

Biomechanical Properties of the Superficial Fascial System

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Background: Surgical repair of the superficial fascial system (SFS) has been claimed to both increase wound strength and enhance surgical outcome through anchoring of deeper tissues.

Objective: The authors assessed the biomechanical properties of the SFS to determine whether repair of the SFS layer improved early and long-term postoperative wound strength.

Methods: Four complementary studies were conducted to study the dermis and SFS junctional architecture and connective tissue content: gross dissection using a dehydrating agent (Pen-Fix; Richard-Allan Scientific, Kalamazoo, MI), a histologic study with hemotoxylin and eosin staining, soft tissue radiography, and immunofluorescence staining. Freshly excised human abdominal and lower back/buttock tissues underwent a midline incision, followed by repair using dermal sutures only (DRM), dermal sutures plus SFS sutures (DRM/SFS) or repair of the SFS only (SFS). Fresh swine abdominal tissues were similarly excised and repaired. Biomechanical tests were undertaken to compare the ex vivo human and swine tissues. Three types of closure—dermal sutures only (DRM), dermal sutures plus permanent 0-braided nylon suture in the SFS (DRM/SFS/N), and dermal sutures plus absorbable 0-vicryl suture in the SFS (DRM/SFS/V) were also tested in an in vivo swine model.

Results: Immunofluorescence studies showed collagen and elastin content and ratios to be comparable in the dermis and SFS. In ex vivo studies of human abdominal and back tissues, cyclic creep did not vary significantly among the different types of repair. DRM/SFS repair had a significantly higher failure load than dermal repair alone in both human abdominal and back tissues. In the in vivo swine study, normal tissue had a significantly higher failure load than all repair groups. The wounds where SFS had been repaired in addition to dermis exhibited an increased tensile strength and, among these, the wounds closed with SFS repair with a nonabsorbable suture exhibited greater tensile strength compared to absorbable suture repair. However, no statistically significant difference was noted, due to the small sample size.

Conclusions: We have determined, using an ex vivo model, that repair of the SFS layer in addition to dermis repair significantly increases the initial biomechanical strength of wound repair. This has the potential to decrease early wound dehiscence. In our in vivo model, the use of a nonabsorbable suture to approximate the SFS demonstrated a trend toward increased long-term wound strength. We believe our studies provide scientific data documenting that SFS is a key contributory strength layer in the early postoperative period, and is likely to be a strength layer even in the later stages of wound healing. (Aesthetic Surg J 2006;26:395–403.)

The superficial fascial system (SFS), the connective tissue network that resides below the dermis, has been implicated as a pivotal structure in excisional¹⁻⁶ and noninvasive^{7, 8} body contouring procedures. Surgical repair of the SFS has been claimed to both increase wound strength and enhance surgical outcome through anchoring of deeper tissues. Lockwood¹ also suggested that repair of the SFS results in a stable scar that heals without migration.

Fascial anchoring techniques using permanent sutures operate on the assumption that the SFS is a viscoelastic

layer in possession of functional biomechanical properties. Since the SFS provides the major structural support for the skin and fat of the body, lifting procedures of the trunk and extremities could theoretically utilize the SFS for suspension. Repair of the SFS would be expected to diffuse the tension on the skin flap, lift areas of soft tissue ptosis, and provide longer-lasting support.⁹ Evidence to support these claims is largely anecdotal. While some surgeons claim that SFS repair more effectively closes dead space, and therefore has the potential to reduce seroma formation,⁹ the potential for introducing addi-

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tional sutures as a nidus for wound infection is hard to ignore. While Lockwood reported decreased scar width, this has not previously been quantified.¹

Elucidation of the role of SFS repair is especially relevant given the incredible rise in postbariatric weight loss body contouring procedures. According to the American Society of Plastic Surgeons statistics, there were approximately 68,000 body contouring procedures performed for massive weight loss patients in 2005.¹⁰ Postbariatric body contouring often involves large surface areas of tissue that are approximated under significant tension. Partial dehiscence is a common complication following these procedures. It would be useful to know whether repair of the SFS increases wound strength, since decreasing wound dehiscence is of primary interest.

