# Biocompatibility of Delrin 150: A creep-resistant polymer for total joint prostheses

**James S. Fister,* Vincent A. Memoli,† Jorge O. Galante,* William Rostoker,* and Robert M. Urban***

*Rush-Presbyterian-St. Luke's Medical Center, *Department of Orthopedic Surgery, †Department of Pathology, Chicago, Illinois 60612*

A thermoplastic polymer, Delrin 150 (polyoxymethylene homopolymer), with creep resistance ten times that of ultrahigh-molecular-weight polyethylene, is used as a material for total joint protheses. A study was made of the local and systemic host response to this polymer when implanted in three different mammalian species. 316 LC stainless steel was used as a control. The materials were implanted into muscle and bone as solid cylinders. A total of 446 samples were implanted into 74 animals. The duration of implantation ranged from 2 weeks to 2 years. A semiquantitative evaluation of local tissue reaction was performed. For each implant, 16 histological criteria were graded for severity of host tissue reaction. The liver, spleen, kidneys, pancreas, and lungs from each animal were also studied for evidence of systemic toxicity. The polymer implants exhibited a mild tissue reaction with the same characteristics as the control. Local tumor formation, bone osteolysis, and surrounding muscle necrosis were not seen. No pathological changes compatible with systemic toxicity by Delrin 150 were observed in the study of the organs. Delrin 150 in solid form did not exhibit local or systemic toxicity and is therefore biocompatible by this study. Powder implantation studies should be performed to simulate tissue response to wear particles.

## INTRODUCTION

One of the more dramatic advances in orthopedic surgery has been the development of total joint prostheses for replacing arthritic and traumatized joints. The prostheses most frequently used today consist of polished metal alloy components articulating with ultrahigh-molecular-weight polyethylene (UHMWPE) components. The increasing demand placed on prostheses to last for longer periods of time has prompted the search for improved materials.

Delrin acetal resins are thermoplastic polymers manufactured by the polymerization of formaldehyde. Their physical attributes include high mechanical strength, creep resistance, good fatigue endurance, and ease of fabrication.[1] Delrin 150, which is a high-viscosity form for extrusion processing, has been proposed as a possible alternative to UHMWPE for use in prosthetic joints.[2,3] In Europe, Delrin has been in use for components of a total hip prosthesis, known as the Christiansen prosthesis.[4] Nevertheless, variable wear rates have been reported for Delrin in laboratory tests[5–11]

Journal of Biomedical Materials Research, Vol. 19, 519–533 (1985)

CCC 0021-9304/85/050519-15$04.00

and recently, excessive wear *in vivo* of some Delrin components has been reported.[12,13]

Polymers exhibit the characteristic of creep or deformation under constant load. Indentation creep must be limited for total joint prostheses in order not to restrain the lateral motion and rotation built into the design of the articulating surfaces.[14] Creep deformation restricting motion has been implicated as a cause of tibial component loosening in total knee replacement. Cold flow (creep) has been observed in loosened UHMWPE tibial components removed from patients.[15,16] *In vitro* studies have shown the creep of Delrin to be substantially less than UHMWPE.[6,17] Also, damage to UHMWPE components, caused by the ingress of bone cement particles, has been widely reported for knee prostheses. Since Delrin has a much higher hardness,[2] the damage due to abrasion should be less severe.

In spite of extensive clinical use in Europe, only preliminary work on Delrin biocompatibility has been reported in the literature. The manufacturer of the material conducted 3-month feeding tests in rats at a dietary level of 25% Delrin resin. These tests resulted in no significant differences between control and test rats in body weight, food consumption, food efficiency, or clinical history. Hematological and pathological studies disclosed no changes attributable to the feeding of Delrin.[1] Human skin patch tests and extensive use of Delrin in contact with skin indicate that Delrin is not a dermatitis hazard.[1]

The purpose of this study was to evaluate the biocompatibility of Delrin 150 by implantation of solid samples into both muscle and bone of several animal species.

## MATERIALS AND METHODS

### Materials

Delrin 150 was obtained from Howmedica International, Inc. of Limerick, Ireland. The 316 LC stainless steel used as a control was supplied by Joslyn Stainless Steel, Fort Wayne, IN. The composition of the 316 LC stainless steel was: carbon 0.01%, manganese 1.83%, phosphorus 0.03%, silicon 0.66%, sulphur 0.02%, chromium 17.21%, nickel 12.64%, molybdenum 2.28%, copper 0.25%, cobalt 0.14%, and iron 64.93%.

### Preparation of implants

Solid cylindrical implants of three sizes were machined from supplied rod samples of Delrin 150 and 316 LC stainless steel. The dimensions of the implants were: 2 mm in diameter × 6 mm in length from rabbit bone and rat muscle; 4 mm in diameter × 12 mm in length for rabbit muscle and dog bone; and 6 mm in diameter × 12 mm in length for dog muscle.

The surfaces of the 316 LC stainless steel controls were passivated and finished according to ASTM Standard F86 for metallic surgical implants.[18]

The surface of the Delrin implants was a 16 micro-inch RMS machine finish. The test (Delrin 150) implants were washed in a mild detergent, rinsed in distilled water, and sterilized by autoclaving at 120°C. The implants were packaged and handled similar to materials for human implantation.

