# Effects of Enzymes and Gamma Irradiation on the Tensile Strength and Morphology of Poly(*p*-dioxanone) Fibers

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## Synopsis

The objective of this study was to examine how and to what extent a new degradable polymeric fiber, poly(*p*-dioxanone), used as a surgical suture material, degrades in the presence of enzymes and after $\gamma$-irradiation. The degradation of the fiber was studied mechanically using an Instron and morphologically by SEM. Both esterase and trypsin enzymes and their corresponding buffer controls were used. The fibers were $\gamma$-irradiated at the dosages ranging from 0 to 20 Mrad, immersed in the solution for up to 70 days, and then removed for tensile strength and morphological examinations. It was found that $\gamma$-irradiation alone lowered the tensile strength of PDS fibers and made them more susceptible to hydrolysis. Esterase and trypsin did not accelerate the hydrolytic degradation of this fiber to any significant level. Both $\gamma$-irradiation and enzymes influenced the gross morphological characteristic of PDS fibers when they were subjected to hydrolysis. The most important morphological observations were the formation of surface cracks and chips on the fibers and the subsequent peeling of the chips. Enzyme-treated PDS fibers exhibited similar morphological findings but the size of the chips was smaller. The morphological observations of PDS fibers were consistent with the tensile strength data.

## INTRODUCTION

Polymeric materials have been used in medical and surgical applications for 30–40 years, often in situations where there is intimate contact with living tissues. Like polymers for any nonmedical use, they may be susceptible to degradation, but the conditions under which this occurs and the kinetics of the reactions are extremely variable, depending on the degradation process. Those processes which involve the absorption of some kind of energy to cause disruption of primary covalent bonds to form free radicals, such as high energy radiation, or thermal oxidation, should not occur within the human body. On the contrary, the existence of both the large amount of aqueous extracellular fluids and the various types of enzymes in the human body environment suggest a rather unique mode of degradation for those biomedical polymers.

Pure hydrolysis and enzymatic catalyzed hydrolysis are the two most important ways which biomedical polymers degrade within the body. A classical example of the latter is catgut sutures.[1] However, because of the known substrate specificity of enzymes, their role in the biodegradation of synthetic high molecular weight polymers has remained unclear and controversial. The availability of the synthetic absorbable sutures, both polyglycolic acid (PGA) and poly(glycolide–lactide) copolymer, provides the center of this confusion and discussion.

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Several investigators have concluded that the degradation of these synthetic absorbable sutures is a simple hydrolytic process.[2,3] Williams and Chu, however, have shown that degradation of these sutures *in vitro* condition can be accelerated in the presence of certain enzymes at the pH range where these enzymes exhibit their maximum activity.[4–7] Furthermore, it has been shown very recently that synthetic high molecular weight polymers, which are initially resistant to enzymatic degradation, could become prone to enzymatic attack after altering their physical and chemical structures.[7]

The recent availability of a new synthetic absorbable polymer, poly(*p*-dioxanone), which differs from PGA in terms of chemical and physical structures, could provide another opportunity to examine the problem of enzymatic degradation of synthetic high polymers. Thus, the objective of this study was to examine how and to what extent this new absorbable polymeric fiber differs from existing synthetic absorbable polymers mechanically and morphologically, particularly its degradation behavior in the presence of enzymes. At the same time, the effects of $\gamma$-irradiation on this degradation were studied.

## EXPERIMENTAL

Poly(*p*-dioxanone), in the form of monofilament surgical sutures of size 2-0 (PDS), supplied by Ethicon, was used in this study. Two enzymes were used: esterase and trypsin obtained from Boehringer and Sigma, chosen on the basis of previous work.[4,6] Their properties are given in Table I. The pH levels were chosen where the corresponding enzymes exhibited their maximum activity. PDS fibers, obtained from the manufacturer unopened, were subjected to $Co^{60}$ (Vickard MK II Cobalt source) $\gamma$-irradiation at a dosage rate of 0.15 Mrad/h for the total dosages ranging from 5 to 20 Mrad. Oxidative degradation of PDS during $\gamma$-irradiation was kept at a minimal and insignificant level by the double-sealed packaging of the fiber material by both plastic film and aluminum foil, which reduced diffusion of oxygen into the fiber specimen. The irradiated PDS fiber specimens were then incubated in the solutions of these enzymes in their appropriate buffers for various periods of time at 37°C. PDS fiber specimens were also incubated in the corresponding buffer solutions without enzymes as controls. Attempts were also made to use concentrations which gave a reasonable degree of activity over a measurable time period by adopting the same procedures as given previously.[4,6]

The degradation of PDS fibers was determined mechanically by using an Instron universal testing unit and morphologically by scanning electron microscopy.

