MECHANISMS OF PLASTICITY AND FRACTURE IN CRYSTALS, POLYMERS UNDER LOW/SHOCK-WAVE STRESSES

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SUMMARY

The effect of applied compressive/extension stresses, s (s = 0.6S to 95S, where S is the resolved shear stress) and stress rates (10 to 10^6 MPa/sec) on dislocation dynamics was investigated in pure NaCl and InSb single crystals in the temperature range $T = 4\ 10^{-3}$ to 0.945 T_{melt}, T_{melt} is the melting point. The general damping character of dislocation unpinning, motion and multiplication (work hardening of crystals, WH) under creep and interrupted loadings manifests in the ultimate mean path lengths of individual dislocations (UMPID). Having covered a certain UMPID determined by crystal prehistory and test parameters, the dislocations exposed to successive exhausting multiplication and then fracture thus forming point defects, slip lines, slip bands, sub-grains, grain boundaries, nano- and microcracks, macrocracks in series in all the materials [1-2].

The first important finding of this work is the fact that the dependences of the UMPID versus creep, impulse, impact and shock wave stresses, temperature and impurity concentration are topologically similar to the conventional macroscopic strain-stress WH curves for the same crystals and test parameters. As for microscopic stresses for dislocation motion and multiplication the concentration dependences of flow stresses under fixed strains or fracture stresses at low and ultra-low temperatures and strain rates [2-4] are similar to the same dependences of impact/shock wave stresses and stress rates at normal and elevated temperatures [4-6]. The climb, dislocation cross-slip and athermal bowing mechanisms are confirmed by the same so-called "memory effect" at low (Figs 23-24 in [7]) and ultra-high (s~ 48S, [8]) stresses and stress rates, because dislocation dipoles-debris are left in the wake of expanded dislocation loops along the whole deformation WH curve. This means that the same micromechanisms govern the dislocations and macroscopic flow up to the flow stresses in nanostructured (NSC) and fractured crystals.

The second important finding is that the micro-/macro-WH varies nonmonotonously to crystal softening according to the pulse length of the unloading (restore) time, and these V-formed dependences are the same for micro-/macroscopic flow up to the extremely high values in NSC crystals [9] and fractured oriented polymers [10]. The last fact and the similarity of the other features of deformation and fracture of crystals and polymers at various length scales corroborate the universality of the mechanisms of plastic flow and fracture in crystals and polymers due to the same elemental slips. The third finding of this work is the same quasi-linear correlation between the starting stresses for dislocation motion, Sst, multiplication, Sm, and for the initiation of micro-, S_f, and macrofracture, S_F, in NaCl and KCl-KBr single crystals (this work), polycrystalline ice, metal alloys and YBCO ceramics (literature data). It is worth stressing that in the low range of stresses (low WH, pure crystals or high temperatures) the crystals fractured in so-called "ductile' mode irrespective of test conditions where dislocations demonstrated noticeable cross-slip, wavy glide, while at higher WH stresses dislocation slip lines were straightened, cross-slips were frequent and small, and their fracture mode was only 'brittle'. These findings clearly evidence for the key role of dislocation double cross-slip, climb and retardation thus forming various dislocation structures during deformation modes. When the deformation modes had been exhaus-ted the beginning of fracture modes (the nano-/microcrack initiation, their spreading and coagulation into macrocracks) followed. This

means that the atomic-size cracks are the cores of dislocations, and they stop and unite one after another into nanosize cracks with the help of their close spacing and cross-slip events at higher WH stages of plastic flow. In all tests new flaws originated at much higher stresses from the parent cracks that had initiated earlier, as well as the new-formed flaws at new sites due to the stopped plastic zones around the tips of previous flaws or new sources of dislocation multiplication (due to concentrated local stresses) in the crystal bulk.

