Material properties of absorbable self-reinforced fibrillated poly-96L/4 D-lactide (SR-PLA96) rods; a study *in vitro* and *in vivo*

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A study was carried out to investigate changes in mechanical properties and degradation of self-reinforced fibrillated poly-96*L*/4*D*-lactide (SR-PLA96) rods *in vitro* and *in vivo*. The viscosity-average molecular weight, M_v, of the intact sterile (gamma irradiated) rods was around 50 000 g mol^{-1}. The SR-PLA96 rods of diameter 1.1 mm by 30 mm and diameter 4.5 mm by 50 mm were immersed in phosphate-buffered saline or implanted in the dorsal subcutis of rabbits. Bending, shear and torsion strength and bending modulus, together with the changes of viscosity and crystallinity, were measured up to 24 wk. The strength values showed only a slight decrease during the follow-up period with the exception of torsion strength, that decreased to 52% of the initial value during 24 wk. There were no statistically significant differences in the strength retention between *in vitro* and *in vivo* groups. Crystallinity increased over time, being 46–49% at 24 wk. The M_v of the rods decreased over 50% by 24 wk. These promising results motivated us to continue the studies with the fixation of experimental cortical bone osteotomies with SR-PLA96 intramedullary rods.

1. Introduction

Poly-*L*-lactide (P*L*LA) has been successfully used in a number of surgical implications as a biodegradable osteosynthesis material both in humans and animals [1–7]. Improvement in manufacturing techniques of self-reinforced poly-*L*-lactide (SR-P*L*LA) devices [8, 9] allowed these biodegradable implants to have initial strength and strength retention probably sufficient even for fixation of cortical bone osteotomies. Both sintered [10] and fibrillated [11] SR-P*L*LA rods have been successfully used for intramedullary fixation of weight-bearing cortical bone osteotomies in rabbits.

However, the complete degradation and resorption of P*L*LA material *in vivo* seems to proceed quite slowly. Several investigators [12–15] have found P*L*LA material present or partly degraded in the operated area up to 5 y or even longer postsurgically. However, Matsusue *et al.* [16] observed total absorption of high molecular weight oriented P*L*LA rods in subcutaneous tissue at 69 mon.

It is recognized that α-hydroxypolyester implants, manufactured from the same polymer batch and especially implants manufactured from polymers obtained from different sources, may have significant differences in their degradation due to differences, for example, in their processing method, sterilizing method, thermal history, crystallinity, purity (especially monomer content) and molecular weight distribution [17–20]. These properties influence the degradation behavior and, consequently, the biological reaction of the host tissues to the implanted polymer.

A late tissue response manifested by a local painless subcutaneous swelling 3 y postoperatively has been reported with large as-polymerized P*L*LA plates and screws used for zygomatic bone fixation in humans [13]. Phagocytosis was triggered by the slowly degrading crystalline P*L*LA polymer debris still present in the subcutaneous tissue. However, the implantation site seemed to be an important factor determing the type and intensity of the inflammatory response against P*L*LA residue. Swelling was not observed in

the intraosseously implanted thread portion of the P*L*LA screws and the internalization of P*L*LA particles by phagocytic cells was very limited. A low incidence of reactions has also been observed in clinical long-term follow-up studies with P*L*LA screw fixation of displaced ankle fractures [7, 14]. Furthermore, only a mild inflammatory response was seen in an experimental study where the late-phase degradation of a large P*L*LA intramedullary nail was studied in a porcine model [21]. These results indicate that there may be a variation in the degradation mechanism between subcutaneous and intraosseous P*L*LA implants and the histological reaction induced by the implant. Anyhow, the resorption time of highly crystalline P*L*LA is excessive in relation to fracture healing time. Therefore, absorbable polymers with a low to moderate degree of crystallinity should be favored for medical applications.

The fact that a racemic polymer of PLA, poly-*DL*-lactide (P*DL*LA), which is totally amorphous, degrades much faster than P*L*LA has been known for a long time [22]. Therefore, an attempt was made to develop self-reinforced P*DL*LA/P*L*LA composites by mixing melt-spun P*DL*LA fibres (40 vol%) and P*L*LA fibers (60 vol%) and sintering the fibers together in a steel mold at high pressure and at a temperature below the melting of the P*L*LA fibers [23]. This composite, although it degraded faster, was not strong enough by its mechanical properties and strength retention for the fixation of cortical weight-bearing bones.

Poly-*L*/*D*-lactide fibers manufactured from copolymers of *L*-lactide with varying amounts of *D*-lactide were shown to be suitable for fiber-reinforced implants because they possessed high initial strength, lower crystallinity, and enhanced degradation rate compared to P*L*LA [24]. Furthermore, aspolymerized poly-96*L*/4*D*-lactide (PLA96) has been studied for material properties and biocompatibility both *in vitro* and *in vivo* and also after pregradation [25–27]. It was shown to have substantially lower crystallinity and enhanced degradation rate compared to P*L*LA.