## Animal hosts and sites

Male rats, strain Fischer-344, adult male New Zealand albino rabbits (5 lb.) and adult male mongrel dogs (50 lb.) were used in this experiment. Twenty-two rats, twenty rabbits, and seven dogs were used as hosts for intramuscular implants, while seventeen rabbits and eight dogs were used as hosts for bone implants. Each rat received one control implant and one test implant; each rabbit received two test implants and one control implant in the paravertebral muscle on each side of the spine or in each femur; and each dog received four test implants and two control implants on each side of the spine or in each femur.

A total of 158 test samples (Delrin 150) and 90 control samples (316 LC stainless steel) were implanted in animal paravertebral muscles. A total of 132 test samples (Delrin 150) and 66 control samples (316 LC stainless steel) were implanted in animal femora. The schedule for the sacrifice of animals was based on ASTM F469[19] and is given in Table I.

## Implantation procedure

After satisfactory anesthesia was obtained, standard aseptic surgical technique was employed, and care was taken to avoid damage to or contamination of the surface of the implants. Soft tissue samples were implanted longitudinally in the paravertebral muscle on each side of the spine through posterior paramedial incisions. The fascia was closed with absorbable suture to prevent implant dislodgement. The femora was approached through a lateral incision. After periosteal elevation, small holes were drilled in the lateral cortex just large enough to accept the respective cylindrical implants

**TABLE I**
**Sacrifice Schedule of Delrin 150 Biocompatibility Study**

| | Muscle | | | Bone | |
|---|---|---|---|---|---|
| Sacrifice periods, (Weeks after insertion of implants) | Rat | Rabbit | Dog | Rabbit | Dog |
| 2 | 4 | 2 | | 2 | |
| 3 | 4 | 3 | | 2 | |
| 8 | 4 | 2 | | 3 | |
| 12 | 4 | 3 | | 3 | |
| 26 | 6 | 3 | 2 | 3 | 2 |
| 52 | | 3 | 2 | 2 | 2 |
| 104 | | 4 | 3 | 2 | 4 |

which were then introduced into the bone. The surgical wounds were closed in a routine manner with absorbable suture.

### Postoperative care

The rabbits and dogs received one dose of intramuscular penicillinase-resistant penicillin immediately postoperative. The animals were fed a standard diet and water ad libitum. Activity was unrestricted. The animals were observed during the period of assay for any abnormal findings. Animals that died during the period prior to the expected date of sacrifice were autopsied to determine the cause of death. The animal was then replaced if the cause of death was unrelated to the test material.

### Tissue preparation

The animals were sacrificed at the intervals listed in Table I. At sacrifice the tissue surrounding the implant was examined for any gross abnormalities in color or consistency. The soft tissue implants were retrieved "en bloc" with an intact envelope of surrounding tissue and were fixed in 10% buffered formalin for a minimum of 48 h. The implants were extracted taking care to preserve the fibrous tissue lining. Subsequently the tissue samples were dehydrated and embedded in paraffin, then sectioned in 5-$\mu$m-thick slices with a Jung microtome. The plane of the sectioning was perpendicular to the longitudinal axis of the cylindrical implant. At least two slides were prepared from each tissue block of test and control implants, and stained with hematoxylin and eosin.

Contact radiographs were taken of the femora containing implants in order to localize the implants and also to study the bone surrounding the implants for radiologic signs of osteolysis or bone proliferation. The bones were sectioned into blocks containing one implant each, fixed in buffered formalin for 48 h, and decalcified in Scientific Products Decalcifying Solution. The implants were then carefully removed from the decalcified blocks which were subsequently sectioned and processed for light microscopy, as described above.

The liver, spleen, lung, pancreas, and kidneys of all the animals were grossly examined and then fixed in buffered formalin. These organs were then sectioned and representative samples from each animal were processed for histologic study. All sections were stained with hematoxylin and eosin and examined by light microscopy and polarized light microscopy.

### Histology and scoring system

The tissue around the implant was examined by routine light microscopy. A histological scoring system modified from that used by Autian[20] was used, and 16 histologic criteria were assessed for each implantation site section.

The histologic criteria were: (1) fibrous membrane thickness, (2) membrane cellularity, (3) inflammation, (4) polymorphonuclear cells, (5) giant cells, (6) macrophages, (7) lymphocytes, (8) plasma cells, (9) foreign debris, (10) surrounding tissue necrosis, (11) surrounding tissue fibrosis, (12) surrounding fat infiltration, (13) tumor formation, (14) blood vessels, (15) osteolysis, and (16) reactive bone formation. For every slide each histological parameter was graded from 0 to +3 except the membrane thickness which was measured in microns. The description of each grade was: 0 = item not present; +1 = item occasionally present; +2 = item present to moderate degree; +3 = item present to marked degree.

The thickness of the fibrous membrane encapsulating the implant was measured with a Zeiss measuring eyepiece at four different points and the average value calculated and recorded. When measuring the membrane thickness, care was taken to avoid the areas in contact with the ends of the implant where increased thickness and cellularity occur. The paired *t*-test was used to statistically compare the thickness of the fibrous membrane between test and control implants. The quantity and quality of cells within the membrane and in the surrounding tissue was studied. The different cell types were identified by their characteristic morphology. Each slide was carefully examined for tumors and foreign bodies. Bone implant sites were also inspected for signs of osteolysis near the implant or reactive bone formation near the implant.

