after reversal $d\omega/dL = -k_1$, and ω decreases linearly with an increase in L until $\omega = 0$. On this segment, $\dot{p}^0 = 0$. Immediately after reversal, dislocations cease forming defects because they leave pileups in their reverse motion. The pileups therefore cease being critical. The reverse-moving dislocations reach previously formed pileups and are annihilated, which decreases ω. Only after ω passes through zero (i.e., after pileups of the opposite sign are formed) does the quantity $|\dot{p}^0|$ begin to increase from zero. Below, for the sake of simplicity, we will examine only the case k_2 = const.

2. The accumulation of dislocations at obstacles plays a very important role in damaging of the material. However, the instantaneous state of dislocation pileups does not determine the damage state at a given moment of time [5]. Within the framework of the model being used, the process of dislocation accumulation at obstacles is augmented by one other damage accumulation process which takes place at the same time – the stage of defect formation. It is natural to suggest that the rate of defect formation – which we will designate as $\dot{\Omega}$ – depends on the flux $\dot{p}_{ij}{}^0$ reflecting the loss of dislocations at obstacles due to their participation in defect formation. Experiments [5, 15, 16] show that the rate of increase in damage depends on the type of stress state (under creep conditions at atmospheric and high pressure [5], in elastoplastic tension and compress [15], and during extrusion and drawing [16], the loosening of the material or the density of nucleated defects differs widely for the same values of L, while the dislocation structures may be similar). These data show that the parameters expressing the type of stress state should go into the equations of the second stage rather than the first:

$$\dot{\Omega} = F(\dot{p}^0_{ij}, \mu_k). \tag{2.1}$$

Closing of defects may be occurring simultaneously with their nucleation. Defects may be closed either by diffusion or by the mechanism of viscous flow [17]. It is assumed that the rate of diffusion closure depends slightly on the configuration of the plastic strain path, so that this phenomenon can be simply accounted for by changing the parameters of the function F (2.1). The monotonic plastic deformation of a material under certain types of stress state – such as under high hydrostatic pressure – may lead to the healing of previously formed or existing defects. This fact can be taken into account by means of a dependence of F on μ_k such that, in certain ranges of μ_k, the value of F will be negative regardless of the value of $\dot{p}_{ij}{}^0$. It was found in [15] that defects formed in tension are partially healed during subsequent compression. According to [16], defects are created in the drawing or extrusion of annealed specimens. At the same time, defects formed in a prior creep test are closed by extrusion or drawing. In [18], it was found that microcracks are closed even in tension if the direction of a microcrack changed by the angle $\pi/2$. This means that closing of defects can be intensified by a sharp change in the direction of deformation (even given the same fairly severe stress state). In fact, a defect formed by the combination of a certain number of defects of one type may "surface" when it is reached by dislocations of the opposite sign. Thus, it is suggested that the rate of defect closure is an increasing function of the angle φ between the direction of the flux $\dot{p}_{ij}$ and ω_{ij}. In particular, we take

$$\dot{\Omega} = F(\dot{p}^0_{ij}, \mu_k)\,\chi(\mu_k, \cos\varphi), \tag{2.2}$$

where

$$\chi = 1 + z(\mu_k)\cos\varphi,\ z(\mu_k) > 0. \tag{2.3}$$

In accordance with (2.3), the value of χ is maximal at $\varphi = 0$ and minimal at $\varphi = \pi$. Negative values of χ (closure) are possible only when $z(\mu_k) > 1$ for angles of change in the strain

path $>\pi/2$ (with a change in the direction of tension by the angle $\pi/2$ [18], the angle of the change in the strain path in the strain space is equal to $2\pi/3$). Thus, a decrease in damage after a sharp change in the strain path is possible only until the angle between ω_{ij} and $\dot{p}_{ij}$ becomes less than $\pi/2$ (this circumstance prevails by virtue of satisfaction of the lag principle if the next strain path is close to monotonic). The effect just discussed was confirmed by microscopic observations [16]. In accordance with the latter, an increase in the degree of extrusion or drawing of specimens with previously initiated defects is accompanied initially by an increase in the density of the material. After passage over a certain part of the strain path, the material then begins to loosen. Density increases more rapidly in the case of extrusion of defective specimens than in the case of drawing. Thus, the value of z in (2.3) may depend on the parameters of the type of stress state.