The aim of the present study was to investigate the material properties of self-reinforced fibrillated poly-96*L*/4*D*-lactide (SR-PLA96) rods during degradation *in vitro* and *in vivo*.

2. Materials and methods

2.1. Manufacturing of the implants

The biodegradable implants were made by self-reinforcing technique (SR) of poly-*L*/*D*-lactic acid stereocopolymer with *L*/*D* lactide molar ratio of 96/4. The polymer was obtained from PURAC biochem b.v., The Netherlands. Specific details of the raw polymer as given in the manufacturer's certificate of analysis were as given in Table I.

In order to prevent extensive thermal degradation during processing, water and air were removed from the polymer by vacuum drying at 100 °C and 0.05 mbar for 1 d. The polymer was then melt extruded (Axon BX-15, Sweden) into cylindrical billets

TABLE I

	Batch number DM362CE
Intrinsic viscosity (dlg^{-1}) (in chloroform at 25 °C)	8.27
Specific rotation (in chloroform at 25 °C)	− 143.6
DSC melting range (°C)	143.7–155.1
Heat of fusion (Jg^{-1})	39.2
Residual monomer content (%)	0.16

of diameters 2.7 mm and 11 mm and subsequently die-drawn in the solid state at a temperature of 125 °C to draw ratios of 5.5 to accomplish the self-reinforced fibrillated structure [28]. The sizes of the finished SR-PLA96 rods were diameter 1.1 mm by 30 mm and diameter 4.5 mm by 50 mm. The implants were sterilized by gamma irradiation at a minimum dose of 2.5 Mrad (actual dose 1.4–1.6 times the specified minimum, Kolmi-Set Ltd, Turku, Finland).

2.2. Degradation *in vitro*

The SR-PLA96 rods were immersed in (2.87 gl^{-1}) Na_2HPO_4 – (0.67 gl^{-1}) NaH_2PO_4 buffered saline (NaCl 5.9 gl^{-1}) at pH 7.4 and 0.127 M, and held at 37 °C. Samples were removed from the solution at 1, 2, 3, 4, 6, 9, 12, 15, 18 and 24 wk and changes in mechanical properties, crystallinity and M_v were determined.

2.3. Degradation *in vivo*

In total, 24 rabbits of both sexes (weighing from 3–4 kg) were used. In the first five groups (three rabbits in each group), three implants of 4.5 mm diameter and two implants of 1.1 mm diameter were placed in the dorsal subcutis of the rabbits. In the following three groups (three rabbits in each group), two implants of 4.5 mm diameter and two implants of 1.1 mm diameter were used. A total of 48 rods of 1.1 mm diameter and 63 rods of 4.5 mm diameter were implanted.

The rabbits were anesthetized with im/sc medetomidine (Domitor 1 mg ml^{-1}, Orion-Farmos, Turku, Finland) at a dose of 0.5 mg kg^{-1} and ketamine hydrochloride (Ketalar 50 mg ml^{-1}, Parke-Davis, Barcelona, Spain) at a dose of 25 mg kg^{-1}. All rabbits were given 60 000 IU kg^{-1} benzylpenicillin procaine/benzathinpenicillin (Duplocillin LA, Intervet, Holland) sc preoperatively. The dorsum of the rabbits was shaved and scrubbed with antiseptic fluid (Klorhexidos, Lääkefarmos, Turku, Finland) and the rods were inserted into the subcutis through small skin incisions.

The follow-up times were 1, 3, 6, 9, 12, 15, 18 and 24 wk. Under anesthesia the rods were removed from the rabbits and immersed in 0.9% saline and the changes in mechanical properties, crystallinity and M_v were measured within 12–24 h.

2.4. Strength measurements

The bending strength and modulus of the intact SR-PLA96 rods and after *in vitro* and *in vivo* exposure

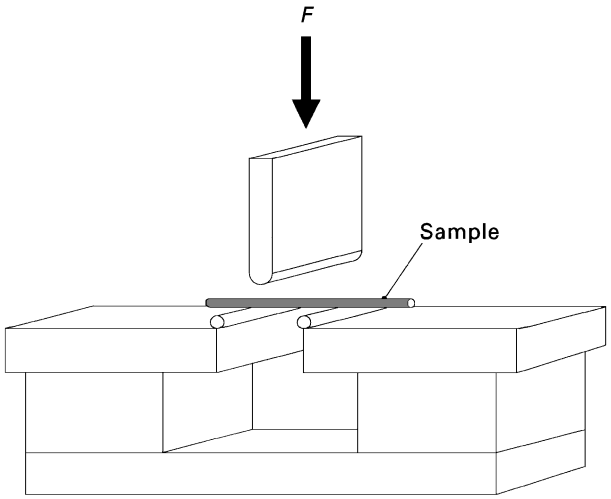

Figure 1 A schematic drawing of the bending strength test by the three-point bending method. The distance between supports was 22 mm for 1.1 mm diameter rods and 42 mm for 4.5 mm diameter rods. The testing speed was 5 mm min^{-1} for 1.1 mm diameter rods and 10 mm min^{-1} for 4.5 mm diameter rods. The radius of the testing part of the loading nose was 5 mm and the radius of the lateral supports was 1.5 mm.