There has been no conclusive scientific evidence that repair of the SFS layer increases biomechanical strength of the surgical wound. Under the assumption that the dermis and SFS are both viscoelastic tissues, they should be amenable to biomechanical testing. We aimed first to elucidate the architecture and ultrastructure of the dermis-SFS junction, characterizing the anatomic and biomechanical relationship. To assess the biomechanical properties of the SFS, we used both an *ex vivo* model of suture repair and biomechanical testing utilizing freshly excised human tissue, and a validated *in vivo* swine model. The aim of our study was to determine whether repair of the SFS layer improved both early postoperative and long-term wound strength.

Materials and Methods

Anatomy and ultrastructure

Four complementary studies were conducted to study the dermis and SFS junctional architecture and connective tissue content: gross dissection using a dehydrating agent (Pen-Fix; Richard-Allan Scientific, Kalamazoo, MI), a histologic study with hematoxylin and eosin staining, soft tissue radiography (xerogram), and immunofluorescence staining. For immunofluorescence staining, type III collagen and elastin, which are the primary components of connective tissue viscoelasticity¹¹ were studied. Collagen IV immunofluorescence was used to study basement membranes, which characterize tissue vasculature.

Ex vivo model

Freshly excised abdominal tissues (6 patients) and lower back/buttock tissues (5 patients) were obtained from patients undergoing elective body contouring after massive weight loss. The tissues were weighed, de-identi-

Table. Repair methods

1. Control (no incision or repair)
2. SFS with 0 braided nylon, dermal repair with 3-0 PDS monofilament,
3. Dermal repair alone with 3-0 PDS monofilament
4. SFS repair alone with 0 braided nylon

Twenty human abdomen specimens (n = 6 per method) and 24 human back specimens (n = 5 per method) underwent a midline incision and repair by one of these methods.

fied, and stored at 4°C in moist gauze. Tissue collection was executed in accordance with the Institutional Review Board policies at the University of Pittsburgh. The tissues were trimmed to obtain blocks measuring 6 × 24 cm. The repair groups underwent a midline incision perpendicular to the long edge, and underwent repair with 1 of 3 methods (Table). Intact tissue blocks without a midline incision served as the control.

The three types of closure were repair with dermal sutures only (DRM), repair with dermal sutures plus SFS sutures (DRM/SFS), and repair of SFS layer only (SFS). The dermis was repaired using interrupted intradermal, 3-0 monofilament absorbable sutures (Polydioxalone) spaced 1 cm apart. A 4-0 monofilament running subcuticular was added to simulate the clinical scenario. The SFS repair incorporated 0 braided nylon sutures placed 1 cm apart in an interrupted fashion, with each bite incorporating 1.5 cm of tissue from each edge of the incision. A single surgeon completed all of the repairs.

Establishment of a swine model

To validate a swine model and any application of our data to human tissue, freshly harvested samples of human abdominal tissue were compared to freshly harvested swine tissue using biomechanical tests. Fresh swine abdominal tissue was excised en bloc, divided into 6 × 24-cm blocks, then divided into the same 4 *ex vivo* groups as the human tissues: intact tissue (control), DRM, DRM/SFS, and SFS alone, with n = 4 in each group. Biomechanical tests were performed to compare the *ex vivo* human tissue to *ex vivo* swine tissue.

In vivo model

Four mature female pigs (approximately 200 lb) were purchased from Sinclair Research Center (Columbia, MO). Anesthesia was induced with pentathol (4 mg/kg, IM) and isoflurane (1%-1.5%) and was maintained with