3. Let Ω be a scalar [this assumption is valid if we are examining deformation of the type (1.8)]. In this case, the function F should depend on the invariants of the tensor $\dot{p}_{ij}{}^0$. Taking the intensity of the tensor $\dot{p}_i{}^0 = \sqrt{\dot{p}_{ij}{}^0\dot{p}_{ij}{}^0}$ as such an invariant, and taking L as the time (we assume that under the given conditions the process of dislocation, combination, with the formation of defects, does not depend on the time scale), we obtain $d\Omega = F_1(\dot{p}_i{}^0, \mu_k)\left[dL + z(\mu_k)\frac{\omega_{ij}dp_{ij}}{\omega_i}\right]$. Alternatively, with k_2 = const, by virtue of (1.6), we obtain

$$d\Omega = \psi(\omega_i, \mu_k)[\omega_i dL + z(\mu_k)\omega_{ij}dp_{ij}]. \tag{3.1}$$

At z = 0, (3.1) becomes the equation of the criterion in [3, 4]. This criterion is usually written in the form $d\Omega/dL = \lambda P$, where λ = const and P is the intensity of the residual microstresses [its role is played by the tensor ω_{ij} in (3.1)]. In the case of the absence of the term $\omega_i dL$ in (3.1), this relation becomes the relation from [19].

Equations (1.4) and (3.1) describe the formation of nucleated defects, which controls damage accumulation [20]. If the transition from scattered damage formation to the formation of a macrocrack takes place at a certain critical density of nucleated defects, then the moment of formation of the crack may correspond to attainment of the critical value of the measure Ω. The latter depends on the stress state [3, 4]:

$$\Omega = \Omega^*(\mu_k). \tag{3.2}$$

To study damage accumulation with a nonproportional change in p_{ij}, it may prove insufficient to represent the damage with a scalar. The general form of a fracture criterion which uses a tensorial damage parameter was presented in [2]. In [4], the damage state during plastic deformation was described by a symmetrical second-rank tensor Ω_{ij} obeying the kinetic equation $d\Omega_{ij}/dL = F_{ij}$, where F_{ij} is the microstress function. The same study examined the following kinetic equation as the simplest variant

$$\frac{d\Omega_{ij}}{dL} = A\frac{p_{ik}p_{kj}}{p_i}, \quad A = \text{const}, \tag{3.3}$$

which at A > 0 ensures a monotonic accumulation of damages [4]. By analogy with (3.3), the function F in (2.2) can be chosen in the form $F(\dot{p}_{ij}{}^0, \mu_k) = F_1(\dot{p}_i{}^0, \mu_k)\dot{p}_{ik}{}^0\dot{p}_{kj}{}^0$. Replacing the time by the length of the plastic-strain arc and using (1.6), for k_2 = const we have

$$d\Omega_{ij} = \psi(\omega_i, \mu_k)\omega_i^{-2}\omega_{ik}\omega_{kj}[\omega_i dL + z(\mu_k)\omega_{mn}dp_{mn}] \tag{3.4}$$

[the multiplier $\omega_i{}^{-2}$ was chosen for convenience in comparing the above with (3.1)]. According to [21], breakup of the core of a dislocation with the formation of a defect occurs either along an excess plane or along the slip plane. Here, Ω_{ij}, determined from (3.4), can be represented as $\Omega_{ij} = \sum_{k=1}^{2}\Omega n_i^{(k)}n_j^{(k)}$, where $n_i(k)$ are components of unit normals to the planes of the formed cracks. The simplest variant of the limiting condition has the form

$$\sqrt{\Omega_{ij}\Omega_{ij}} = \Omega^*(\mu_k). \tag{3.5}$$

With deformation (1.8), we find from (3.4) that $\Omega_{ij} = \Omega p_{ik}{}^* p_{kj}{}^*$, where $d\Omega = \psi(|\omega|, \mu_k)(|\omega|dL + z(\mu_k)\omega dp)$, $|\Omega| = \Omega^*(\mu_k)(p_{ik}{}^* p_{kj}{}^* p_{im}{}^* p_{mj}{}^*)^{-1/2}$. In accordance with (3.1), the value of Ω satisfies the same equation with limiting condition (3.2). Thus, the scalar and tensor

variants will actually give the same results for proportional (in the case of nonmonotonic) deformation. These results will be discussed briefly below.