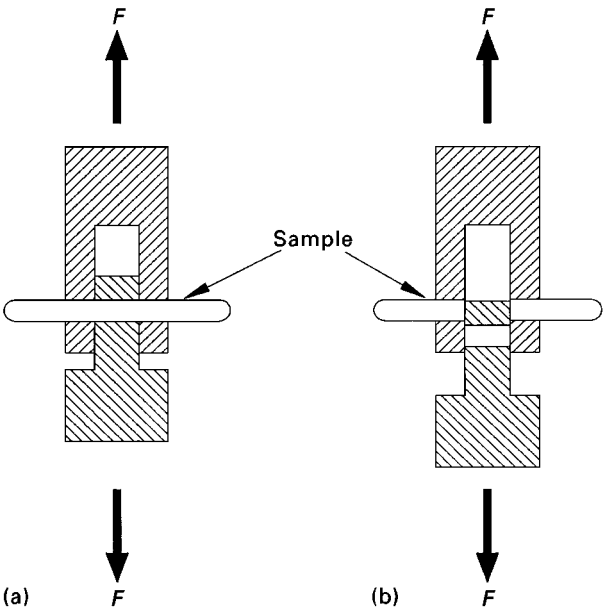

Figure 2 A schematic cross-sectional drawing of the shear strength test. During the test, the two parts of the hardened stainless steel test tool were pulled apart such that the test sample resting in a drill hole, and initially joining the parts of the tool together (a), was cut into three pieces perpendicular to the long axis of the rod (b). The size of the drill hole was chosen such that it was easy to push the test sample through the holes in the tool (approximate diameter of the drill hole 0.1 mm larger than the test sample). The testing speed was 10 mm min^{-1}.

were measured by the three-point bending method (Fig. 1), using a Lloyd 6000R Materials testing Machine (Lloyd Instruments PLC, Fareham, UK) at room temperature (22–23 °C). Triplicate samples were tested at each follow-up time. The measurements were performed on wet samples because drying of the incubated rods leads to a decrease of their strength. The support spans and crosshead speeds were 22 mm and 5 mm min^{-1}, and 42 mm and 10 mm min^{-1}, for 1.1 and 4.5 mm diameter rods, respectively. The radius of the loading nose was 5 mm and radius of each support was 1.5 mm. The bending strength was calculated using the following equation

$$\sigma_b = \frac{8F_{max}L}{\pi d^3} \quad (1)$$

where σ is the bending strength (MPa), F_{max} the maximum bending force recorded (N), L the support span (mm) and d the diameter of the rod (mm).

The bending modulus was calculated from

$$E = \frac{4L^3}{3\pi d^4}\left(\frac{\Delta F}{\Delta x}\right)\frac{1}{1000} \quad (2)$$

where E is the flexural modulus (GPa), $\Delta F/\Delta x$ the slope of the force–deflection curve of the initial linear section (N mm^{-1}), L the support span (mm) and d the diameter of the rod (mm).

The shear strength of the intact SR-PLA96 rods and after *in vitro* and *in vivo* exposure, was measured by means of a tool, which was constructed by modifying the standard BS 2782, Method 340B [29]. The tool consisted of two parts, which were joined together by the implant (Fig. 2). During the test, the parts were pulled apart using a Lloyd 6000R materials Testing Machine operating at a crosshead speed of 10 mm min^{-1}. Thus the implant, resting in a drillhole, was cut into three pieces perpendicular to the long axis of the rod (Fig. 2). The shear strength was calculated using

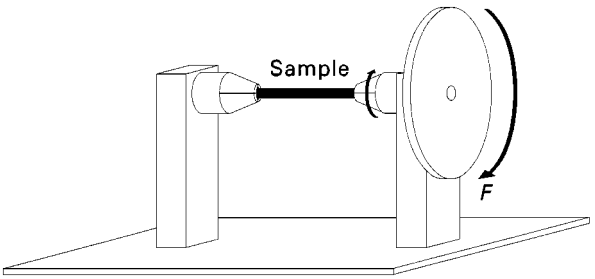

Figure 3 A schematic drawing of the torsion strength test. One end of the test rod was fixed to a stationary grip (standard Jacob's chuck) while the other end was mounted to a rotating grip and rotated clockwise at the speed of 0.2 rev min^{-1}. The distance between the grips was 20 mm for 4.5 mm diameter rods.

$$\tau_s = \frac{2F_{max}}{\pi d^2} \quad (3)$$

where τ is the shear strength (MPa), F_{max} the maximum force recorded (N) and the d the diameter of the rod (mm).