The organs (liver, spleen, lung, pancreas, and kidneys) were examined by light microscopy. Pathological findings in the organs were compared with the expected spontaneous incidence of similar pathology in laboratory animals of each respective species.

## RESULTS

The macroscopic and histologic study of the organs revealed no evidence of systemic toxicity attributable to the Delrin 150 implants. A lung mass was discovered in a dog 26 weeks after muscle implantation. Histopathology revealed this to be a papillary adenocarcinoma of the lung. A cystic bile duct carcinoma occurred in the liver of a rabbit 104 weeks after bone implantation. Fifty percent of the rats had mild perivascular infiltration of mononuclear cells in their lungs; however, no foreign material could be identified, even after inspection of sections by polarized light microscopy. Three rabbits died within 3 days of surgery, but autopsies revealed death to be due to postoperative cardiovascular complications and not due to the test implant.

Roentgenographic study of the bone implant sites did not show any osteolysis, but mild bone proliferation was noted around the surface of the cylindrical implants and where the implants protruded from the cortex. Radiologically, test and control samples elicited the same response in bone.

Macroscopic evaluation of soft tissue and bone at the implant sites showed minimal reaction, with no change in color or consistency and no sign of muscle necrosis.

**TABLE II**
**Histology of Local Tissue Response for Muscle Implantation**

| Animal | Sacrifice period (weeks) | Average membrane thickness(μm) | | Membrane cellularity | | Inflammation (excluding macrophages) | | Macrophages | |
|---|---|---|---|---|---|---|---|---|---|
| | | T | C | T | C | T | C | T | C |
| Rat | 2 | 25 | 17 | +3 | +3 | +2 | +2 | +1 | +1 |
| | 3 | 8 | 17 | +2 | +2 | +1 | +1 | +1 | 0 |
| | 8 | 29 | 26 | +1 | +1 | 0 | 0 | +1 | +1 |
| | 12 | 28 | 32 | +1 | +1 | 0 | 0 | 0 | 0 |
| | 26 | 33 | 21 | +2 | +1 | 0 | 0 | +1 | 0 |
| Rabbit | 2 | 44 | 52 | +2 | +2 | +1 | +1 | +2 | +2 |
| | 3 | 85 | 81 | +2 | +2 | +1 | +1 | +1 | +1 |
| | 8 | 26 | 67 | +1 | +2 | 0 | 0 | 0 | 0 |
| | 12 | 32 | 29 | +1 | +1 | 0 | 0 | 0 | 0 |
| | 26 | 42 | 42 | +1 | +1 | 0 | 0 | 0 | 0 |
| | 52 | 30 | 26 | +1 | +1 | 0 | 0 | +1 | 0 |
| | 104 | 36 | 25 | +1 | +1 | 0 | 0 | +1 | 0 |
| Dog | 26 | 39 | 38 | +1 | +1 | 0 | 0 | +1 | +1 |
| | 52 | 36 | 44 | +1 | +1 | 0 | 0 | +1 | +1 |
| | 104 | 30 | 37 | +1 | +1 | 0 | 0 | +1 | 0 |

*Note.* T = test implant (Delrin 150); C = control implant (316 LC stainless steel).

Table II summarizes pertinent histological criteria and grades each criteria for the test and control samples implanted into muscle. The "inflammation" category in Table II excludes "macrophages" in order to isolate and highlight the macrophage reaction. This semiquantitative evaluation data reported in Table II represents an average of the values assigned to either all test or all control implants in all those animals sacrificed at the designated time period.

Twelve of the sixteen histologic criteria assessed are not included in Table II to simplify the table, but are summarized in the text. Polymorphonuclear cells, giant cells, lymphocytes, plasma cells, and increased vascularity were seen only in the 2- and three-week sacrifice periods when inflammation due to surgical trauma was still present. The test and control implants were identical with regard to these inflammatory cells. Inflammation resolved before the 8-week sacrifice period.

Tumor formation at the implant site, necrosis of muscle surrounding the implant, and osteolysis were not seen on any of the tissue slides. Small birefringent particles were seen concentrated in and around the fibrous membrane only for rats with 6-month muscle implants. These particles are not significant since they were seen for both test and control implants, and may represent digested suture material. Inflammation was not associated with the particles.

Fibrous tissue infiltrated the muscle surrounding the implants for the short-term rabbits and dogs. In long-term rabbits and dogs, adipose tissue replaced fibrous tissue which had been present in the short-term animals. These changes in the surrounding tissue were identical for test and control implants. Reactive new bone formation occurred at all bone implants, even

for the 2-week sacrifice periods, and was independent of the type of material implanted.

## Rat muscle

Histologic examination of the rat muscle samples showed hypercellularity of the lining membrane during the first few weeks, with moderate inflammation. Giant cells, lymphocytes, eosinophils and macrophages were present focally in small numbers during this period. The membrane thickness was quite variable for both Delrin and control implants, ranging from 8 to 33 $\mu$m, and the membrane was consistently thicker where the surface of the implant was near the surgical incision. There were no significant differences in these parameters between control and test implants for the first few months.