TABLE I
Information of Enzyme Studies

| Enzyme | Source | Form | Specific activity | Concentration | Buffer | pH |
|---|---|---|---|---|---|---|
| Esterase | Hog liver | Suspension in 3.2*M* ammonium sulfate solution | 100 U/mg | 10 $\mu$L/mL (0.1 mg/mL) | 0.1*M* tris | 7.5 |
| Trypsin | Bovine pancreas | Lyophilized | 33 U/mg | 1 mg/mL | 0.1*M* tris | 8.0 |

The gauge length and crosshead speed were 25 mm and 30 mm/min, respectively. Five specimens of each group were tested and the mean tensile breaking strength in kilograms was calculated at 95% confidence limit. The mean percentage of original strength (MBSR) was also calculated on the basis of unirradiated and unhydrolyzed PDS fibers. Attempts were made to obtain molecular weight of PDS samples by gel permeation chromatograph. Due to the problem of solubility, the data lacked consistency and hence were not used as an indicator for PDS degradation by $\gamma$-irradiation.

For the morphological observation, the same fiber specimens of a predetermined length were mounted on holders made of clear acrylics during the immersion period. The use of these holders could minimize the artifacts introduced during the handling of the specimens for SEM observation. This is particularly important at the later stage of degradation because the specimen was so fragile that the preparation for SEM observation could destroy the original morphology. For longitudinal observation the square-shaped holder had a spherical hole of diameter 5 mm in the center and the two ends of the fiber specimen were snapped on two groves carved on the two opposite edges of the plastic holder. For a cross-sectional view, a hole of the same diameter of the monofilament fiber was drilled on a solid, clear square plastic holder and the specimen was slid into the hole. Any protruded end out of the plane of the holder was cut by a surgical scalpel. Both longitudinal and cross-sectional holders containing the fiber specimens were immersed in the same vials containing the specimens for mechanical strength studies. After each predetermined period of immersion, the holders were removed and dried in a desiccator for 7 days, then sputtercoated with gold. A Joel 35C scanning electron microscope was used to examine the morphological change of the fiber specimens.

## RESULTS

Figures 1 and 2 summarize the effect of both esterase and trypsin on the mean percentage of tensile breaking strength retention (MBSR) of PDS monofilament fiber at various levels of $\gamma$-irradiation and duration of immersion.

In general, no significant enzymatic effect on the degradation of unirradiated and irradiated PDS was observed, particularly at the higher radiation dosage level (i.e., 10 Mrad and above). Slight differences between the enzyme and its corresponding buffer solution were observed at the lower dosage level and early stage of degradation (7 days). This difference in degradation became increasingly small as the extent of immersion increased beyond 7 days. Furthermore, both esterase and trypsin exerted the same order of influence on the degradation of PDS fibers over the periods of immersion (0–28 days) and levels of dosage (0–20 Mrad) studied, as shown in Figure 3.

It was also found that the resistance of these fibrous materials to $\gamma$-irradiation depended on the dosage and extent of immersion. The degree of $\gamma$-irradiation degradation in the unhydrolyzed PDS fibers in terms of MBSR is not proportional to the dosage of radiation. For example, the losses of about 7%, 25%, and 64% of MBSR at 5, 10, and 20 Mrad, respectively, were observed. Thus the greatest loss of tensile strength of unhydrolyzed PDS fibers was observed between 10 and 20 Mrad.

This relative smaller $\gamma$-irradiation effect at the lower dosage as observed in