The torque strength of the 4.5 mm diameter intact SR-PLA96 rods and after *in vitro* and *in vivo* exposure, was measured by means of the tool shown in Fig. 3. One end of the rod was fixed to a stationary grip, while the other end of the rod turned by the rotating grip. The torque was transmitted to the rotating grip by means of a rotating wheel and driveshaft mounted to a bearing stand. The test device was connected to a Lloyd 6000R Materials Testing machine and the rotating wheel was turned by means of a chain

attached to the load cell at a rate of 0.2 r/min min^{-1}. The load pulling the chain was recorded and used for calculation of the torsion strength according to

$$\tau_t = \frac{16F_{max}R}{\pi d^3} \quad (4)$$

where τ is the torsion strength (MPa), F_{max} the force at yield (N, approximate rotation 90°), R the radius of the rotating wheel (= 40 mm), and d the inner diameter of the rod (mm).

2.5. Thermal analysis and molecular weight measurements

A Perkin–Elmer DSC-7 differential scanning calorimeter (DSC) (Perkin–Elmer Co., USA), calibrated with indium standards, was used to determine the heat of fusion of intact SR-PLA96 rods and after *in vitro* and *in vivo* exposure. The DSC was operated at a heating rate of 20 °C min^{-1}. Dry 6 ± 0.1 mg samples evacuated at room temperature for 3d, were used in each case. The samples were heated in a nitrogen atmosphere from room temperature to 200 °C (about 50 °C above the melting temperature to ensure melting of all crystallites), and the heat of fusion was estimated from the area enclosed by the DSC curve and baseline [30]. The level of crystallinity was estimated from the heat of fusion, assuming 93.7 J g^{-1} calculated by Fischer *et al.* [31] for perfectly crystalline P*L*LA. Duplicate samples were used in each case.

The solution viscosities (according to ASTM D 445-88) of intact SR-PLA96 rods and after *in vitro* and *in vivo* exposure were measured in chloroform at 25 °C with an Ubbelohde capillary viscometer (type 0a according to ASTM D 446). Inherent viscosities (η, dl g^{-1}) in a 0.1% solution (0.1 g dl^{-1}) were determined as an indication of molecular weight decrease during *in vitro* and *in vivo* exposure [32]. Viscosity-average molecular weights, M_v g mol^{-1} were estimated using the Mark–Houwink equation and parameters determined by Schindler and Harper for P*L*LA [33]

$$[\eta] = 5.45 \times 10^{-4} M_v^{0.73} \quad (5)$$

where η is the inherent viscosity, and M_v the viscosity-average molecular weight.

The initial strength values, crystallinity and M_v of sterilized SR-PLA96 rods can be seen in Table II.

TABLE II The initial strength values, crystallinity and viscosity-average molecular weight, M_v, of SR-PLA96 rods after gamma irradiation

	Rod diameter	
	1.1 mm	4.5 mm
Bending strength (MPa)	228	232
Bending modulus (GPa)	8.4	5.4
Shear strength (MPa)	152	140
Torque strength (MPa)	–	53
Crystallinity (%)	38	39
M_v (g mol^{-1})	49 175	53 879

2.6. Statistics

The effect of time on bending and shear strengths was estimated by fitting regression lines for both *in vivo* and *in vitro* experiments. Intercept terms were set equal and differences between the slopes were tested. In the case of non-significant *in vivo*/*in vitro* effect, a common regression line was fitted. Based on regression models, population mean and its 95% confidence interval (CI) at different times were estimated.

3. Results

3.1. Physical changes

The measured changes in inherent viscosity (i.v.), viscosity-average molecular weight, M_v and crystallinity of SR-PLA96 rods during *in vitro* and *in vivo* exposure expressed as observed means ± standard deviation (S.D.) can be seen in Table III.

3.2. Strength changes

The measured changes in bending modulus values of SR-PLA96 rods during *in vitro* and *in vivo* exposure

TABLE III Changes in inherent viscosity (i.v.), viscosity-average molecular weight, M_v and crystallinity of SR-PLA96 rods during *in vitro* and *in vivo* exposure. Values are means ± standard deviation (S.D). Raw material: i.v. 8.27dl g^{-1} M_v 534 007 g mol^{-1}