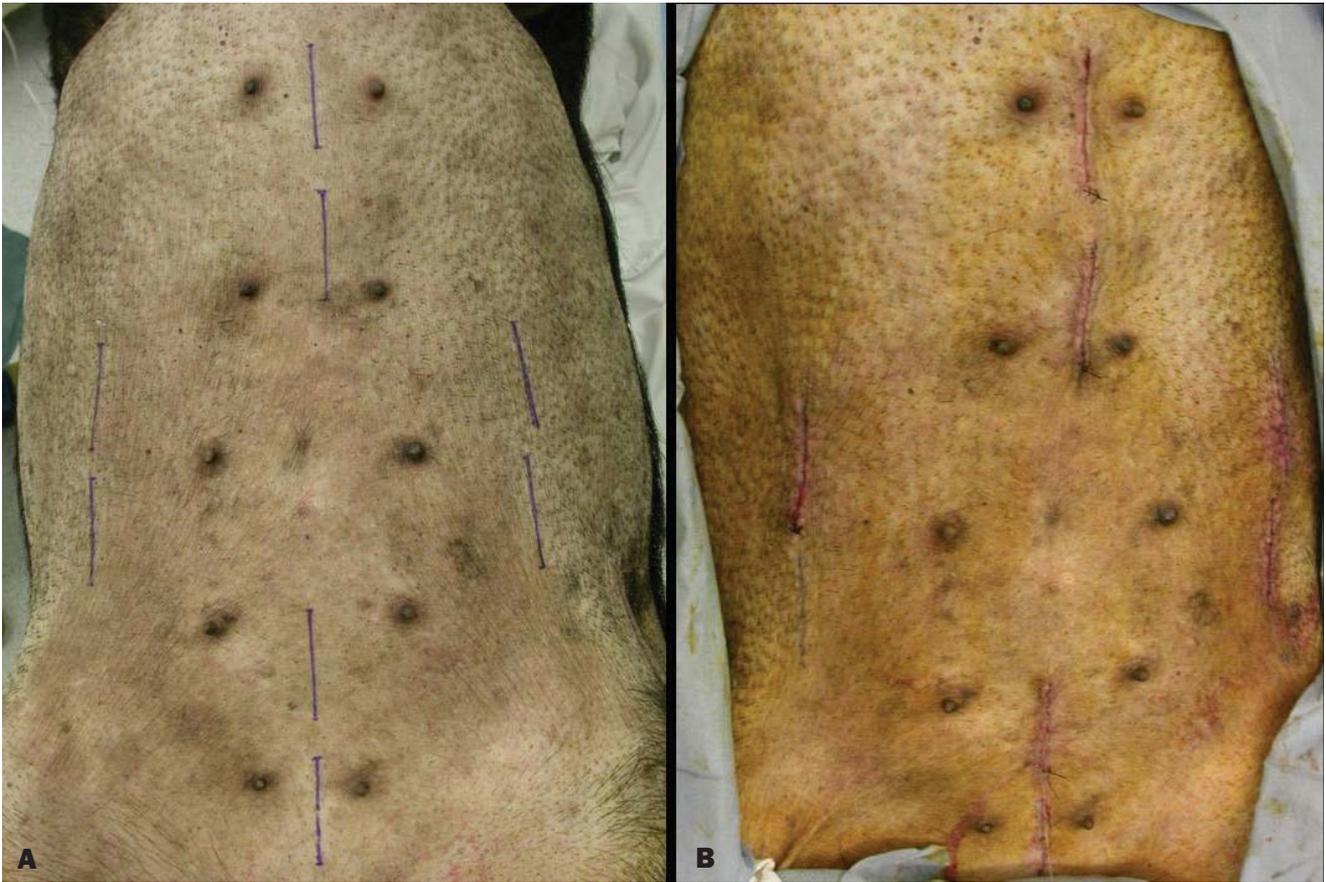


Figure 1. Distribution of excision sites. **A,** Six elliptical incisions, each measuring 6×2.5 cm were made along the drawn lines, sparing two lines as control. **B,** At each marked site, subcutaneous tissue was excised to the deep fascia and repaired using one of three closure methods.

isoflurane alone (4%). At the appropriate point in the study, animals were euthanized using pentobarbital (72 mg/kg, IV). All procedures were approved by the Animal Care and Use Committee of the University of Pittsburgh and complied fully with the American Veterinary Medical Association Panel on Euthanasia. After induction of anesthesia, each pig was placed on the operating bed on its dorsum and the abdomen and the flanks were prepped with Betadine (Purdue Pharma L.P., Stamford, CT). Six elliptical incisions, each measuring 6×2.5 cm, were made along the midline in the epigastric area and on the flanks in the lower abdomen, each at least 6 cm apart from the adjacent site (Figure 1). At each site, subcutaneous tissue was excised to the deep fascia. With careful hemostasis, the subcutaneous tissue adjacent to the wound margins of each ellipse was then undermined for a distance of 3 cm to create tissue flaps. In each animal, the 6 wounds were then randomly assigned among 3 closure techniques: repair with dermal sutures only (DRM), repair with dermal sutures plus permanent 0-braided

nylon suture in the SFS (DRM/SFS/N), and repair with dermal sutures plus absorbable 0-vicryl suture in the SFS (DRM/SFS/V). The SFS, dermal, and subcuticular repairs were performed in an identical manner to the ex vivo model. The animals were returned to their housing area, where their surgical wounds were inspected daily for any sign of infection, fluid collection, cellulites, or dehiscence.