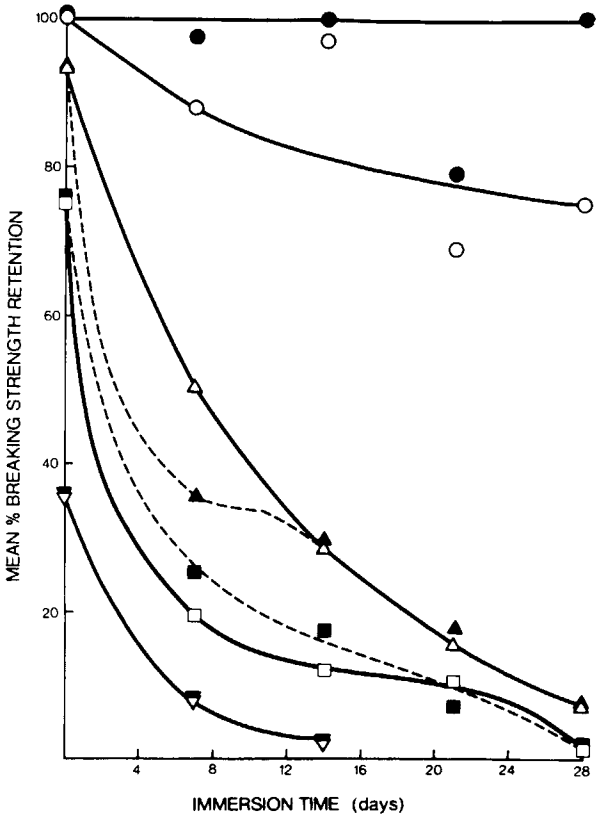



Fig. 1. The effects of $\gamma$-irradiation and esterase enzyme on the degradation of PDS monofilament fibers expressed in terms of mean percent tensile breaking strength retention: (▲, ▼, ●, ■) buffer controls; (△, ▽, ○, □) esterase treated; (○) 0 Mrad; (△) 5 Mrad; (□) 10 Mrad; (▽) 20 Mrad.

the unhydrolyzed PDS fibers does not apply to the hydrolyzed PDS fibers. For example, the hydrolyzed PDS fibers at the lower $\gamma$-irradiation dosage (i.e., 5 Mrad) lost strength significantly more than the unhydrolyzed PDS fibers at the same dosage level (63% loss in 5 Mrad buffer hydrolyzed vs. 7% loss in 5 Mrad unhydrolyzed). This quicker loss of strength in the irradiated PDS specimens became even more pronounced as the extent of hydrolysis proceeded. Hence, the difference in MBSR between the hydrolyzed unirradiated and irradiated PDS fibers increased with an increase in the duration of hydrolysis. A similar effect was observed in the trypsin and buffer control solutions. Thus, $\gamma$-irradiation at the level as low as 5 Mrad was sufficient to significantly increase the susceptibility of PDS fibers to hydrolysis.

The morphological changes of PDS fibers as a result of $\gamma$-irradiation, hydrolysis and enzymes are shown in Figures 4–6. In the absence of enzymes and hydrolysis (Fig. 4), the micrographs indicated that the irradiation alone did not drastically alter the morphology of PDS fibers, though a few surface cracks perpendicular to the longitudinal direction of the fiber were observed at 10 and 20 Mrad levels. A significant difference in morphology, however, was found when the irradiated fibers were subjected to hydrolysis in both the buffer control and

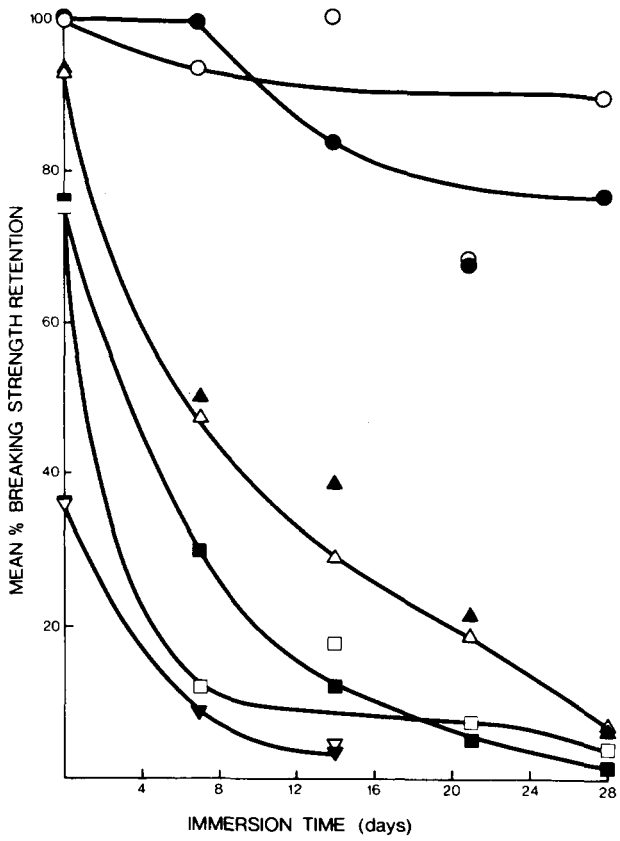



Fig. 2. The effects of γ-irradiation and trypsin enzyme on the degradation of PDS monofilament fibers expressed in terms of mean percent tensile breaking strength retention: (▲, ▼, ●, ■) buffer controls; (△, ▽, ○, □) trypsin-treated; (○) 0 Mrad; (△) 5 Mrad; (□) 10 Mrad; (▽) 20 Mrad.