Rod diameter (mm)	*in vitro*/ *in vivo*		Follow-up time (wk)				
			0	3	6	12	24
1.1	*in vitro*	i.v. (dl g^{-1})	1.45 (0.03)	1.41 (0.02)	1.31 (0.03)	1.05 (0.00)	0.76 (0.02)
		M_v (g mol^{-1})	49175	47326	42789	31602	20297
		Cryst. (%)	37.9 (0.08)	38.60 (0.53)	38.70 (1.96)	40.00 (0.08)	46.10 (0.30)
	in vivo	i.v. (dl g^{-1})	1.45 (0.03)	1.46 (0.07)	1.28 (0.04)	1.07 (0.05)	0.73 (0.03)
		M_v (g mol^{-1})	49175	49640	41453	32430	19207
		Cryst. (%)	37.9 (0.08)	35.30 (0.83)	39.00 (2.04)	41.70 (0.08)	47.60 (0.83)
4.5	*in vitro*	i.v. (dl g^{-1})	1.55 (0.02)	1.49 (0.07)	1.43 (0.18)	1.20 (0.04)	0.83 (0.04)
		M_v (g mol^{-1})	53879	51042	48248	37945	22900
		Cryst. (%)	39.0 (0.35)	35.40 (0.14)	35.30 (0.78)	42.50 (0.71)	48.90 (1.27)
	in vivo	i.v. (dl g^{-1})	1.55 (0.02)	1.44 (0.07)	1.42 (0.02)	1.15 (0.05)	0.89 (0.05)
		M_v (g mol^{-1})	53879	48711	47786	35796	25198
		Cryst. (%)	39.0 (0.35)	35.00 (0.23)	37.90 (0.30)	42.10 (0.08)	46.20 (1.51)

expressed as observed means ± S.D. can be seen in Table IV.

3.2.1. 1.1 mm diameter rods

The measured changes in bending and sheer strength values of the 1.1 mm diameter SR-PLA96 rods during *in vitro* and *in vivo* exposure expressed as observed means ± S.D. can be seen in Fig. 4. The estimated line represents population mean (± 95% CI) of strength values.

Bending strength decreased fairly linearly with time and no statistically significant ($p = 0.5856$) differences between *in vivo* and *in vitro* groups were demonstrated. Decrease of bending strength (mean ± 95% CI) was estimated to be 0.65(±) 0.63 MPa wk^{-1} ($p = 0.0437$). Baseline estimate was 208.1 (±) 7.8 MPa and at 24 wk the population mean (± 95% CI) bending strength was 192.5 (±) 10.0 MPa.

Shear strength measurements showed no difference between *in vivo* compared with *in vitro* ($p = 0.3803$). The mean (± 95% CI) rate of decrease was minimal though statistically significant, 0.30 (±) 0.22 MPa wk^{-1} ($p = 0.0060$). The baseline estimate was 148.9 (±) 2.6 MPa and the mean (± 95% CI) shear strength at week 24 was 141.6 (±) 3.5 MPa.

TABLE IV Changes in bending modulus values of SR-PLA 96 rods during *in vitro* and *in vivo* exposure. Values are means (± standard deviation)

Diameter (mm)	*in vitro*/ *in vivo*	Bending modulus (GPa)				
		0 wk	3 wk	6 wk	12 wk	24 wk
1.1	*in vitro*	8.4 (0.5)	7.3 (0.3)	7.4 (0.4)	7.1 (0.9)	6.9 (0.2)
	in vivo	8.4 (0.5)	6.5 (0.7)	7.6 (0.6)	6.6 (0.3)	6.6 (1.4)
4.5	*in vitro*	5.4 (0.1)	4.9 (0.1)	5.8 (0.3)	6.6 (0.3)	6.4 (0.3)
	in vivo	5.4 (0.1)	5.1 (0.2)	5.9 (0.5)	5.6 (0.3)	6.6 (0.2)

3.2.2. 4.5 mm diameter rods

The measured changes in bending and shear strength values of the 4.5 mm diameter SR-PLA96 rods during *in vitro* and *in vivo* exposure expressed as observed means ± S.D. can be seen in Fig. 5. The estimated line represents population mean (± 95% CI) of strength values.

Bending strength decreased fairly linearly over time and there was no statistically significant difference between the *in vitro* group and the *in vivo* group ($p = 0.7416$). The mean rate (± 95% CI) of the decrease in bending strength was 1.72 (±) 0.48 MPa wk^{-1} ($p = 0.0001$). Baseline estimate was 243.5 (±) 5.7 MPa and at 24 wk mean bending strength was estimated to be 202.3 (±) 8.0 MPa.

Shear strength values tended to stay at a higher level *in vivo* longer than *in vitro*. Because of fluctuation in values from weeks 6 to 18, one common regression line was fitted. The mean (± 95% CI) rate of decrease was 0.16 (±) 0.18 MPa wk^{-1} ($p = 0.0738$). Baseline estimate was 136.3 (±) 2.1 MPa and at 24 wk 132.4 (±) 3.0 MPa.