Two animals were sacrificed at 6 weeks and the remaining 2 at 12 weeks. At the sites of previous incisions, 24×6 -cm pieces of tissue containing the overlying skin along with the entire underlying subcutaneous tissue were harvested such that the healed incisions were captured in the midline of each piece (Figure 2).

Biomechanical testing

The ends of the tissue samples were wrapped with gauze and placed into customized clamps designed with teeth to grip the ends of the tissue samples. The distance between each clamp was standardized at 7 cm. The tissue was tightly fixed in the clamps. The clamps were fixed to the

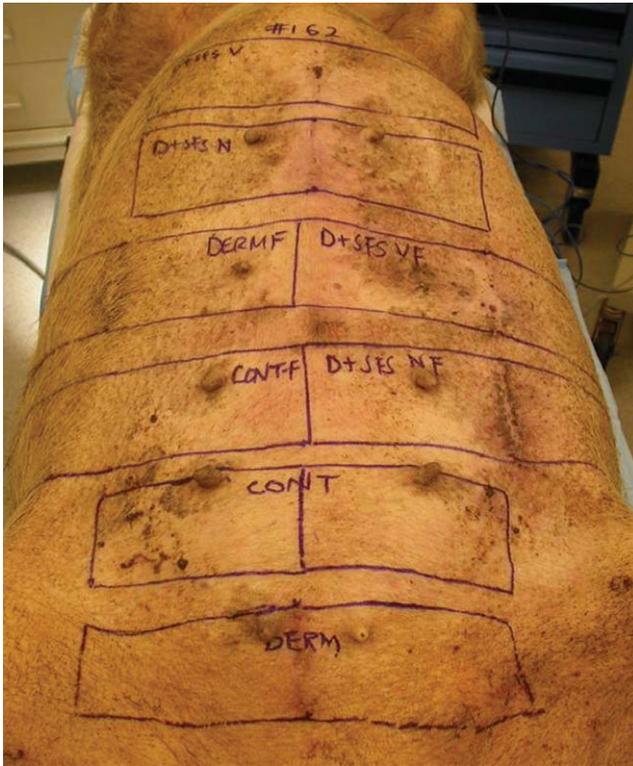


Figure 2. Tissue harvest from pig abdomen. Abdominal incision after 6 weeks. The strips to be harvested were designed such that the healed incision would be at the mid-length of each piece. Additional pieces were harvested to serve as controls.

crosshead and base of a materials testing machine (Instron, Model 4502, Canton, MA), and the tissue sample was adjusted to room temperature (Figure 3). Each specimen was centered and subjected to an initial preload of 5 N, and then preconditioned between 0 and 13 mm of elongation for 20 cycles at 50 mm/min. Biomechanical testing was performed after preconditioning. Cyclic creep (percent elongation), load to failure (N) and stiffness (N/mm) were determined using the following protocol, which contains modifications from previously published works.^{12,13}

Cyclic creep was determined by cyclically loading the complexes from 20 to 75 N for 20 cycles, followed by 20 minutes of recovery in an unloaded state. Creep was defined as the amount of elongation between the peaks of the first and last cycle of loading. After the recovery period, load to failure was determined by placing the tissue in a bidirectional strain along its long axis, at an elongation rate of 50 mm/min. The load-displacement curve was recorded. Ultimate load was defined as the maximum obtained load at the failure point of the tissue samples. In our study, ultimate load and load to failure were interchangeable.

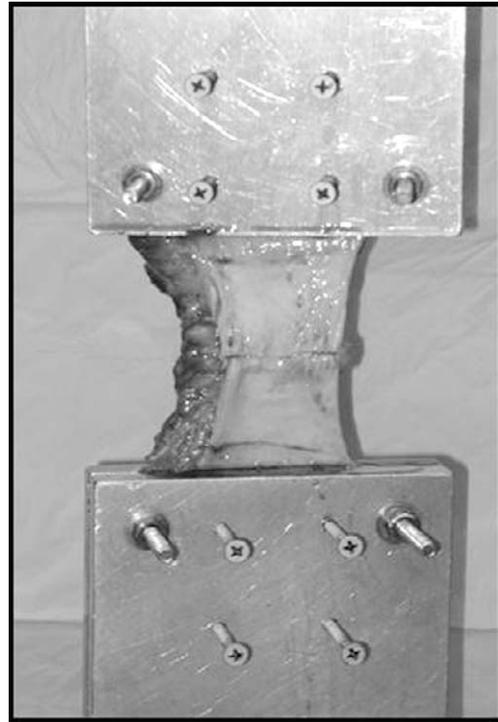


Figure 3. Tissue placement in tensiometer. The ends of human specimens were wrapped with gauze and were fixed in customized clamps designed with teeth to grip the tissue. The upper clamp was then mounted to the crosshead of the tensiometer while the bottom clamp was fixed to the base.