The membrane cellularity was markedly diminished and consisted of occasional mature fibrocytes by the third month. Vascularity had uniformly decreased, and inflammation had completely resolved. At 6 months a difference was noted in the fibrous membrane between test and control samples. Whereas the fibrous membrane around control implants was uniform with low cellularity, the membrane around test implants had a layer of fibroblastic and macrophage-appearing cells immediately adjacent to the implant site with mature fibrous tissue located peripherally (Fig. 1). The average membrane thickness was 12 $\mu$m greater for the test implants at six months com-

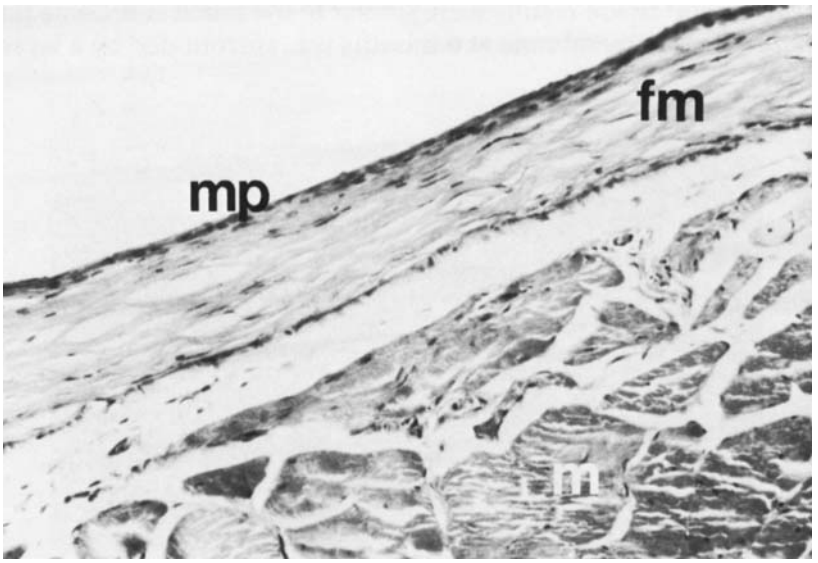


**Figure 1.** Photomicrograph of the fibrous membrane (fm) around a Delrin 150 test implant in rat muscle (m), 6 months after implantation. A single, incomplete layer of fibroblastic and macrophage-appearing cells (mp) is adjacent to the implant site, with hypocellular fibrous membrane peripheral to the macrophage layer. (Original magnification 125×.)

pared to the controls, although this difference was not statistically significant ($p = 0.32$). No muscle necrosis was seen.

### Rabbit muscle

The rabbit muscle implants showed membranes with moderate cellularity and mild inflammation during the first few weeks. Moderate vascularity was present for both implants, with macrophages, giant cells and eosinophils in surrounding tissue. Fibrous tissue infiltrated into surrounding muscle for both implants, making the fibrous membrane–muscle interface indistinct (Fig. 2). The cellularity of the membrane decreased with time, and the inflammatory response became minimal. The surrounding muscle which had been infiltrated by fibrous tissue was gradually replaced by fat, so that by 6 months there existed a distinct thin hypocellular fibrous membrane surrounded by a layer of fat which then was surrounded by normal muscle (Fig. 3). The membranes were partially torn on implant extraction by cleavage through the fat layer. The average membrane thickness varied from 25 to 85 $\mu$m. The average membrane thickness of long-term test implants was slightly greater than that observed for controls, but still minimal. In addition rare foci of macrophages were seen along the membrane periphery for some of the test implants for 2 years.

### Dog muscle

The canine soft tissue results were similar to the rabbit soft tissue findings. The mature fibrous membrane at 6 months was surrounded by a layer of fat,

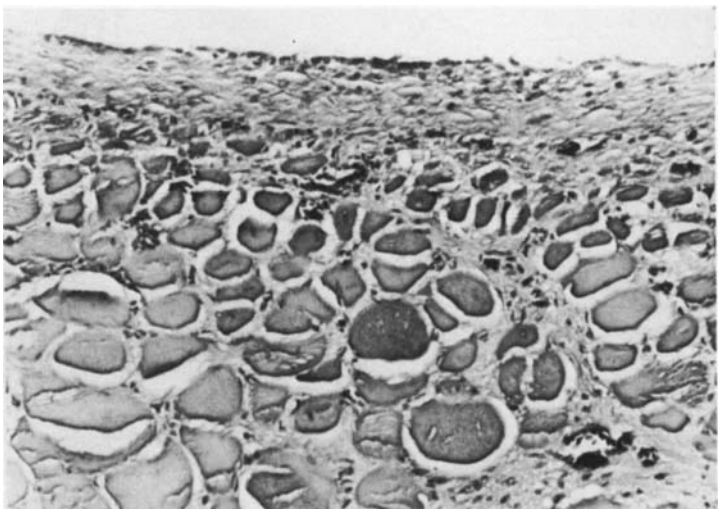

**Figure 2.** Microphotograph of the tissues in contact with a 316 LC stainless-steel implant, in rabbit muscle 2 weeks after implantation. Fibrous tissue infiltrates into surrounding muscle, making the membrane–muscle interface indistinct. The fibrous tissue has moderate cellularity and mild surrounding inflammation. (Original magnification 80×.)

