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MOLECULAR MODEL OF FRACTURE OF FIBROUS POLYMERIC MATERIAL

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ABSTRACT

The fibrillar model of fibrous structure assumes a dense packing of long and narrow microfibrils into spindle shaped slightly skewed fibrils. The ends of microfibrils located mainly on the outer boundary of the fibrils represent an interruption of the massive axial connection by taut tie molecules. They are prime candidates for microcrack formation upon tensile loading which deforms the sample by shear displacement of adjacent fibrils and microfibrils. The axial and radial growth of microcracks ruptures the tie molecules in its path. The sample fails as soon as one of cracks reaches critical dimensions. The rest of the sample in a new loading run exhibits a lower load-elongation curve but practically the same or even higher strength because the reopening of old microcracks is easier than their initial formation while the growth of microcracks beyond the limits of the first run requires an increased load.

MICROFIBRILLAR MODEL OF FIBROUS STRUCTURE

A highly drawn polyethylene (PE) or nylon 6 sample has as its main structural element the very long, 5-20 μm, and thin, 10-20 nm, microfibril as shown in Figure 1. The microfibril as formed by micronecking of a lamella of the original unoriented material solidified from a relaxed melt consists of alternating folded chain blocks and amorphous layers. The blocks have the chain axis fully oriented in the axial direction of the microfibril. The amorphous layers consist of chain loops, free chain ends and tie molecules (t.m.) connecting subsequent crystalline blocks of the same microfibril (introfibrillar t.m.). Tie molecules originate from unfolding of chains during tearing off of crystal blocks from the lamella at micronecks on the crack boundary of lamella. Some of them are taut and hence transmit mechanical forces through the amorphous layer as almost crystalline bridges joining the blocks. Tie molecules of the starting material connecting two different lamellae yield interfibrillar tie molecules connecting a block of one microfibril with a block of another microfibril.

Another important feature of fibrous structure is the parallel packing of microfibrils into long fibrils [2]. A single fibril seems to arise from necking of a stack of parallel lamellae. The variation of lamella and micronecking zone orientation from stack to stack yields slight variation of draw ratio and crystal lattice orientation of adjacent fibrils which makes the cohesion in the boundary between them a little less strong than between adjacent microfibrils of the same fibril. Moreover, the ends of microfibrils are primarily located on the outer boundary of the fibrils producing there structural defects with the characteristics of a point vacancy. The axial connection by taut t.m., so strong and important inside

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each microfibril, seems to be either missing or at least very much reduced at such an end thus making the axial force transmission much less efficient. The amorphous layer at the ned of a microfibril can be opened much easier than that inside the microfibril because very few if any tie molecules have to be ruptured in the process. Besides that the last folded chain crystal block on the microfibril is interfering strongly with the smooth fit of microfibrils along the boundary between adjacent fibrils. This in addition reduces the cohesiveness of the boundary.

In PE the number of interfibrillar t.m. is substantially smaller than that of the intrafibrillar t.m., their ratio being 1:10 or even smaller. In highly drawn nylon 6, however, the situation seems to be quite different as a consequence of a large increase of interfibrillar molecules during homogeneous drawing of fibrous structure as it was just obtained by necking. Such a drawing mainly proceeds by shear displacement of adjacent microfibrils and hence enormously extends the interfibrillar t.m. by unfolding the chain sections included in the two blocks located on adjacent microfibrils. Here the ratio may be 1:1 or even larger in favour of interfibrillar t.m.]3].

Moreover, the fraction of taut t.m. seems to be substantially different in these two materials. While in highly drawn PE a large fraction of interfibrillar t.m. is rather taut thus yielding a high initial elastic modulus which can be up to 40 times larger than that of the unoriented lamellar material the t.m. in nylon 6 seem to be rather slack yielding a substantially smaller elastic modulus. With increased strain the stress increases less than linearly in both cases, in PE up to the final fracture [4], while in nylon 6 after a while it starts to increase again faster than linearly [5] as if new connectors were activated at higher strain. The drop can in both cases be attributed to the non-linearity of covalent chemical bonds of taut t.m. and of Van der Waals bonding adjacent chains in amorphous conformation. The new connectors in nylon 6, however, must be originally slack t.m. which get fully extended at an intermediate strain so that from then on, one observes with them the same type of covalent bond stretching as in PE from the early beginning. Moreover, the persistence of gradual increase of differential elastic modulus over a finite range of strain up to the break indicates a broad distribution of slackness of t.m. so that they get only gradually fully extended and thus in increasing numbers contribute to the more than linear increase of load with strain.