For low-cycle fatigue with strain-controlled proportional loading [22] in a steady-state regime, of change in ω_{ij}, the Coffin–Manson low-cycle fatigue equation $\Delta = p_*/N^{\alpha}$ is satisfied if $\psi(\omega_i, \mu_k) = \gamma(\mu_k)\omega_i^{n(|\mu_k|)} + \Phi(\omega_i, \mu_k) - \Phi(\omega_i, -\mu_k)$. Here, N is the number of cycles to fracture; Δ is the range of plastic strain in a cycle; Φ is an arbitrary function which cannot be determined from low-cycle fatigue tests; it is assumed that the values of the parameters μ_k for forward and reverse deformation will have different signs. The material functions $\gamma(\mu_k)$ and $n(|\mu_k|)$ are connected by the following relations with cyclic toughness p_* and the exponent of the low-cycle fatigue curve α, which are easily determined in the experiment

$$\alpha = \frac{1}{n(|\mu_k|)+2}, \quad p_* = 2\left\{\frac{q\Omega^*(\pm\mu_k)[n+2]}{2[\gamma(\mu_k)+\gamma(-\mu_k)]k_1^{n+2}}\right\}^{\frac{1}{n+2}}, \tag{3.6}$$

where the + of − sign in the expression $\Omega^*(\pm\mu_k)$ is chosen in relation to whether fracture occurs during forward or reverse deformation; q = 1 for (3.1) and $q = (p_{ik}^* p_{kj}^* p_{i\ell}^* p_{\ell j}^*)^{-1/2}$ for (3.4), (3.5). The quantity $z(\mu_k)$ does not go into the low-cycle fatigue equation because the result of integration of the closing term $z(\mu_k)\omega dp$ in a closed cycle is equal to zero in the case of a steady-state regime of change in ω_{ij}.

For a low number of cycles, particularly with asymmetric deformation, there is a reduction in endurance compared to that obtained with the Coffin–Manson equation [22]. This empirical fact is described both qualitatively and quantitatively within the framework of the model being examined [12, 13]. The size of the deviation increases with a decrease in the parameter k_2 (1.5). Thus, a test involving strain-controlled asymmetric deformation makes it possible to determine this parameter of the model. It follows from the equations of the model that preliminary monotonic deformation lowers endurance during subsequent low-cycle loading with sufficiently large amplitudes of plastic strain. In contrast to the theory of damage summation – where the effect of a preliminary monotonic strain path reduces to the addition of damage from this path to the total damage – in the present model the rate of damage accumulation on the cyclic section is increased by the presence of the section of monotonic deformation. The intensity of the effect of one part of the strain path on the rate of damage accumulation during deformation along the other parts of the path can be varied by changing the parameters of functional (1.2) [with a reduction in k_2 in (1.6), the mutual effect is reinforced].

The model describes experimental data on residual ductility [3, 13], i.e., on monotonic deformation to fracture after preliminary cyclic deformation. The following power relation is satisfied for the value of the plastic-strain parameter at the moment of fracture $p^* \ll 1/k_2$

$$p^* = \mu\left[1 - \frac{\Omega_0}{\Omega^*(\mu_k)q}\right]^{\beta}, \tag{3.7}$$

if

$$\psi(\omega_i, \mu_k) = \Theta(\mu_k)\omega_i^{m(\mu_k)}, \tag{3.8}$$

$$\beta = \frac{1}{m(\mu_k)+2}, \quad \mu = \left(\frac{\Omega^*(\mu_k)q}{\beta\Theta(1+z)k_1^{m(\mu_k)+1}}\right)^{\frac{1}{m(\mu_k)+1}}.$$