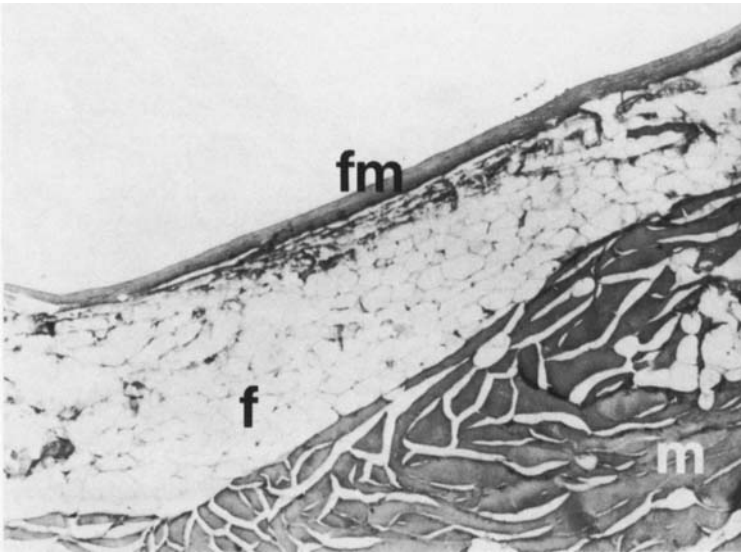


**Figure 3.** Microphotograph of the fibrous membrane (fm) and adjacent tissues in contact with a Delrin 150 implant, in rabbit muscle 2 years after implantation. The fibrous membrane is hypocellular and is separated from normal muscle (m) by a layer of fat cells (f). (Original magnification 35×.)

but again there was no necrosis of muscle tissue. This adipose layer increased only slightly between 6 months and 2 years. Occasional focal areas of macrophages were seen at the periphery of the membrane for both test and control implants. The average membrane thickness for the control implants (37–44 $\mu$m) was slightly increased over the test implants (30–39 $\mu$m), but this was not statistically significant.

### Rabbit and dog bone

In the rabbit and canine femora sections, the tissue response to the implant varied according to its location in bone; whether the section being examined was cut through cortical bone or near the medullary cavity. When the level of section was through cortical bone, the newly remodeled cortical bone had a circumferential pattern directly adjacent to the surface of the implant. Little if any fibrous membrane was seen between the new bone and the implant site. Bone marrow elements were occasionally found within the lumen or along the bone–implant site interface. Clefts containing marrow elements, vessels, red blood cells, and fibrous strands were intermittently seen along the edge of the new bone. These clefts did not contain osteoclasts, and no sign of osteolysis was present. Occasional osteoid seams were seen directly adjacent to the implant site. The reactive bone progressed from woven to lamellar bone as remodelling occurred.

When the level of section was from medullary bone, a fibrous membrane formed around the implant and reactive bone trabeculae surrounded this membrane (Fig. 4). Bone marrow elements and adipose tissue were in-

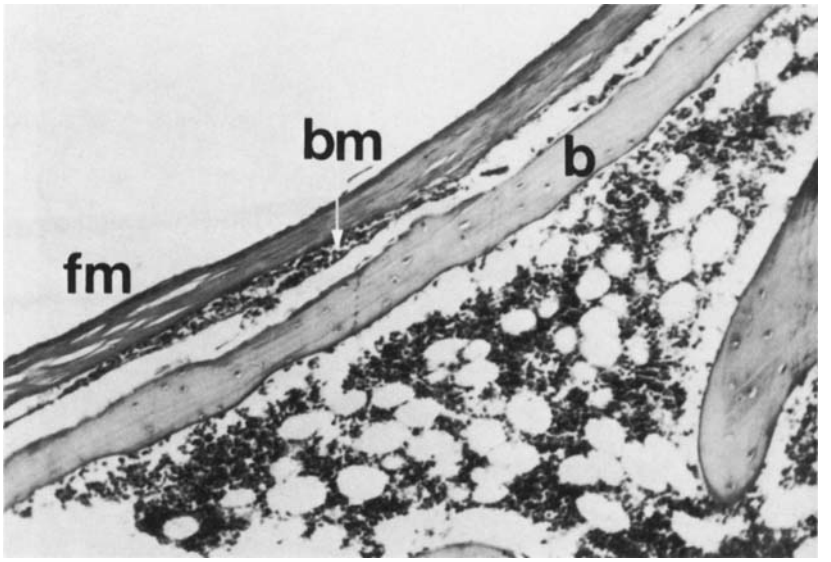


**Figure 4.** Photomicrograph of a Delrin implant site in canine bone, one year after implantation. The level of section is in the medullary canal. A fibrous membrane (fm) is adjacent to the implant, and a trabecula of new bone (b) surrounds the membrane. Bone marrow elements (bm) are interspersed between the membrane and bone. (Original magnification 50×.)

terspersed between the membrane and the trabeculae of new bone. This fibrous membrane evolved from a membrane with moderate cellularity and occasional inflammatory cells to a mature hypocellular membrane without inflammation. The average membrane thickness for sections near the medullary canal was 20 $\mu$m for rabbit bone implants at all sacrifice periods and 30 $\mu$m for dog bone implants at all sacrifice periods. Test and control samples elicited the same response in bone for each animal species.