Such a difference between PE and nylon 6 seems to be caused by the difference in drawing. In the former case the fibrous structure is achieved at a draw ratio $\lambda^* \sim 10$ while in the latter case λ^* is between 2 and 3. Hence one expects in PE much longer microfibrils, and longer and narrower fibrils than in nylon 6. The subsequent plastic deformation of fibrous structure proceeding mainly by longitudinal displacement of fibrils strains the fibrils in a shear mode. The resulting shear displacement of microfibrils inside each fibril is larger, the wider and shorter the fibril. It turns out that the ratio of width to length of the fibrils as formed during micronecking is proportional to $\lambda^{*3/2}$, i.e., to about 30 in PE and 4 in nylon 6. Hence the microfibrils are very little displaced axially in the former and very much in the latter case. Such a shear displacement very much increases the fraction of interfibrillar t.m. which hence in nylon 6, play quite a substantial role while in polyethylene their contribution seems to be rather small (Figures 1a, 1b). After drawing the contraction of the sample upon load release slackens the interfibrillar t.m. more than the intrafibrillar t.m. Hence the effect is more conspicuous with nylon 6 than with PE as a consequence of the larger fraction of interfibrillar t.m. in the former than in the latter case.

The least affected part of fibrous structure during the plastic deformation from λ^* to λ_b (draw ratio at break) are the folded chain blocks of the microfibrils. The shear displacement of adjacent microfibrils induces shear displacement of chains in the crystal blocks which makes more diffuse the boundary between the crystal core and the amorphous layer containing the folds and tie molecules. As a consequence the small angle X-ray scattering becomes so much broader that it is almost undetectable at highest draw ratios.

ELASTIC MODULUS OF FIBROUS STRUCTURE

At very small loads, i.e., in the linear range of stress-strain curve, the response of the axially strained fibrous sample is described by the axial elastic modulus which can be fairly well calculated by Takayanagi's model [6]. If one forgets about the defects of microfibrillar structure the fibrous material can be described by a unit cell model containing in series one crystalline block and one amorphous layer with the latter element bridged by an almost crystalline link representing the intrafibrillar taut t.m. (Figure 2). By assigning to the PE crystal and to the link the elastic modulus E_c between 240 and 300 GPa, derived from the X-ray measurement of the expansion of the c-axis of crystalline lattice under load [7] and to the amorphous component the value $E_a = .5$ GPa one obtains from the measured axial elastic modulus E and crystallinity $\alpha = .8$ for the area fraction β of the taut t.m. link bridging the amorphous layer the minimum value

$$\beta_{min} = \frac{1}{1-E_a/E_c}\left(\frac{(1-\alpha)E/E_c}{1-E_a/E_c} - E_a/E_c\right) \qquad (1)$$

if the link is considered to contribute to the amorphous component of the sample. With $E_c = 240$ GPa and $E = 25$ (PE drawn at 60°C [8]) or 70 GPa (PE drawn between 75 and 90°C [9,10]) one has $\beta_{min} = .025$ or .074, i.e., a very small percentage of chains in the crystal lattice has to act as taut t.m. while the rest can fold back at the surface of the block. The number of slack t.m. can be substantially larger. But it cannot be derived from E since such molecules do not contribute to it in such a clear manner as do fully extended taut t.m.

It is worthwhile to stress that from fumic nitric acid treatment one derived a substantially larger fraction, up to 30%, of taut tie molecules in a sample with E = 25 GPa [11]. This more than 10 times larger number than that derived from elastic modulus may be an indication that also slightly slack t.m. not contributing to E may be sufficiently resistant to acid attack. Such a resistance may be less a question of tautness than of close packing of t.m. which seems to be the decisive factor in chemical degradation as seen from the high resistance of chains on the lateral faces of the crystals as compared with those on the (001) faces.

The bundling of t.m. may also induce another types of crystal deformation which is not considered in Takayanagi's model which assumes a perfectly uniform strain in the crystal and the amorphous layer. This is alright if the taut t.m. are more ore less uniformly distributed over the whole cross section. As soon as they are bundled one has to take into account the lateral force transmission in the crystals from the area of the bundle (cross section β) to the rest of the crystal with a cross-section $1-\beta$. If the bundles are all on the same side of the amorphous layer one can almost completely neglect the force carrying ability of the section $(1-\beta)$

of the alternating crystalline and amorphous component. In such a case of parallel arrangement of almost crystalline column of cross section β with elastic modulus E_c and a column of alternating crystalline and amorphous layers with cross sections $1-\beta$ one obtains for the upper limit of β the estimate

$$\beta_{max} = \frac{E}{E_c}\left(1 - \frac{E_c/E-1}{(1-\alpha)(E_c/E_a-1)}\right) \qquad (2)$$

i.e., .095 for E = 25 GPa and .29 for 70 GPa. Both parallel elements are considered completely independent from each other so that the strains in adjacent crystal domains need not be identical.

This is certainly an oversimplification. One will have a transition region between the two parallel elements in the crystals which means also shearing of load carrying between them. Hence, the correct value of β will be between β_{min} and β_{max}. If one takes the arithmetic mean value $\beta = (\beta_{min} + \beta_{max})/2$ one has $\beta = .0578$ and .1814 which seems to be a reasonable estimate of the fraction of taut tie molecules in the amorphous layers. Their number increases a little faster than linearly with the axial bulk elastic modulus.