The proposed chain of events clearly demonstrates that the mechanisms of plasticity and fracture have the same origin as a sequential chain of the WH deformation modes in crystals, the very beginning of which is demonstrated in the work [1]. It is the above scaling of the parameters of deformation stresses and fracture just with the dislocation multiplication one that points to the crucial role of dislocation double cross-slip and climb mechanisms in all these processes irrespective of other experimental conditions. Moreover, it is clear that the scaling of microfracture and macrofracture stresses with each other and with the flow stresses strictly confirms the crucial and inevitably key role of precursor and intermittent plasticity in nucleation, multiplication and coagulation of initial atomic-size cracks into nano-cracks and so on. It is the applied stress and stress rate that synchronize the successively lengthening of dislocation paths, slip lines and bands, cell walls and bands, etc., thus promoting the initiation and sequential spreading of the waves of plastic modes under various kinds of deformation [1]. The increase of the deformation stress and stress rate raises the production of point defects and their clusters due to cross-slip-jog dragging, reduces the dislocation spacing in deformation structures, activates the numerous new sources of dislocation nucleation and multiplication. The above chain of events initiates closer lay-out of cracks and nano-/micromacrocleavage transitions between them, thus forming the special structures of fracture surfaces (the spacing, sizes, and the form of microcracks coagulated into the larger rupture structures, microcleavage steps, dimples and microvoids on fracture surfaces) up to the sample explosion with the dust fragments at very high stress rates and stresses. The practical art of cleavage for various crystals clearly proves that it is the parameters of dislocation cross-slip in precursor plasticity favors fracture propagation and damage. It is well known that the crystals which are extremely hardened with high impurity content, irradiation dose, very low temperature of cleavage are cleaved much easier than the very plastic (soft) ones. Second, fast cleavage mackrocracks are usually produced with extremely sharp chisel and with very rapid and strong blow struck on the chisel (with high stress rate and stress) which has to be along the cleavage planes. Only in these cases the preceding deformation is extremely hardened and localized, the dislocation bands are straight, dense and very narrow (the lowest cross-slip heights and feebly marked climb of dislocations - the so-called 'brittle' fracture mode). But in the case of the soft crystals or very slow, weal and non-crystallographic attacks with blunt chisel at the struck sur-face these opposite actions lead to much larger areas of crystal deformation zones with all the signs of the crystal softening near the contact points. Here the dislocations are prompt to higher rare cross-slips which make the dislocation slip bands to be wide and diffuse. So, these heavily deformed places have a high density of diffuse microcracks and micropores, which color dense 'milk' due to their intensive day-light scattering.The spacing of these cracks/pores is so large that they rarely unite into macrocracks with rare dislocation cross-slips and hardly form the fracture surfaces. All the above statements are valid for the polymers and all other types of matter [2].

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DEFORMATION OF MOLECULAR AND CELL STRUCTURES IS THE KEY MECHANISM OF AGEING AND ILLNESS

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Literature data irrefutably evidence for the deformation origin of physiological, physical and chemical stress effects on biological tissues (BT), cells growth and proliferation, differentiation, diseases and ageing, which is irresistibly identical to the stiffening or softening deformation of solids with the appropriate production of lattice defects with various dimensions (reactive oxygen species, ROS, in mitochondria of BT stimulate oxidative stress in cells, etc.). Our comparisons of the mechanisms of plasticity (MP) in living BT under arterial blood pressure oscillations (this work), metabolic transformations and stresses, in various states of different materials under load [1] show that MP are strictly the same on atomic-to-cosmic scale lengths. This is the irrefutable argument in proof of the new paradigm [1] of decisive role of MP and phase mismatch-interface stresses in or between growing and differentiating cells, in each stage of their phase transitions: biochemical reactions, the origin, development and medical treatment of endogenous diseases, kinetics of ageing and growth of robust and cancerous cells, adaptation, origin of species and populations, etc. In terms of this paradigm the ageing of BT is a typical fatigue mechanical deformation of cells up to their stiffening and fracture (apoptosis). Softening of BT with physical, biochemical, physiological, etc. methods changes the mechanical fatigue limit of the materials, longevity or lifespan of BT and the rate of their hardening/ageing. And this is in line with the epidemiological, clinical and experimental investigations.

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