enzyme solutions. For example, both longitudinal and circumferential cracks started to appear as early as 21 days hydrolysis in the 10 Mrad PDS fibers. The longitudinal cracks were parallel to each other and more severe and larger in size than the circumferential cracks. As hydrolysis proceeded further, the cracks propagated in both longitudinal and circumferential directions and eventually chips of either rectangular or square shapes of various sizes started to peel off and separate from the underlying layers of the fiber. On several occasions, the peeling process resulted in a rolling of a rectangular chip into a spool shape that left a clear smooth track on the underneath surface of the fiber. The thickness of the chips ranged from 3 to 5 μm, while the width of the chips ranged from 10 to 60 μm, depending on the dosage level as well as the extent of hydrolysis. The unirradiated PDS fibers, however, did not show any peeling of surface cracks even at the longest period of immersion we conducted (i.e., 60 days). For the irradiated specimens, the fibers eventually disintegrated either through a circumferential scission into smaller fragments and/or longitudinal fragmentation, as shown in Figure 6.

In the presence of enzymes, the morphological observations of PDS fibers were generally similar to those in the absence of enzymes, except the extent of hy-

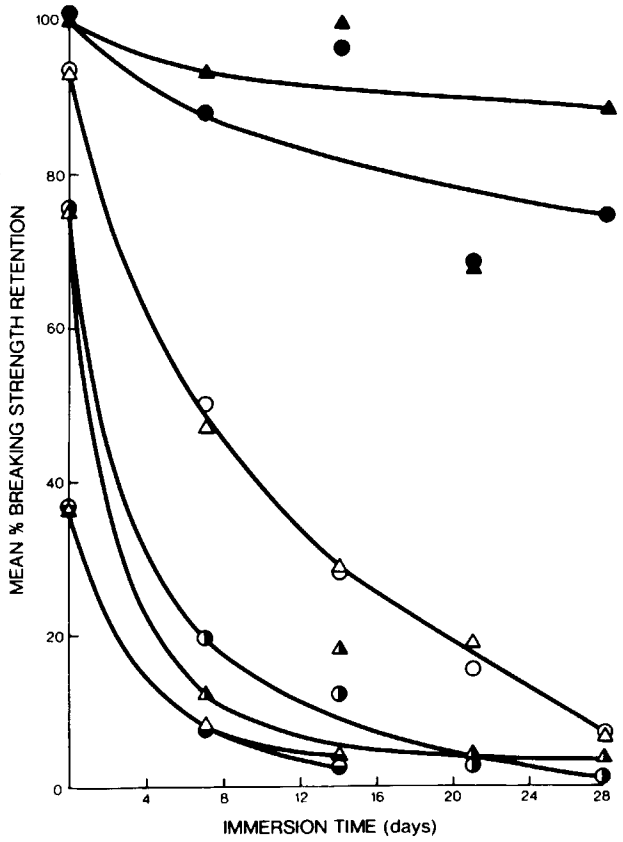



Fig. 3. A comparison of the effects of esterase and trypsin enzymes on the degradation of PDS monofilament fibers irradiated at various dosage of γ-irradiation: (▲, △, ▲, ▲) trypsin; (●, ○, ◑, ◒) esterase; (▲, ●) 0 Mrad; (△, ○) 5 Mrad; (▲, ◑) 10 Mrad; (▲, ◒) 20 Mrad.

drolysis and level of dosage which were required to show the surface cracks formation were different. Figure 5 illustrates this.

In the unirradiated but enzyme treated specimens, a few surface cracks of longitudinal shape appeared as early as 28 days of hydrolysis. This was not observed in the same specimens immersed in the corresponding buffer control solution.

In the irradiated and enzyme-treated specimens, massive surface cracks were observed in the 10-Mrad PDS fibers at 21 days in enzyme solution. As the hydrolysis proceeded beyond 21 days, at this level of radiation dosage, the specimen started to narrow at some points along the fiber as found at 28 days. Further hydrolysis of this fiber exhibited a morphology of a pine tree with the chips extended out along the trunk of the fiber. This treelike morphology was also found on the 20 Mrad specimens.

Another unique morphological feature of PDS fibers in the presence of enzymes was that the size of the chips were generally smaller than that of the same fiber in the absence of enzymes when compared at the same extent of the hydrolysis and level of radiation dosage (compare Fig. 4 with Fig. 5). Chip size greater than 30 $\mu$m were not frequently found in the enzyme-treated PDS fibers.