The model of fibrous structure as used for the calculation of the fraction β of taut t.m. assumes and ideal lattice of alternating amorphous and crystalline layers. The microfibrillar model points out the existence of structural defects of such a lattice at the ends of microfibrils which as a rule are located at the outer boundary of fibrils. The interruption of the axial force transmission by taut t.m. reduces the elastic modulus by a factor proportional to the reduction of the average cross section of the fully operative amorphous regions of microfibrils as a consequence of the presence of such structural defects. If the microfibril has an average length $\ell_{mf} \sim 10$ µm and the long period $L \sim 20$ nm one has one end per 250 amorphous layers ($= 1/2 \times 10^4 : 20$) and hence a reduction of the cross section of the fully operative amorphous layers for force transmission by a factor proportional to 1/250. Even if each end of a microfibril disturbs the transmission in a wider area, e.g., as a consequence of poor lateral contact with adjacent microfibrils the amorphous layers close to the end of microfibril may carry a substantially smaller load than those farther away, the reduction of elastic modulus of such structural defects will not be very much larger than a few percents. Hence such a reduction can be completely neglected and the elastic modulus of fibrous material calculated as if the microfibrillar structure were ideal.

FRACTURE

Fracture like plastic deformation depends much more on large scale morphology than elastic modulus E. In the latter case, the contribution of crystal defects and of defects of fibrillar structure hardly affects the estimates of E which with sufficient precision can be obtained from consideration of alternation of crystalline and amorphous regions and their modification by taut tie molecules. In the former case, however, just these defects play the dominant role.

Besides the formation of new surface at the break one also observes a large number of chain scissions which show up in a decrease of average molecular weight [12, 13], appearance of free radicals detectable by ESR [4], and new double bonds detectable if IR [5]. The number of ruptured

chains based on the number of free radicals formed upon straining is many orders of magnitude higher than the maximum number of chains $A_f \beta n$ which can be ruptured at the fracture surface having an area A_f if there are n chains per unit area of crystal lattice. With $\beta = .1$ and $n = 5 \times 10^{14}$ cm^{-2} this number would be 5×10^{13} per cm^2 of fracture surface and independent of strained volume. Experiments, however, show that the number of ruptured chains is proportional to the strained volume and not to the single cross section, i.e., $R \sim 10^{17}/\text{cm}^3$. This leads to the conclusion that the chains are ruptured all over the sample during straining [16]. Since there is no catastrophic increase of chain rupture preceding the sample fracture and the unbroken part of the sample shows not decline of fracture strength during second run although no new chains are ruptured [4] one has to conclude that the fracture is not caused by chain rupture although both effects are closely connected.

A more detailed analysis of anisotropy of ESR absorption spectra [17] and of accessibility of radicals [12] shows that the chains are mainly ruptured in the amorphous layers. They are indeed the most strained tie molecules connecting the crystal blocks and thus each carrying a larger load than the unbroken chains [18].

The investigation of decrease of intrinsic viscosity [12, 13] yields a much larger fraction of ruptured chains than indicated by the number of radicals if one assumes random chain scission under the applied stress. One calculated on this basis 3.3×10^{18} in nylon 66 fibers, 1.9×10^{18} in poly(ethylene terephthalate) fibers, $.04 \times 10^{18}$ in drawn polypropylene fiber and $.10 \times 10^{18}$ in drawn polypropylene film [13]. ESR, however, yields only $.54 \times 10^{18}/\text{cm}^3$ in nylon 6 and $.08 \times 10^{18}\text{cm}^{-3}$ in nylon 66 fibers while the number of broken chains in PET, PP, and PE is hardly measureable (between .5 and $5 \times 10^{16}\text{cm}^{-3}$). This disagreement can be resolved by three considerations.

Firstly, the longer chains have a greater chance for rupture while the shorter chains are not sufficiently well anchored in the crystal blocks so that it is easier to pull them out of the crystal lattice than to break them. Hence the straining would primarily reduce the high molecular weight fraction of the same which affects more the weight or viscosity average of molecular weight than a random scission.

Secondly, one has to consider secondary chain rupture which does not increase the number of radicals [15]. The usually extremely unstable radicals formed by primary chain rupture can react with close-by chains by proton transfer. Thus the end-of-chain primary radicals are transformed into exactly the same number of center-of-chain radicals while the end-of-chains double bonds or new CH_3 groups are formed at the ends of originally broken chains. The covalent bonds adjacent to such a radical have a so much smaller bond energy that they are broken very much easier than the bonds in a saturated chain. Hence as a consequence of the formation of center-of-chain radicals a great many such secondary chain ruptures occur without any increase of radical population beyond that caused by primary rupture. The average molecular weight decreases more than one would calculate from the number of radicals. A much better indicator of the total number of broken chains is the increase of end-of-chain double bonds and CH_3 groups.

Thirdly, the radicals may be so short lived that only a small fraction of them remains detectable by ESR. The primary end-of-chain radicals are certainly of that type. They are almost instantaneously transformed into center-of-chain radicals which may be sufficiently stable for ESR observation. If a substantial fraction of them decays very rapidly one detects by ESR absorption a much smaller number of broken chains than were actually ruptured during sample straining even if no secondary rupture takes place.