The torsion strength values of the 4.5 mm diameter SR-PLA96 rods during *in vitro* and *in vivo* exposure expressed as observed means ± S.D. can be seen in Fig. 6. The estimated line represents population mean (± 95% CI) of torsion strength values. *In vitro* values of torsion strength were measured only upto 12 wk and torsion strength decreased linearly until this point. The difference in torsion strength decline between *in vitro* and *in vivo* just failed to reach statistical

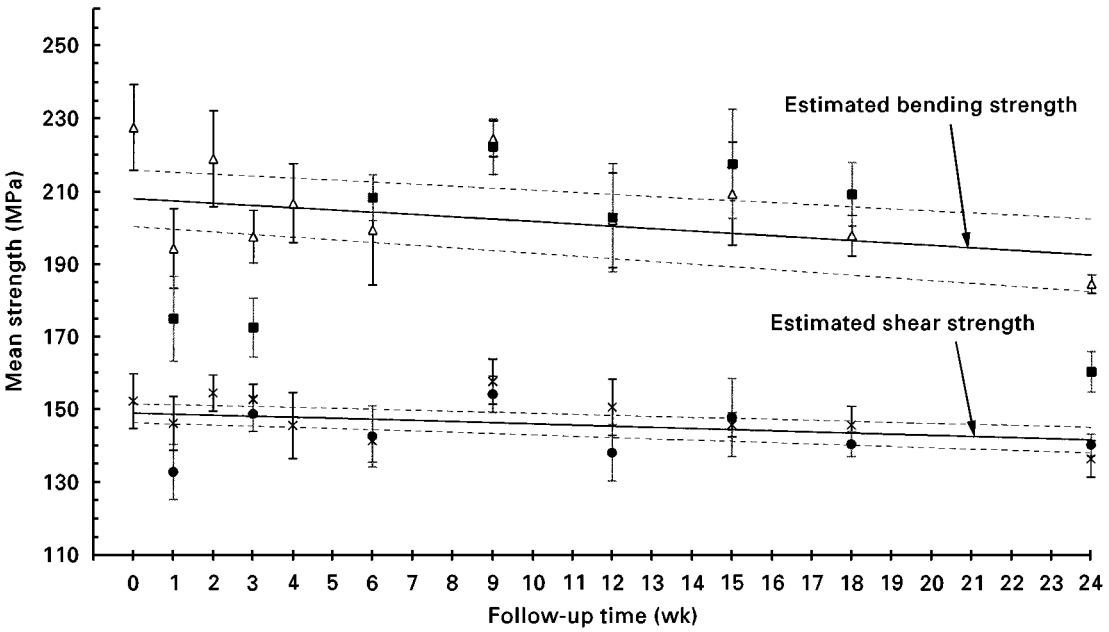

Figure 4 The strength values of the 1.1 mm diameter SR-PLA96 rods expressed as observed means ± standard deviation. The estimated line represents population mean ± 95% confidence interval of strength values. Bending: (△) *in vitro*, (■) *in vivo*. Shear: (*) *in vitro*, (●) *in vivo*.

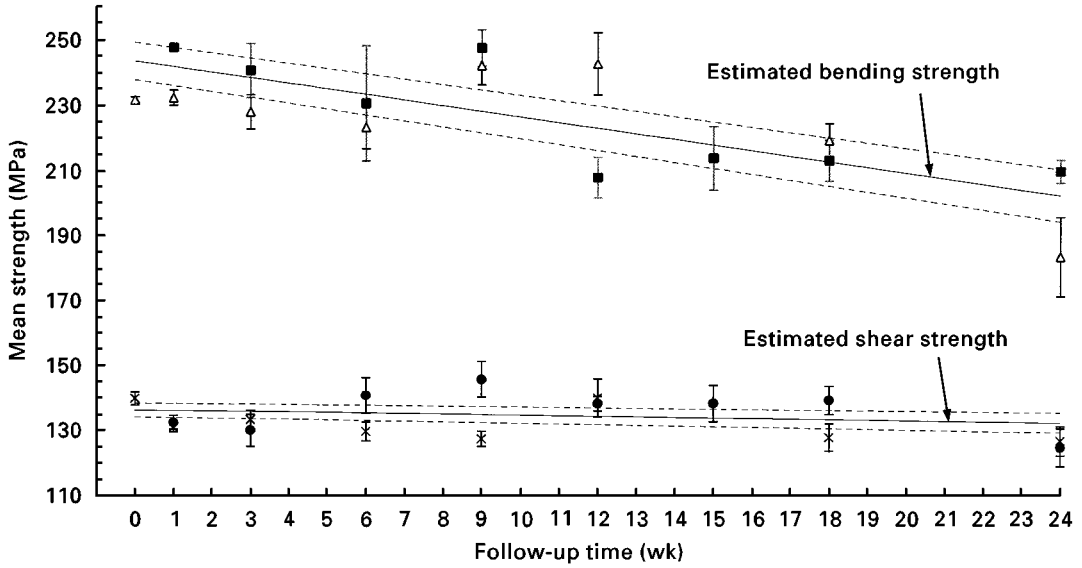

Figure 5 The strength values of the 4.5 mm diameter SR-PLA96 rods expressed as observed means ± standard deviation. The estimated line represents population mean ± 95% confidence interval of strength values. Bending: (△) *in vitro*, (■) *in vivo*. Shear: (∗) *in vitro*, (●) *in vivo*.