## DISCUSSION

This 2-year implantation study of Delrin 150 solid cylinders in animal muscle and bone indicates that this material is well tolerated by the criteria examined and exhibited tissue responses very similar to those of a 316 LC stainless-steel control.

In addition to local physical effects, tissue response to a biomaterial can be related to the release of a substance from the material to the adjacent environment. For polymer implants the offending agent may be: (1) Low-molecular-weight polymers, (2) one of the additives intentionally added to the polymer, (3) contaminants becoming part of the product during manufacturing, (4) impurities in the additives, or (5) degradation products of the polymer or additives.[31] Delrin has been shown to leach formaldehyde in minute quantities under certain conditions,[2] and for that reason questions have been raised about its biocompatibility. Neither local or systemic toxicity could be demonstrated in this study.

In evaluating local tissue response to implantation of solids, many investigations[22–27] have successfully used measurement of the thickness of the fibrous tissue membrane. Because of the many physical variables influencing membrane thickness, Kupp[28] has stated that differences in membrane thickness should exceed 25 $\mu$m to be significant. Laing et al. have observed that the thickness of the fibrous membrane around solid metallic implants was proportional to the degree of metallic dissolution.[26] Solid polymer implants with leachable toxic constituents would be expected to likewise produce a thicker fibrous membrane. In this study the fibrous membrane thickness around the solid implants was variable, being quite dependent on the degree of surgical trauma. However, there were no significant differences in membrane thickness between control and test implants. Some of the variability in membrane thickness might have been eliminated by percutaneous muscle implantation of the cylindrical samples through large bore needles, thus minimizing surgical trauma.[20]

A technical problem encountered with the muscle implants was the tendency to disrupt the fibrous membrane during implant extraction. Marion[29] has reported this same problem. Ideally, histologic processing should be performed without implant removal, but for implants that cannot be cut with conventional microtomy, new cutting or implant extraction techniques need to be developed.

Inflammation is a nonspecific physiologic response to tissue damage such as trauma, infection, foreign materials, local cell death or as part of an immune response. Some studies[14,26] have indicated a correlation between surrounding tissue inflammation and fibrous membrane thickness. This study does not reveal the same correlation, but surrounding tissue inflammation was inversely proportional to length of implantation time as would be expected in normal wound healing. The only significant difference in cellular response between test and control implants was the presence of macrophages in long-term Delrin 150 rat muscle implantation. A focal and sparse layer of macrophages and fibroblasts was observed immediately adjacent to the implant site for rats sacrificed at 6 months. The significance of these macrophages in assessing the biocompatibility of Delrin 150 is not known. Since this period represented the longest implantation time in rats, the possibility of extremely long-term toxicity cannot be excluded with certainty in these animals. However, this reaction was conspicuously absent in dogs and rabbits studied for longer time periods.

Local tumor induction has been shown to be a toxic effect of some biomaterials in animals.[30–36] Local tumor formation was not seen at any of the implant sites in this study.

Severe toxic effects of moieties released by the implant may lead to tissue necrosis for muscle implants and osteolysis for bone implants, but neither reaction was seen in this study. The adipose tissue that formed between the membrane and muscle in long-term implants appears to be related to the local mechanical effect of the implant. There was no coinciding cellular reaction nor change in the surrounding muscle shell. This phenomenon has been reported in other implantation studies.[37]

For the bone implants, little if any fibrous tissue was interposed between the implant and the reactive bone when the level of section was through cortical bone. This observation has been documented elsewhere,[23,27] and Harms[37] found that for implanted ceramic solids there was a thin histiocytic layer between bone and implant which was often interrupted in tissue preparation. The medullary aspect of the implant was encapsulated by a thin fibrous membrane which was surrounded by trabeculae of new bone. These observations were seen for both test and control implants.

The pathology detected in the study of the organs corresponds to the findings expected to occur spontaneously in laboratory animals.[38] The two cases of carcinoma reflect the spontaneous occurence of these tumors in rabbits and dogs[39] and birefringent polyacetal particles were not present at the tumor sites. Antigenic reaction was not exhibited on any of the histology reviewed, but the propensity of monomers, curing agents, etc., to incite allergic responses in chemical industry workers should alert us to the possibility of allergic reactions when similar compounds are used in polymer implants.[21]

Total joint prostheses have wearing surfaces that produce particles. As a result of particle formation, the surface area of the biomaterial in contact with tissues is considerably increased. It has been postulated that the granulation tissue formed in response to the particulate material can invade the bone–cement interface and lead to prosthetic loosening.[40,41] In addition, the particulate polymer is phagocytized and transported to lymph nodes and remote reticuloendothelial system sites, where its long term effects are not known at this time.