All three effects may play a role in the apparent reduction of radicals below the number corresponding to the actual number of broken chains. As a consequence one has to assume for the latter a larger number than formerly deduced from ESR data but very likely also a smaller number than derived from viscometric data. Hence one can put the maximum number of broken chains in nylon 6, nylon 66 and PET equal to between 1 and $2 \times 10^{18}/cm^3$ and in PP and PE equal to between 1 and $4 \times 10^{16}/cm^3$.

These figures have to be compared with the number of t.m. per cm^3 of the fibrous material. If one assumes 10% of chains in the crystalline lattice acting as t.m. one finds $5 \times 10^{19}/cm^3$ t.m. in nylon with long period L = 10 nm and about half this number in PE with L = 20 nm. Hence in a broken sample one has ruptured between 2 and 4% of t.m. in the former and between .04 and .16% in the latter case. Hence their number is much too small to affect appreciably the elastic modulus of the strained material.

One has to conclude that those t.m. which are ruptured during tensile testing of the fibrous material are responsible only for a fraction of the observed elastic modulus and its reduction during straining close to break. The following up of chain rupture by ESR or IR, although very interesting by itself, does not yield the information about the actual damage of the sample which could help in predicting the failure either as function of stress or time. It only reveals a very important side effect during the extension of the sample under applied load but not the critical effect causing the failure.

Since the number of ruptured chains is so much smaller than the number of taut t.m. responsible for the elastic modulus of the fibrous material only a small fraction of them is so much strained that the covalent chemical bond is ruptured during the time of experiment [3, 5, 19, 20, 21]. Concurrent observation of microcrack formation of strained sample by SAXS supports the concept that chains are ruptured just at the areas where also microcracks are formed, i.e., at the weakest points of the fibrous structure, [22].

According to the microfibrillar model of fibrous structure these structural defects are primarily the ends of microfibrils where the axial connection by t.m. is interrupted or at least very reduced. As a consequence, under applied tensile stress the strain at these areas is larger than the bulk strain. Its time dependence is still a function of the efficiency of microfibril packing which determines the rate of sliding of their ends under applied load, (Figure 3). If originally the whole area is homogeneously strained each amorphous and crystalline layer at the end of the microfibril contains a certain amount of elastic energy proportional to the square of local strain. It can be released by sliding motion of the ends opposed by frictional resistance of interfibrillar boundaries. Such a motion will reduce the strain the more the closer one is to the end of the microfibril. The translation of the ends, however, is only possible by the formation of a hole which required a certain amount of surface free energy and the energy for fracture of the few tie molecules bridging the hole. If this

amount can be supplied by the energy release in the sections of the microfibril close to its end the hole will be formed. Otherwise, the process must wait for a higher strain. But at any sufficiently high strain the rate of sliding determines the time needed for formation of the hole *(nucleation time)*.

Additional support for this model of microcrack nucleation at structural defects of fibrous material by fibril and microfibril displacement comes from the fact that the rupture of covalent bonds in the aliphatic main chain of linear polymers occurs at an extremely high strain. According to quantum mechanics calculation by Boudreaux [31] the straining first extends the chains by straightening the chemical bond angle and only later by extending the bonds. Chain rupture occurs at $\varepsilon_b \sim 35\%$ which is so much above the strain to break of fibrous material that it can take place only in areas where some structural peculiarity of the sample drastically enhances the local strain, i.e., exactly at the point defects of microfibrillar structure.

The formation of a hole fractures the few tie molecules which are present at the end of a microfibril. They are subjected to a much higher stress concentration than inside the microfibrils with higher fraction of tie molecules per unit cross section. As a consequence of the hole the axial force has to be transmitted completely through adjacent microfibrils. Hence they are under higher tensile stress than before formation of the hole. The ensuing increase in strain enhances the rupture of t.m. in the amorphous layers in the immediate surrounding of the hole (lateral crack growth) and the sliding motion of fibrils (crack propagation along the boundary between adjacent fibrils with the fracture of interfibrillar tie molecules). One sees that the sliding motion of microfibrils and fibrils is the main cause of stress concentration on intrafibrillar and interfibrillar tie molecules, of the *formation* of primary microcracks at microfibrillar ends, and of lateral and axial *growth* of these cracks. In all cases the resulting higher stress concentration on the tie molecules in these areas as compared with tie molecules in the rest of the sample leads to their rupture.

The removal of such connectors eases the shear deformation and hence contributes to the progress of sample failure but does not determine it. Upon load release the sample will not retract to initial length because the sliding motion of fibrillar elements is not fully recoverable. Hence the microcracks do not close completely. In a second run less energy is required for extension and opening of the old microcracks which were only incompletely closed during strain recovery without any compensation for the ruptured tie molecules. That results in a smaller elastic modulus and a lower stress as function of strain. But as soon as the maximum strain of the first loading is reached the load-elongation curve is practically a continuation of the curve of first loading. The small deviations at this point, dependent on the rate of strain change, are a straightforward consequence of time requiring relaxation phenomena.