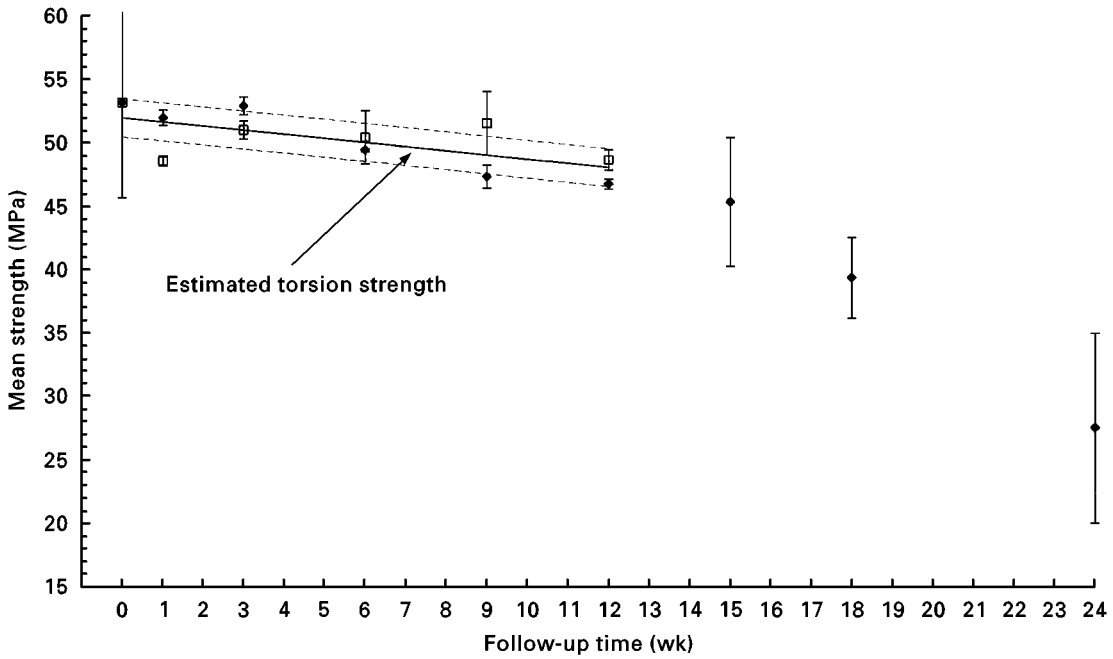

Figure 6 The torsion strength values (□) *in vitro* and *in vivo* of the 4.5 mm diameter SR-PLA96 rods expressed as observed means ± standard deviation. The estimated line represents population mean ± 95% confidence interval of torsion strength values.

significance ($p = 0.0692$). One common regression line was fitted and the mean (± 95% CI) rate of decrease was 0.33 (±) 0.21 Mpa wk^{-1} ($p = 0.0015$). Based on this model, the mean (± 95% CI) torsion strength at week 12 was 48.1 (±) 1.5 MPa. Compared to the baseline estimate of 52.1 (±) 1.5 MPa, reduction is minimal during the first 12 wk. After 12 wk, *in vivo* strength values decreased rapidly and the observed mean (± S.D.) torsion strength at week 24 was 27.5 (±) 7.5 MPa.

4. Discussion

The subcutaneous model was chosen for the *in vivo* study as it has been reported that there are no statistically significant differences between the strength retentions of the absorbable osteosynthesis rods in the bone compared with subcutaneous tissue [34, 35].

We found no significant differences in the strength retentions of the rods between *in vitro* and *in vivo* groups. There has been some controversy over the degradation mechanism of PLLA. Several authors have found no difference between *in vitro* and *in vivo* environments except when PLLA devices have been dynamically loaded [27, 36–38]. In these studies, degradation has been attributed solely to hydrolysis. Some studies, however, have reported a faster loss of strength *in vivo* and explained this phenomenon to be due to cellular enzymes and stress-cracking [35, 39, 40]. Gogolewski *et al.* [41] suggested there are two

different mechanisms in the degradation process of P*L*LA *in vivo*, i.e. non-enzymatic and enzymatic, the latter being more extensive at the later stage of the partially hydrolyzed polymer. Our study agrees with that of Cordewener *et al.* [27], who found no significant differences in degradation of PLA96 between the *in vitro* and *in vivo* measurements. Thus, the degradation process of PLA96 *in vivo* appears to occur through pure hydrolysis and a more extensive involvement of enzymes seems unlikely.