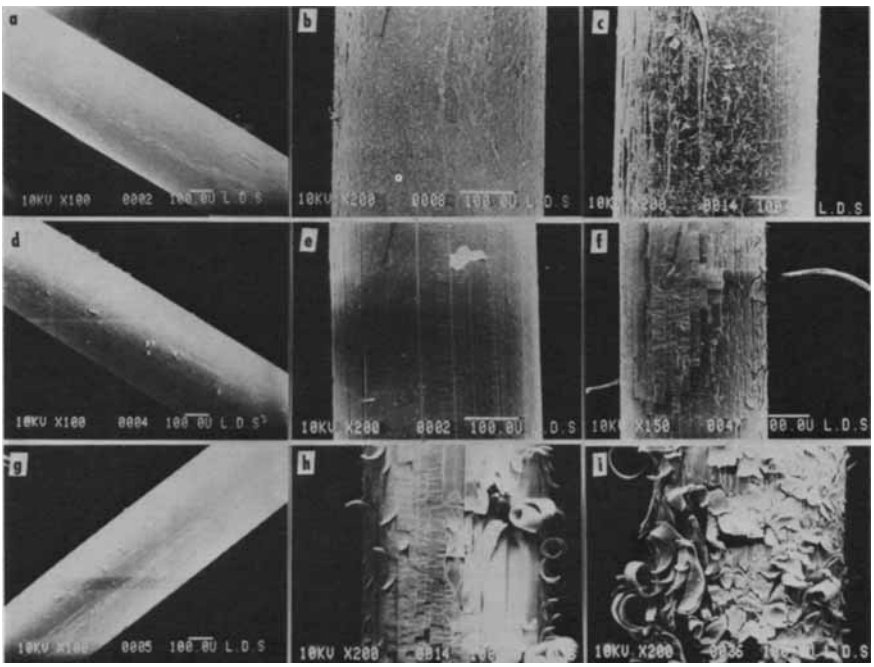


Fig. 4. Scanning electron micrograph of PDS monofilament fibers irradiated at various levels of $\gamma$-irradiation and immersed in tris buffer of pH = 7.5 at various extents: (a) 0 Mrad, 0 day; (b) 0 Mrad, 21 days; (c) 0 Mrad, 28 days; (d) 10 Mrad, 0 day; (e) 10 Mrad, 21 days; (f) 10 Mrad, 28 days; (g) 20 Mrad, 0 day; (h) 20 Mrad, 21 days; (i) 20 Mrad, 28 days.

Because of this size difference, the peeling mode of the chips of the enzyme-treated fiber was also different from the untreated ones.

## DISCUSSION

The morphological observations were consistent with the tensile strength data. The accelerated decrease in strength with the increasing level of radiation dosage in the unhydrolyzed PDS fibers was parallel to the increasing formation of circumferential surface cracks on the highly irradiated PDS fibers. These surface cracks were apparently responsible for the faster loss of tensile strength in the irradiated but unhydrolyzed specimens, and made the PDS fibers more susceptible to subsequent hydrolytic degradation.

It is believed that the arrangement of the surface cracks, their orientation on the filaments, and the direction of crack propagation could provide very useful information for determining the mechanism of hydrolytic degradation in this class of fibrous materials.

Similar observations of surface cracks on the hydrolytically degraded polycaproamide and polyglycolic acid have also recently been reported,[8,9] although the pattern of the cracks on the PDS fibers was quite different from those previously reported.

Research on semicrystalline polymers has given rise to many models of fiber structure; examples include the fringed micelle fibrillar model suggested by Hess, Mahl, and Guter,[10] the string model suggested by Statton,[11] the paracrystalline