The lateral and axial growth of micronecks and their coalescence finally yield a critical size crack according to Rivlin-Thomas-Orowan-Griffith criteria. Recent investigations by Zhurkov and Kuksenko [22] lead one to believe that the ratio of average microcrack spacing and linear dimension is practically constant for as different fibrous substances as PE, PP, polyvinly chloride, nylon 6 while the microcrack concentration varies between 10^{12} and 10^{17} cm^{-3} and the microcrack diameter from 9 to 300 nm. This means that at such a moment the crack coalescence yields the critical

size cracks which propagate catastrophically through the cross section of the strained sample. As already mentioned the number of chains broken during this last step is unmeasureable small, below $10^{14}/cm^2$.

SHEAR DISPLACEMENT OF MICROFIBRILS AS THE MAIN MECHANISM OF PLASTIC DEFORMATION AND FAILURE

If the chain fracture is only a side-effect of sample deformation leading to failure and not the main cause of it one has to look for other effects which can be responsible for it. The formation of microcracks and their final coalescence to a crack of critical dimensions is certainly the most important step in the failure of axially strained fibrous material. As already mentioned, the microcracks seem to form at the weak spots of fibrous structure, i.e., at the ends of microfibrils. The main deformation leading to the opening of microcracks is the sliding displacement of microfibrils and fibrils. A rather similar type of plastic deformation takes place during neck propagation through the unoriented lamellar material (sliding motion of chains and crystal blocks inside the lamellae) and during homogeneous drawing of fibrous structure (sliding of microfibrils). The temperature dependence of neck propagation velocity at constant stress yields the same activation energy [23] as derived from the life-time of strained samples [4]. This indicates a similarity of the basic deformational mechanism in both cases.

The longitudinal displacement of microfibrils is opposed by the friction forces in the boundary between them. The force field along the z-axis of the crystal lattice of the polymer is derivable from the potential energy along this axis. Calculations performed for the orthorhombic lattice of polyethylene show an energy barrier about 20 kJ per CH_2-CH_2 mole separating the equilibria positions of the chains with a period c = 0.25 nm, [20]. Along a chain with 80 monomers in a straight section, corresponding to a long period L = 20 nm if one neglects completely the amorphous layer of thickness $(1-\alpha)L$ = 4 nm, one has an activation energy U of 40 kcal/mole for an axial chain displacement if the chain is displaced simultaneously as one unit.

This very likely is indeed the case since one knows that the long period growth of single crystals during annealing proceeds by exactly the same mechanism, i.e., by axial displacement of chains and not be a propagation of some crystal defect which would gradually shift the chain in axial direction by c or c/2 if the chain at the same time rotates by 180°, [25]. Also the temperature dependence of the second moment of NMR absorption of drawn PE as function of angle between the chain and magnetic field is explained by flip-flop motion of the chain by 180° and concurrent axial translation by c/2, [26]. In both cases the axial displacement of the strait section is a cooperative effect and not a consequence of a translation of a double kink through the chain which would achieve the same geometrical effect but with a completely different dynamics. The activation energy in such a case is the sum of energy barriers for the axial translation of all chain elements. Even if such a barrier is relatively small, 2.1 kJ/mole, the sum over the whole length of the straight section is quite large and becomes soon comparable with the binding energy of the C-C chemical bond which is about 300 kJ/mole. One has the feeling that the covalent bonds of the chain will break as soon as the frictional forces for chain translation require an activation energy higher than the bond energy.

The large value of activation energy for translational displacement of the whole strait section of linear macromolecule in a single step seems to qualify this model for the explanation of creep and failure under tensile load. The model has to explain the high activation energy, between 200 and 300 kJ/mole, of sample life time and the approximate constancy of this energy over the whole range of stress investigated, [27].

The constancy of apparent ativation energy yields a straight line for the plot of log t_b over the applied stress according to the Tobolsky-Eyring formulation of the life time t_b of covalent chemical bond, [28]

$$t_b = t\hat{o} \exp (U-\gamma\sigma)/kT \tag{3}$$

where γ is the activation volume of the bond, i.e., the product of bond extension $\delta\ell$ from equilibrium length to rupture and of the area A over which the load is concentrated on the bond. Equation 4 is a very rough approximation valid only in the case that the stress field does not change noticeably the location of minimum and maximum of potential energy.

For an isolated chain of polyethylene or nylon A is about 0.2 nm^2. The extension, however, depends on σ. It is the difference of bond length between the minimum and maximum of potential energy under consideration of the influence of the applied tensile stress σ. In any model of covalent force field, Lennard-Jones or Morse for instance, the energy difference between the minimum and maximum decreases with σ and so does the difference between the corresponding link lengths. By considering both effects one obtains for the log t_b curved lines convex to the abscissa. The curvature is not large but certainly of such a magnitude that it would be detectable even in the small range of observed t_b between 1 and 10^8s. The linearity of experimental data is hence one of the main objections against the concept that the rupture of covalent chemical bond determines the life time of strained polymer with fibrous structure.