Conflicting data for the wear rate of Delrin, especially as compared to UHMWPE, appears in laboratory studies.[5–11] Part of the discrepancy may be due to different testing conditions and methods of measurement. For example, an early study which reported a lower wear rate for Delrin compared to UHMWPE, actually measured deformation of the UHMWPE.[5] In another study, using blood plasma as a lubricant, it was reported that the wear rate of Delrin was lower than that of UHMWPE, but the opposite was true when water was used as the lubricant.[8] In general, other laboratory studies have reported higher wear rates for Delrin compared to UHMWPE.[6,7,9–11]

*In vivo,* in a study of five Christiansen protheses removed for loosening or infection, one showed signs of wear.[2] More recently, excessive wear of some Delrin components removed from failed arthroplasties has been reported.[12,13] Wear of the Delrin acetabular cup has been attributed in one study to irregularities on the head of the femoral component.[42] In addition, a high incidence of failure of the Christiansen prosthesis compared to the Charnely prosthesis, due to aseptic loosening, has been present in different studies.[13,43] Whether aseptic loosening in these cases is due to wear particles,[44] or other factors,[13,42] is unclear. A report of histological studies of tissue surrounding Christiansen prostheses removed for cause, such as cup loosening, found only minimal to moderate inflammatory response with mononuclear cells and rare giant cells.[2]

In conclusion, Delrin 150 in solid form appears to be biocompatible, but other considerations may determine its use in prosthetic components, and powder implantation studies could serve to evaluate the biologic response to wear particles.

This investigation was supported by Research Grant No. AM16485 from the Department of Health and Human Services, Public Health Service, The National Institute of Health. The authors thank Ms. Rene Balos, Ms. Eva Dunneisen, and Ms. Elizabeth Gaughan for their excellent technical assistance.

## References

1. *Delrin-Design Handbook,* E. I. DuPont de Nemours and Co., Wilmington, DE, 1967.
2. J. H. Dumbleton, "Delrin as a material for joint prostheses of a review," in *Corrosion and Degradation of Implant Materials,* B. C. Syrett and A. Acharya, Eds., Am. Soc. Test. Mater., Philadelphia, 1979, p. 41–60.
3. A. Poli, "A laboratory experiment on total hip prostheses," *Proc. Inst. Mech. Engrs.,* **181,** 140–141 (1966).
4. T. Christiansen, "A combined endo- and total hip prosthesis with trunnion bearing," *Acta. Chir. Scand.,* **140,** 185–188 (1974).
5. I. Duff-Barclay and D. T. Spillman, "Total human joint prosthesis — A laboratory study of friction and wear," *Proc. Inst. Mech. Engrs.,* **181**(3J), 90–103 (1966–67).
6. H. Amstutz, "Polymers as bearing materials for total hip replacement: A friction and wear analysis," *J. Biomed. Mater. Res.,* **3,** 547–568 (1968).
7. J. O. Galante and W. Rostoker, "Wear in total hip prostheses; an experimental evaluation of candidate materials," *Acta Orthop. Scand. (supp.),* **145,** 6–46 (1973).
8. C. Shen and J. Dumbleton, "The friction and wear behavior of polyoxmethylene in connection with joint replacement," *Wear,* **38,** 291–303 (1976).
9. H. McKellop, I. Clarke, K. Markolf, and H. Amstutz, "High wear rates with Delrin 150 — A clinical hazard?" *Transactions of the Annual Meeting of Orthopedic Research Society,* **3,** 7 (1978).
10. I. C. Clarke and H. McKellop, "The wear of Delrin 150 compared with polyethylene, polyester and PTPE," *Mechanical Properties of Biomaterials,* G. W. Hastings and D. F. Williams, Eds., Wiley, New York, 1980, pp. 27–37.
11. H. McKellop, I. Clarke, K. Markolf, and H. Amstutz, "Friction and wear properties of polymer, metal and ceramic prosthetic joint materials evaluated on a multichannel screening device," *J. Biomed. Mater. Res.,* **15**(5), 619–653 (1981).
12. L. I. Havelin, N. R. Gjerdet, O. D. Lunde, M. Rait, and E. Sudmann, "Measurement of wear in Christiansen acetabular cups (abstract)," *Acta Orthop. Scand.,* **54,** 949 (1983).
13. E. Sudmann, L. I. Havelin, O. D. Lunde, and M. Rait, "The Charnley versus the Christiansen total hip arthroplasty. A comparative clinical study," *Acta Orthop. Scand.,* **54,** 545–552 (1983).
14. W. Rostoker and J. O. Galante, "Materials for human implantation," *J. Biomech. Eng.,* **101,** 2–14 (1979).
15. H. Shoji, R. D'Ambrosea, and P. Lipscomb, "Failed polycentric total knee prostheses," *J. Bone Joint Surg.,* **58A,** 773–777 (1976).
16. H. A. Lacey, "A statistical review of 100 consecutive U.C.I. low friction knee arthroplasties with analysis of results," *Clin. Orthop. Rel. Res.,* **132,** 163–166 (1978).