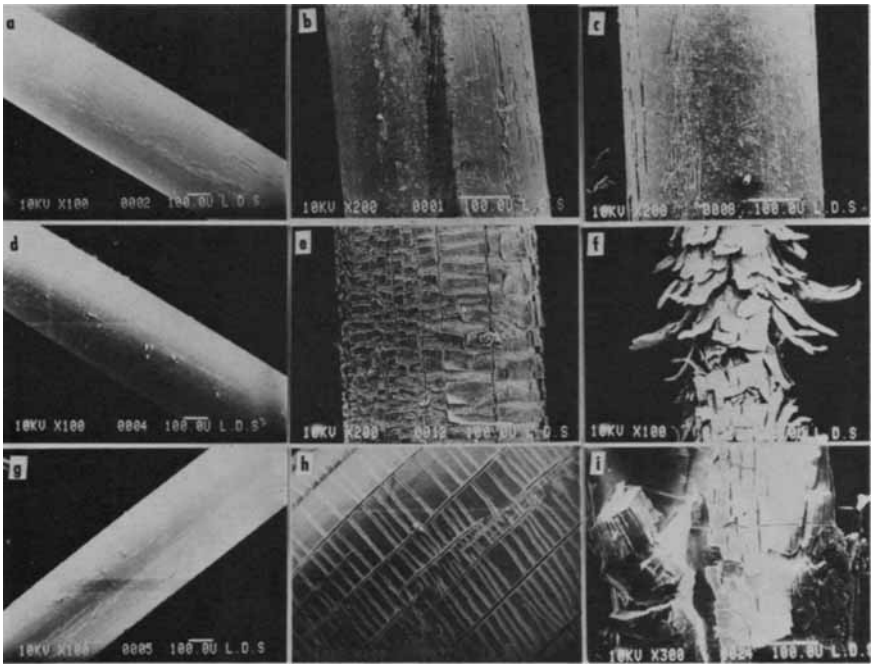


Fig. 5. Scanning electron micrograph of PDS monofilament fibers irradiated at various levels of $\gamma$-irradiation and immersed in esterase buffer solution at various extents: (a) 0 Mrad, 0 day; (b) 0 Mrad, 21 days; (c) 0 Mrad, 28 days; (d) 10 Mrad, 0 day; (e) 10 Mrad, 21 days; (f) 10 Mrad, 42 days; (g) 20 Mrad, 0 day; (h) 20 Mrad, 14 days; (i) 20 Mrad, 28 days.

state proposed by Bonart and Hosemann,[12,13] and the microfibrillar model proposed by Peterlin.[14]

The microfibrillar model of fiber structure was recently used to depict the mechanism of crack formation and crystallinity change on polyglycolic acid upon hydrolytic and $\gamma$-irradiation degradation.[9,15] It is believed that degradation proceeds through two main stages: in the amorphous region and then in the crystalline regions. When the material is immersed in an aqueous solution, the water molecules that diffuse through the polymer accommodate themselves readily in the amorphous zone, but not at all in the crystalline zone.

Because of this difference, hydrolytic degradation starts in the amorphous regions as the tie-chain segments, free chain ends, and chain folds in these regions degrade into fragments. As degradation proceeds, the size of the fragments reach the stage where they can be dissolved into a buffer medium. This dissolution removes the fragments from the amorphous regions and a loss of material results. As sufficient amounts of chain segments in these regions are removed, the spaces originally occupied by chain segments become vacant and large enough to be visible as cracks by SEM.

The cracks are believed to be initiated on the surface of the fiber and then grow into the interior of the fiber. This is because alkaline hydrolysis of a fiber has been reported to be a surface phenomenon.[16] As microcracks form on the surface of fibers, more hydroxyl ions penetrate into these areas and the microcracks propagate more deeply into the bulk of the fibers, until the cracks cut through

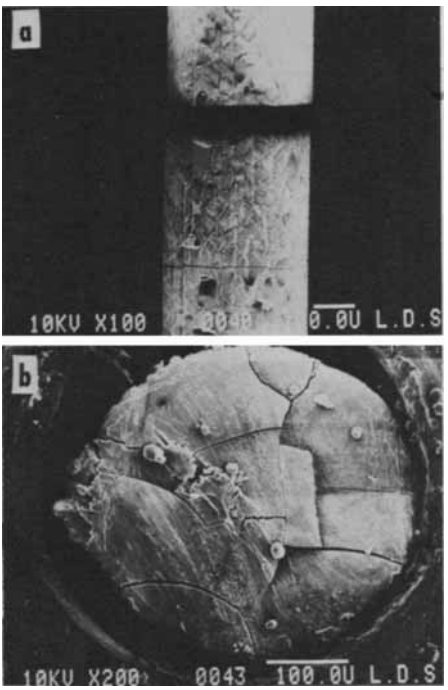



Fig. 6. Scanning electron micrograph of PDS monofilament fibers irradiated at 20 Mrad and immersed in tris buffer of pH = 7.5 for 28 days: (a) longitudinal view and circumferential cracks which resulted in the fragmentation of the fiber are evident, (b) cross-sectional view of the same fiber.

the whole fibers and scissions result. Similar patterns of crack formation have been found from undrawn and drawn nylon 66 filaments treated in oxygen–water systems.[17] Stress concentration, due to crystallization of smaller molecules produced during the oxidative degradation, was suggested as a possible cause of the cracks.