The model of translation of fibrils as the main and the most time consuming step in plastic deformation of fibrous structure which finally leads to failure bases the frictional resistance on the sume of activation energies for the axial translation of the monomer $CH_2 \cdot CH_2$. The potential energy has very nearly a sinusoidal shape modified linearly by the effect of the tensile force F.

$$\phi = U_f (1-\cos(2\pi x/c))-Fx \tag{4}$$

$$\frac{U_{f\sigma}}{U_f} = \frac{\phi_{max}-\phi_{min}}{U_o} = (1-y^2)^{1/2} - \frac{y}{2}(\pi-2 \arcsin y)$$

$$y = cF/\pi U_f$$

$$t_b = t_o \exp (-U_{fo}/kT)$$

which makes the shift of maxima and minima by the applied force field much smaller than in the case of Lennard-Jones or Morse type potential energy of the covalent bond which has no maximum in the absence of external force field. The life time calculated for such periodic potential energy is much closer to the approximation by equation 3 and yields in the interval of observed life times a more straight line in the log t_b versus σ plot than the potential energy of the covalent chemical bond, (Figure 4).

The next question concerns the magnitude of activation energy for such sliding motion of microfibrils and fibrils. Even the best lateral packing of microfibrils is less perfect than the lateral packing of polymer chains in the crystal lattice. Hence the activation energy for axial translation of one monomer is substantially less than 2.1 kJ/mole calculated for axial translation in the crystal, i.e., against the force field of 4 adjacent chains. One can indeed assume that by translating one folded chain block against the adjacent block one is faced with the same energy barrier per monomer as in the case of translating one chain against another one with the distance between them a little larger than between two adjacent chains in the crystal lattice. The reduction to one neighbour reduces the barrier to 1/4 and the increase of distance to $(r_c/r_{mf})^6$. Here r_c is the average distance of chains in crystal lattice and r_{mf} the average distance of chains across the boundary between adjacent microfibrils. Both corrections may yield a reduction of U_a to $U_a/10$, i.e., to 200 J/mole.

The shear displacement of adjacent blocks demands the simultaneous crossing of the monomer barrier $U_a/10$ by all the monomers on the lateral surface A_{ac} or A_{bc} of the block. This number n is A_{ac}/ac or A_{bc}/bc with a = .74 nm, b = .49 nm, c = .25 nm if one considers only the (100) and (010) faces. Any area of the lateral surface is the produce of αL and the width of the microfibril. Without a substantial error one can put for the width 20 nm and assume the same value for L. With $\alpha = 0.8$ one obtains $n_{ac} = 1700$ and $n_{bc} = 2600$. The cumulative energy barrier in such a case is 360 and 550 kJ/mole, respectively, which is very high but not excessive. Any uneveness of the surface caused by some chain end or interfibrillar t.m. would reduce this value so much that the cumulative activation energy for the shear displacement of two adjacent blocks would be in the range of observed activation energies for rupture, i.e., between 200 and 300 kJ/mole.

Such a calcuiation of activation energy may serve as one possible guess about the elementary act of block displacement. It seems most likely that the estimate of reduction of U_a to $U_a/10$ is too extreme. If that is indeed the case one has such a tremendous energy barrier that other mechanisms have to be considered. One can imagine a propagation of a cooperative array of chain double kinks, i.e., of an edge dislocation through the boundary layer of adjacent crystal blocks which makes possible the shear displacement at a reasonable activation energy without requiring the instantaneous collective jump of all chain elements.

According to this concept that the shear displacement of adjacent blocks of microfibrils is the elementary step in deformation of fibrous structure the shear displacement of adjacent microfibrils must be made up of consecutive displacement of blocks. Between such elementary steps the amorphous layers are strained so much that they can transmit to the next block the force necessary for its axial displacement. Hence during this action the local stress and strain in amorphous layers are substantially larger than the bulk stress and the average strain of amorphous layers. This may lead to the rupture of the most strained t.m. inside the microfibrils which seems to be a necessity in order to explain the already mentioned large fraction of broken chains, [13].

In the cases of nylon 6 and 66 one has a larger energy barrier for axial displacement as a consequence of hydrogen bridges and a great many interfibrillar t.m. which may act as a lubricating medium replacing the crystal block to crystal block contact by a pseudoamorphous layer. This makes the sliding easier while the hydrogen bridges make it more difficult. A reduction of sliding resistance is also caused by smaller L, about 10 nm and small α, about 0.5, thus reducing the number of chain elements in the lateral surface of the block by a factor of 6.5 as compared with PE.

The sliding motion of microfibrils and particularly that of fibrils gradually opens the micronecks at the ends of microfibrils. Since they are mainly located on the outer boundary of fibrils they tend to coalesce along this boundary which requires separation of the less tightly packed microfibrils. This finally leads to longitudinal cracks and almost complete separation of fibrils which at the end are fractured individually as nicely demonstrated on the fracture of an experimental polyamide, [29]. But the full transfer of load on adjacent microfibrils as soon as a microcrack forms overstrains the affected microfibrils so much that they tend to break by a crack cutting through at least one amorphous layer close to the microcrack under consideration. Any such radial extension of microcrack additionally increases the load on the still intact microfibrils and thus accelerates the crack expansion. Both effects, axial and radial growth of microcrack, and the coalescence of growing cracks finally yield a critical size crack leading to sample failure.