Here, Ω_0 is the damage accumulated during the preliminary cyclic deformation. If the number of preliminary cycles is large enough so that ω_{ij} changes in the steady-state regime during cyclic deformation, then Eq. (3.7) takes the form

$$p^* = \mu\left[1 - \frac{k}{N}\right]^{\beta}, \tag{3.9}$$

where k is the number of preliminary cycles completed; N is the number of cycles of the same amplitude necessary for fracture. Equation (3.9) was validated empirically in [23, 24]. If

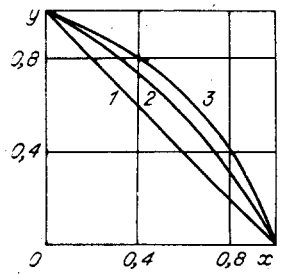

Fig. 1

we simultaneously require satisfaction of power relations for both low-cycle fatigue and for the residual ductility, then the superscript m should be an even function: $m = m(|\mu_k|)$. Meanwhile, according to (3.6) and (3.8), the exponents of the curves describing low-cycle fatigue and residual ductility should coincide. This is consistent with the results in [24].

The model predicts the following effects in regard to residual ductility during nonmonotonic deformation. Residual ductility after one pulsating cycle is greater when the deformation is carried out in the direction of the completed cycle than in the case of the opposite direction [13]. Residual ductility after one symmetrical cycle is greater for deformation in the direction of the last half-cycle. These effects were confirmed experimentally on specimens of brass LS-59 and steel U-8 in [12, 13]. The results of the experiments conducted in [25, 26] can be explained on the basis of the presence of the term describing defect closure in the model. According to these results, specimens brought almost to fracture in torsion in one direction can be twisted through a fairly large angle in the opposite direction without fracture. It was shown in [27] for the case of nonproportional cyclic deformation within the framework of the scalar model [3] that the safe life of the material is always exhausted more rapidly than with a proportional cyclic change in the components p_{ij} with the same strain-path diameter per cycle. This finding was confirmed experimentally in [28] for specific strain paths. It can be proven that for tensor model (3.4) in the case when defect closure is negligible, the length of the strain path to fracture with a nonproportional change in the components will be greater than the length for the corresponding scalar model. Thus, the tensor model somewhat reduces the effect of nonproportionality on endurance compared to the scalar model. This is illustrated in the example given below. Let there be n_1 cycles of strain-controlled deformation (1.8) with the directrix deviator $p_{ij_1}^*$. Then let there be n_2 cycles with the directrix deviator $p_{ij_2}^*$. It is assumed that the quantities n_1 and n_2 are large enough so that the change in ω_{ij} on both sections of the strain path can be assumed to be steady. Using tensor models (3.3) or (3.4) – which are indistinguishable in the present case – we obtain the damage summation law

$$\left(\varkappa\frac{n_1}{N_1}\right)^2+\left(\frac{n_2}{N_2}\right)^2+2\frac{n_1}{N_1}\frac{n_2}{N_2}\varkappa\cos\beta=1, \tag{3.10}$$

where N_1 and N_2 are the number of cycles corresponding to fracture for the first and second amplitudes; β is the angle between the deviators $p_{ik_1}^*p_{kj_1}^*$ and $p_{ik_2}^*p_{kj_2}^*$, $\kappa = \Omega_1^*/\Omega_2^*$ (Ω_1^* and Ω_2^* are limiting values of the quantity $\sqrt{\Omega_{ij}\Omega_{ij}}$ for the first and second types of stress state). In accordance with scalar model (3.1), for any β

$$\varkappa(n_1/N_1)+n_2/N_2=1. \tag{3.11}$$

Equation (3.11) is obtained from (3.10) at $\beta = 0$, i.e., for a proportional change in the components p_{ij}. Figure 1, in the coordinates $x = n_2/N_2$, $y = \varkappa n_1/N_1$, shows the graph of (3.11) (line 1) and (3.10) (lines 2 and 3 with $\cos\beta = 1/2$ and $1/3$).

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