17. W. Rostoker and J. O. Galante, "Identation creep of polymers for human joint applications," *J. Biomed. Mater. Res.*, **13,** 825–828 (1979).
18. "Recommended Practice for Surface Preparation and Marking of Metallic Surgical Implants," ASTM F86, 1978.
19. "Standard Practice for assessment of Compatibility of Nonporous Polymeric Materials for Surgical Implants with Regard to Effect of Materials on Tissue," ASTM F469, 1978.
20. J. Autian, "The new field of plastics toxicology — methods and results," *CRC Crit. Rev. Toxicol.*, **2**(1), 1–40 (1973).
21. J. Autian, "Toxicologic aspects of implants," *J. Biomed. Mater. Res.*, **1,** 433–449 (1967).
22. F. Escalas, J. O. Galante, W. Rostoker, and P. Coogan, "Biocompatibility of materials for total joint replacement," *J. Biomed. Mater. Res.*, **10,** 175–195 (1976).
23. R. Tetik, J. O. Galante, and W. Rostoker, "A wear resistant material for total joint replacement — Tissue biocompatibility of an ultrahigh molecular weight polyethylene–graphite composite," *J. Biomed. Mater. Res.*, **8,** 231–250 (1974).
24. S. Gourlay, R. Rice, A. Hegyeli, C. Wade, J. Dillon, H. Jaffe, and R. Kulkarni, "Biocompatibility testing of polymers: *In vivo* implantation studies," *J. Biomed. Mater. Res.*, **12,** 219–232 (1978).
25. F. Escalas, J. O. Galante, W. Rostoker, and P. Coogan, "$MP_{35}N$: A corrosion resistant, high strength alloy for orthopedic surgical implants: Bio-assay results," *J. Biomed. Mater. Res.*, **9,** 303–313 (1975).
26. P. G. Laing, A. Ferguson, and E. Hodge, "Tissue Reaction in rabbit muscle exposed to metallic implants, " *J. Biomed. Mater. Res.*, **1,** 135–149 (1967).
27. H. Denissen, K. DeGrott, P. Makkes, A. van den Hooff, and P. Klopper, "Tissue response to dense apatite implants in rats," *J. Biomed. Mater. Res.*, **14,** 713–721 (1980).
28. T. Kupp, P. Hochman, J. Hale, and J. Black, *Advances in Biomaterials,* Wiley, London, 1980.
29. J. Marion, E. Haugen, and I. Mjor, "Methodological assessments of subcutaneous implantation techniques," *J. Biomed. Mater. Res.*, **14,** 343–357 (1980).
30. F. C. Turner, "Sarcomas at sites of subcutaneously implanted bakelite discs in rats," *J. Nat. Cancer Inst.*, **2,** 81–83 (1941).
31. B. S. Oppenheimer, E. T. Oppenheimer, I. Danishefsky, A. Stout, and F, Eirich, "Further studies of polymers as carcinogenic agents in animals," *Cancer Res.*, **15,** 333–340 (1955).
32. E. T. Oppenheimer, M. Willhite, I. Danishefsky, and A. Stout, "Observations on the effects of powdered polymer in the carcinogenic process," *Cancer Res.*, **21,** 132–134 (1961).
33. J. Lavorgna, J. Burstein, A. Schiller, and W. Harris, "The carcinogenesis of plastics used in orthopedic surgery," *Clin. Orthop.*, **88,** 223–227 (1972).
34. R. Carter and F. Roe, "Induction of Sarcomas in rats by solid and fragmented polyethylene: Experimental observations and clinical implications," *Br. J. Cancer,* **23,** 401–407 (1969).
35. J. Harrison, D. McLain, R. Hohm, G. Wilson, J. Chalman, and K. MacGowan, "Osteosarcoma associated with metallic implants," *Clin. Orthop. Relat. Res.*, **116,** 253–257 (1976).
36. K. Sinibaldi, H. Rosen, L. Si-Kwang, and M. DeAngelis, "Tumors associated with metallic implants in animals," *Clin. Orthop. Relat. Res.*, **118,** 257–266 (1976).
37. J. Harms and E. Mausle, "Tissue reaction to ceramic implant material," *J. Biomed. Mater. Res.*, **13,** 67–87 (1979).
38. W. Ribelin and T. McCoy, *Pathology of Laboratory Animals,* Charles C. Thomas, Springfield, IL, 1965.

39. J. Moulton, *Tumors in Domestic Animals,* University of California Press, Los Angeles, 1978.
40. H. Willert and M. Semlitsch, "Tissue reactions to plastic and metallic wear products of joint endoprostheses," *Total Hip Prosthesis,* Williams and Wilkins Co., Baltimore, 1976.
41. J. Mirra, H. Amstutz, M. Matos, and R. Gold, "The pathology of the joint tissues and its clinical relevance in prosthesis failure," *Clin. Orthop. Relat. Res.*, **117,** 221–240 (1976).
42. A. Ohlin and T. Erikson, "A rough surface as a cause of wear of the cup in the Christiansen total hip (abstract)," *Acta Orthop. Scand.*, **55,** 101, 1984.
43. G. Josefsson, B. Eriksson, M. Fagerlund, G. Gudmundersson, H. Helgasson, O. Karlsson, B. Nordstrom, B. O. Olnas, and S. Wijkstrom, "Non-septic loosening in a multi-center study of 1686 THR (abstract)," *Acta Orthop. Scand.*, **52,** 696 (1981).
44. E. B. Mathiesen, E. Sudmann, U. Lindgrun, and F. Reinholt, "Tissue reaction to wear products from Delrin and UHMW-polyethylene (abstract)." *Acta Orthop. Scand.*, **54,** 485 (1983).


Received August, 1984
Accepted December, 1984