The chain rupture takes place during the longitudinal straining of amorphous layers of all microfibrils, during the opening of microcracks at the ends of microfibrils, and during their lateral and axial growth and coalescence. In spite of their large number the reduction of the total number of t.m. is so small that it can explain only a small fraction of the reduction of load as function of elongation observed in a second loading. This was slowly recognized even by the most persistent protagonists of the concept that in the tensile experiment one breaks just those t.m. which are responsible for the axial mechanical properties, [30]. One certainly breaks the most strained t.m. located primarily in and around the defects of microfibrillar structure which, however, are not the main source of elastic and plastic resistance to deformation. This resides in the more ideal sections of microfibrillar structure.

Upon removal of load the sample contracts but usually does not assume its length before the experiment. Even after a long waiting time it usually does not recover completely. In a second run a lower load or stress is needed for the same elongation than in the first run up to the maximum strain of the first run. This is a consequence of the fact that the reopening of the incompletely closed microcracks requires a smaller stress than the initial microcrack formation. One certainly has not to rupture the t.m. which in the virgin sample have connected the crystal blocks across the amorphous layer of the microcrack before its opening. More important seems to be the fact that as indicated by the incomplete length recovery a detectable fraction of longitudinal displacement still remains in the unloaded sample so that the microcracks do not close completely. The free surface in the case of still open voids and the decreased density in the not ideally closed voids reduce the energy requirement for reopening during the second run. Less surface free energy is to be supplied in both cases and less resistance to straining takes place in the second case. The remains of longitudinal voids also make the axial displacement substantially easier. One has indeed a great many effects not detectable by any method based on the registration of broken chains which affect the mechanical response of fibrous material.

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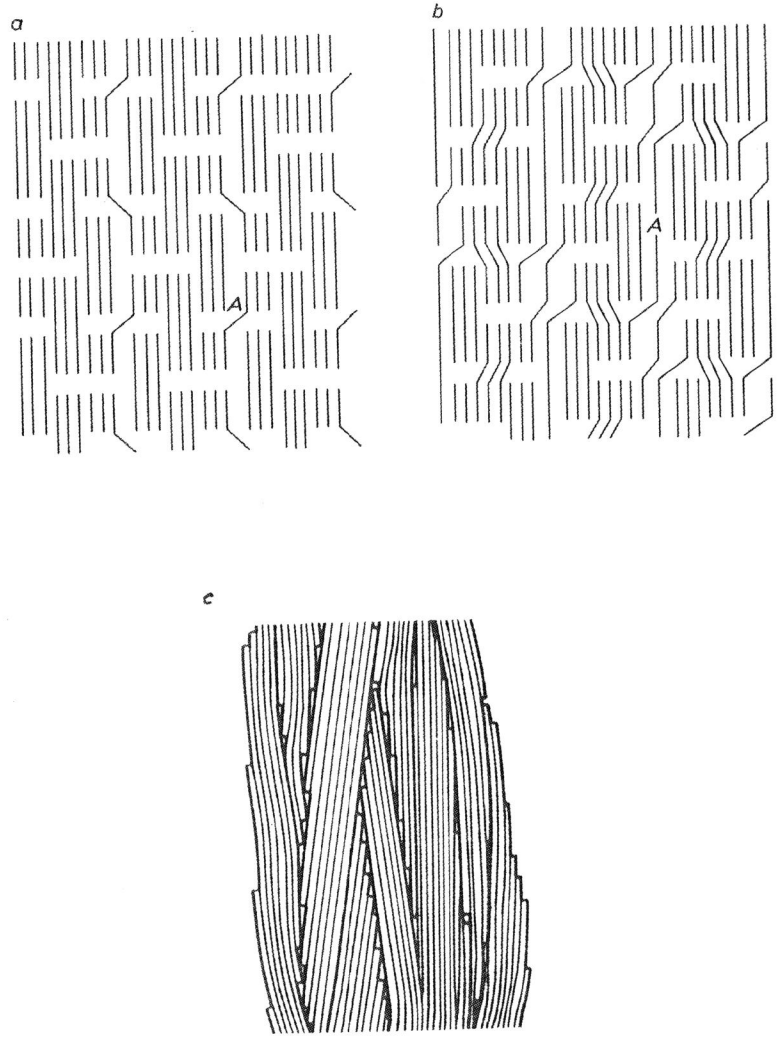

Figure 1 Schematic Model of Microfibrils:
(a) in PE with very few Interfibrillar t.m.;
(b) in nylon 6 with a great many such t.m.; and
(c) of Fibrillar Structure with the Point Defects at the ends of each Microfibril

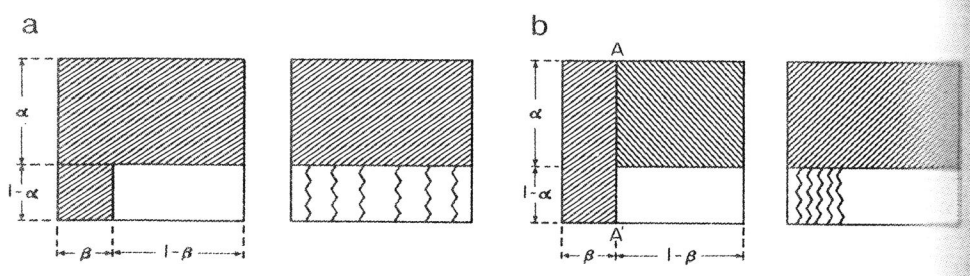

Figure 2 Schematic Model for the Calculation of Elastic Modulus of Fibrous Structure:
(a) Takayanagi's Model Valid for Uniform Distribution of Taut Tie Molecules over the Amorphous Layer (Equation 1);
(b) Extreme Case of Complete Bundling of t.t.m. on One Side of the Amorphous Layer (Equation 2) and no Cohesion Along AA