The circumferential propagation of a crack is always continuous and in a direction perpendicular or close to perpendicular to the fiber axis. An examination of the microfibrillar model clearly indicates that amorphous domains of the adjacent microfibrils are located at a common cross-sectional plane normal to the fiber axis. This suggests that as a microcrack forms at one point of the filament surface (on the outermost microfibrils), it will propagate circumferentially around the fiber and deeply into the fiber along this common cross-sectional plane, where the amorphous domains of adjacent microfibrils are located. As hydrolysis proceeds, the microcracks cut through the fiber along this common plane and result in relatively smooth and sharp scissions along various parts of the fiber. The SEM micrographs at 28 days of immersion for 20 Mrad specimens (Fig. 6) show these sharp and smooth cross-sectional scissions.

Two rather interesting phenomena to note at this stage are the formation of severe, deep, and parallel longitudinal cracks and a peeling off of the chips observed in the irradiated and hydrolyzed PDS fibers.

Longitudinal cracks were observed in both the enzyme and buffered treated irradiated PDS fibers but not in the hydrolyzed but unirradiated specimens.

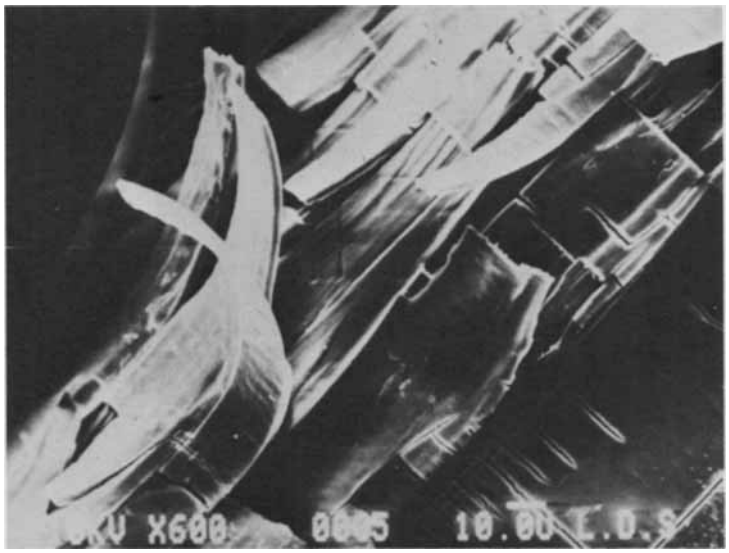


Fig. 7. Scanning electron micrograph of PDS monofilament fibers. The microfibrils beneath the skin layer are evident (20 Mrad, 14 days in buffer control).

These cracks were nicely parallel to each other and more severe than the circumferential ones. The width of the two adjacent longitudinal cracks (>10 $\mu$m) was too large for the size of a microfibril. It is believed that the longitudinal crack formation was due to the dissolution of disordered polymer chains and chain segments which have been excluded from and located in the lateral position of the ordered crystalline lamellae. These amorphous regions were sandwiched between two adjacent fibrils.

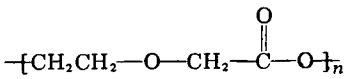
From this morphological observation, the structure of a fiber can be briefly elucidated as follows. The basic components of a fiber are microfibrils in the order of 100 Å width which, in turn, aggregate into macrofibrils. A similar aggregation of ultrafibrils into the smectic character of the paracrystalline state during the fiber manufacturing (i.e., hot stretch) was also reported by Hosemann before.[13] These macrofibrils are separated from the adjacent one by amorphous layers located in the lateral side of the macrofibrils. Hundreds of parallel macrofibrils then group together into a final fiber form. The size of the macrofibrils is expected to depend on the processing conditions such as drawing. When the fiber, with these layers of structure, is treated by $\gamma$-irradiation and hydrolysis, the parallel amorphous regions between the two adjacent macrofibrils degrade and dissolve away and become large enough to be visible as longitudinal cracks by SEM. The reason why the longitudinal cracks are more severe than the circumferential ones is not clear at this stage. It could be that the lateral amorphous regions between the two adjacent macrofibrils extend into the interior of the fiber deeper than the amorphous regions alternately located between two crystalline lamellae.

The concept that a macrofibril consists of a number of microfibrils can be illustrated by Figure 7. Beneath the peeling layers, an array of microfibrils of the size ranging from several hundred to thousand angströms were evident. This microfibrillar structure was not apparent on the peeling layer, which is the ou-

termost skin of the fiber. This difference in morphological appearance seems to be attributed to the so called skin-core effect. Since the polymer chains on the skin layer are exposed to the ambient environment first during the melt spinning of the fiber, they crystallize faster than the internal polymer molecules. They experience less shear stress in the molten stage, and the formation of microfibrils are thus less apparent than the inner polymer molecules.