The initial observed bending strength in this study for the SR-PLA96 rods was 228 MPa for the 1.1 mm diameter rods and 232 MPa for the 4.5 mm diameter rods. At 12 wk, the bending strength was still close to the initial value in both rod sizes and at 24 wk at the level of the cortical bone (160–209 MPa versus 120–210 MPa, respectively) [42]. At 24 wk the 1.1 mm diameter rods had 81%/70% (*in vitro*/*in vivo*) and the 4.5 mm diameter had 79%/91% (*in vitro*/*in vivo*) of their initial observed bending strength left. Thus, our fibrillated SR-PLA96 rods retained their strength even longer than sintered 100% SR-P*L*LA rods successfully used in cortical osteotomies in rabbits [10, 33].

The initial bending modulus for the 1.1 mm diameter rods was 8.4 GPa and for the 4.5 mm diameter rods, 5.4 GPa. At 24 wk, the bending modulus of the 1.1 mm diameter rods had decreased by 18%/21% (*in vitro*/*in vivo*) but the bending modulus of the 4.5 mm diameter rods had increased by 19%/22% (*in vitro*/*in vivo*). These observed bending modulus values are close to that of bone. Therefore, the risk of osteoporosis from stress shielding, seen with metallic implants, can be avoided [2].

The shear strength did not markedly change during the follow up of 24 wk in either size of rod. Majola *et al.* [34] reported a slower rate of loss of the shear strength than that of the bending strength with the SR-P*L*LA rods. At 24 wk, the 1.1 mm diameter rods had 90%/92% (*in vitro*/*in vivo*) and the 4.5 mm diameter rods had 91%/89% (*in vitro*/*in vivo*) of their original observed shear strength left. Interestingly, the shear strength of our SR-PLA96 rods at 24 wk had the same value as fibrillated SR-P*L*LA rods had at 3 wk [43]. The latter rods have been successfully used in fixation of cortical osteotomies in rabbits [11].

The observed bending and shear strengths showed a temporary decline at 1–3 wk but then raised back to the original level. This is probably attributed to softening of the rods due to absorption of water and body fluids. The subsequent increase is expected to occur when the crystallinity of the rods increases.

The torsion strength was measured only on the 4.5 mm diameter rods. The initial observed torsion strength was higher than the initial torsion strength of the fibrillated or sintered 100% SR-P*L*LA rods mentioned above [43]. The SR-PLA96 rods retained their initial observed torsion strength surprisingly well, being 88% at 12 wk and 52% at 24 wk.

Crystallinity increased over time. It has been suggested that in the initial stages, the hydrolysis of P*L*LA and PLA96 begins in the amorphous regions, thus increasing the relative amount of crystalline regions and, consequently, the crystallinity [27, 37]. High crystallinity is probably one of the factors that makes P*L*LA particles very stable and not very susceptible to hydrolysis [13]. Although the crystallinity of PLA96 in our study increased, it was, however, still substantially lower when compared to crystallinity of pure P*L*LA as Bergsma *et al.* also have reported [25]. Lower crystallinity indicates enhanced degradation of PLA96 compared to 100% P*L*LA.

The M_v began to decrease as soon as the rods were implanted and was less than 50% of the original M_v within 18–24 wk. This is in accordance with earlier studies where the rate of molecular weight decrease was found to be fastest in the beginning of hydrolysis and slower towards the end of degradation [36, 38]. Decrease of molecular weight is the first sign of degradation before changes in strength values or macroscopic appearance are seen [44]. It has been suggested that the degradation rate increases significantly when the molecular weight of P*L*LA reaches 5000 daltons [13], but there have also been contrary observations [26].

Cordewener *et al.* [27] reported a complete loss of mechanical properties of as-polymerized PLA96 after 7 wk. The implants in the present study were manufactured by the self-reinforcing technique introduced by Törmälä *et al.* [8, 9]. The SR composite structure consists of a matrix, reinforced by fibers of the same substance, which results in a substantial increase in initial strength values for P*L*LA implants, as shown in the present study. Experimental studies and a review by Zhang *et al.* [20] have shown that the purity of the polymer samples is the most critical factor affecting the degradation rate of polylactides. Therefore, well-characterized commercial medical grade polymer, carefully purified from residual monomer, was used in this study.

In conclusion, the initial strength of the SR-PLA96 rods, as well as their strength retention, indicate that they can be adequate for fixation of cortical bone osteotomies. The follow-up period in this study should have been longer in order to demonstrate a major decrease in strength values, but 24 wk is sufficient for normal healing of cortical fractures or osteotomies. However, based on previous studies, the degradation of PLA96 is enhanced compared to P*L*LA [25, 27]. Consequently, we have continued to study this material in fixation of femoral cortical osteotomies with a long-term follow-up.

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