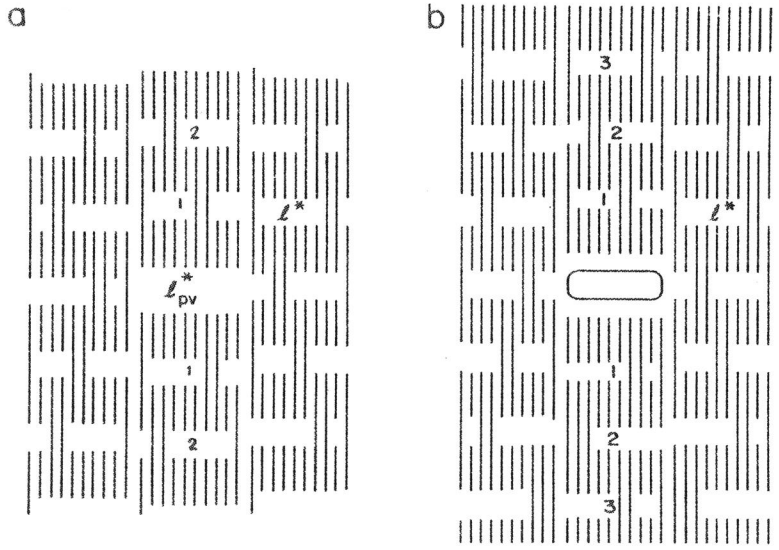

Figure 3 Sliding Motion of the End Sections of Microfibril Leading to Microcrack Formation at the Point Vacancy with Thickness ℓ_{pv}:
(a) The Amorphous Layers 1, 2, ..., at the Ends are Strained as much as the Interior Parts of Microfibrils and Hence Contain a Substantial Amount of Potential Energy;
(b) The Sliding Motion of the Ends Reduces the Strain (to zero in Layer 1) and Hence Releases Gradually Decreasing Amounts of Potential Energy as One is Farther Away from the Ends. The Energy Release Supplies the Surface Energy Requirement of the Hole.

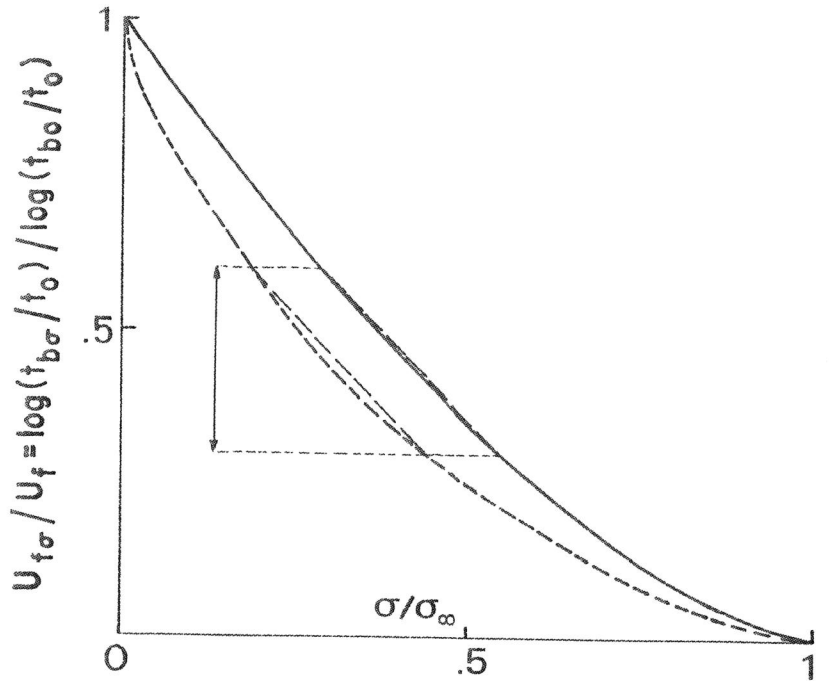

Figure 4 The Normalized Log t_b Versus Reduced Stress σ/σ_∞ Plot for Lennard-Jones Type (Broken Line) and Sinusoidal Potential Energy (Full Line), (Equation 4). The Ordinate is the Relative Effective Activation Energy $U_{f\sigma}/U_f$. The Abscissa is Normalized in such a way that the Effective Activation Energy $U_{f\sigma}$ Disappears at $\sigma = \sigma_\infty$. The Life Time in this Limited Case is $t_o = h/kT = 1.6 \times 10^{12}$s. The arrows span the Experimentally Observed Life-Times.