The observed morphological findings on PDS fibers resulted also from the effect of $\gamma$-irradiation. $\gamma$-Irradiation of fiber-forming materials can result in simultaneous chain scission and crosslinking. Which of these will characterize the reaction depends on several factors, including the chemical structure of materials to be irradiated, the amount of dosage, the rate of dosage, the environment of the material during irradiation, and the heat of polymerization.[18] The orientation of long chain molecules has also been reported to have some influence on the direction of the overall radiation reaction, and was due to the differences in chain mobility.[19]

According to the constituents of the repeating unit and the observed reduction in mechanical properties of PDS upon $\gamma$-irradiation, the predominant effect of $\gamma$-irradiation on PDS fibers is thought to be scission across the dosage range studied. The susceptibility of this fibrous polymer to $\gamma$-irradiation chain scission must be attributed to the inherent chemical structure of the repeating unit. The basic structural unit of PDS consists of one ester and one ether functional group as shown below:



It has been demonstrated previously that the presence of a saturated aliphatic ester group, such as the one shown above, would make the polymer chain more susceptible to chain scission than crosslinking upon $\gamma$-irradiation.[19] Previous experimental results on PGA fibers demonstrate the susceptibility of a saturated aliphatic ester group to $\gamma$-irradation degradation further.[9] This is attributed to the weakened bond between acyl oxygen and the methylene group resulting from the ester bond resonance. Chain scissions will occur in these already partially weakened bonds when irradiated. In addition to the ester linkage, the repeating unit of PDS fibers also contains an ether linkage in the main chain backbone. It is well known that the incorporation of oxygen atoms into the main chains leads to a significant increase in the probability for main chain scissions.[20] This susceptibility toward chain scission is also influenced by the presence of the number of $CH_2$ groups in the ether linkage or the relative concentration of the oxygen. For example, poly(methylene oxide) undergoes exclusive chain scission,[21] while poly(tetramethylene oxide) undergoes chain scission and crosslinking.[22] This lower ratio of chain scission to crosslinking in the poly(tetramethylene oxide) was attributed to the fact that the C—O/C—C ratio is lower in this polymer than in polymethylene oxide.[23] The ethylene oxide portion of the PDS repeating unit has the content of ether linkage falling between methylene and tetramethylene oxides. Thus, PDS would be expected to undergo mainly but not exclusively chain scission. In the absence of oxygen, however, poly(ethylene oxide) will gel when irradiated at the dosage of $4.5–7.5 \times 10^4$ rad.[24]

The scission of polymer chains will be more pronounced in the amorphous

regions than in the crystalline regions. This might be due to the so-called "cage effect."[23] The crystalline regions of the microfibrillar model act as effective cages because of the dense and compact packing of immobile chain segments. The trapped free radicals can recombine and reduce the number of effective scissions. Therefore, $\gamma$-irradiation would result in more chain scissions in the amorphous regions than in the crystalline domains. This additional degradation (due to $\gamma$-irradiation) of the chain segments located in the amorphous regions would also reduce the degree of long-chain entanglement in the amorphous domains before immersion, and result in a more open amorphous structure than in unirradiated segments. This effect makes the chain segments in these regions more accessible to the hydrolytic species. Thus, the specimens after $\gamma$-irradiation become more susceptible to hydrolysis. After immersion, the observed earlier appearance of surface cracks on PDS fibers exposed at high radiation dosages demonstrates this point of view.

The earlier appearance of surface cracks was also found in the enzyme-treated PDS fibers, particularly in the unirradiated specimens. The esterase enzyme must have the capability to accelerate the hydrolytic degradation of these specimens. This enzyme accelerated the appearance of surface cracks in the unirradiated PDS fibers and is consistent with the observed slightly faster loss of tensile strength in the esterase-treated unirradiated PDS specimens, as shown in Figure 1.

In the irradiated PDS specimens, however, no statistically significant enzymatic effect ($p < 0.01$) was found, as demonstrated in both tensile strength and morphological observation. The SEM pictures revealed that the presence of enzymes in the irradiated PDS fibers only increased the density of surface cracks. In other words, a greater number of cracks were shown in a unit area than in the irradiated PDS specimens in an absence of enzymes. Thus, enzymes made the chips smaller in size, which appeared not to affect the tensile strength of the fibers to any significant detectable level. The insignificant influence of enzymes on the irradiated PDS fibers, particularly at the higher level of radiation dosage, is probably attributed to the fibrous structure being degraded to such an extent that buffer solutions alone produced very significant degradation without the assistance of the enzymes.

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