For each tissue specimen, stiffness was calculated by determining the slope of a line that fit the most linear portion of the load-displacement curve. All determined slopes resulted in an R^2 (squared correlation coefficient) greater than or equal to 0.80.

Paired t tests were used to detect the differences between the reconstructions of the tissue samples for each parameter (failure load, stiffness, creep, and initial stiffness). The statistical significance was set at $P < .05$.

Results

Anatomy and ultrastructure

The SFS, grossly and under light microscopy, did not appear distinct from the dermis at the sites of attachment (Figure 4). The SFS had two major structural components, the first being fibrous, thick columns and lattices that form the scaffold for the fat; the second consisted of papery, thin sheets that emerged from the fibrous SFS to encase adipose lobules. Between Scarpa's fascia and the skin, the fibers of the SFS primarily ran



Figure 4. Anatomy of dermal-SFS junction. Multiple junctions between the dermis and the SFS. Notice the web-like collagen tendrils that diverge away from the thicker SFS columns. These “webs” formerly encased large fat globules.

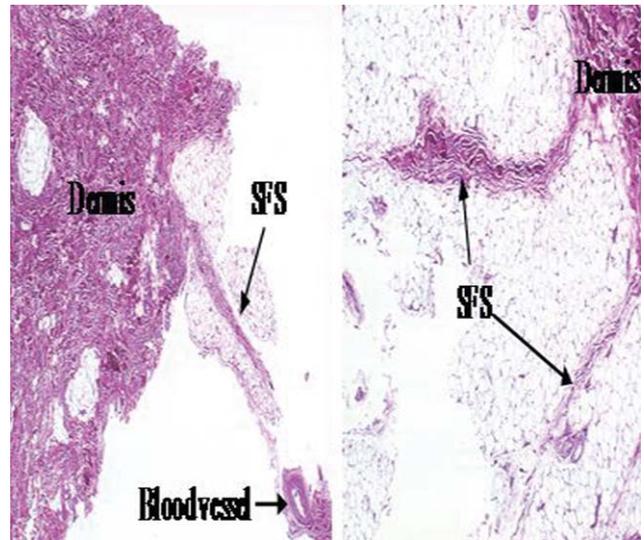


Figure 5. Hematologic study of the dermal-SFS junction. There is no distinct anatomic difference between the dermis and the SFS. In the deeper reticular dermis, the collagen fibrils gradually orient themselves to converge into septae that emerge perpendicular to the dermis. (H & E stain, $\times 20$.)

parallel to the direction of the columns and perpendicular to the dermis.

Histologically, the collagen and elastin fibrils of the deeper reticular dermis began to orient themselves in a manner more perpendicular to the epidermis near the dermal-SFS junction (Figure 5). The transition zones from the dermis to SFS appeared ordered; the collagen fibers gradually angled themselves to be oriented perpendicular to the epidermis.

In the immunofluorescence studies, the collagen and elastin content and ratios were comparable in the dermis and the SFS (Figure 6). The percent area of fluorescence indicating collagen III was mean 15.2% in the dermis, and 15.4% in the SFS, and elastin was a mean 3.1% in the dermis, and 3.7% in the SFS. The Type III collagen-to-elastin ratio was closely comparable in the two areas (Dermis: 4.17 ± 2.21 ; SFS: 4.24 ± 2.07 ; $n = 5$).

Ex vivo study

In human abdominal tissue, cyclic creep, or percent tissue elongation after 20 successive loading cycles, did not vary significantly among different types of repair (DRM: $57.0\% \pm 22.4\%$ vs DRM/SFS: $60.1\% \pm 26.1\%$). Neither repair group varied significantly from the control. In human lower back/buttock tissue, cyclic creep also did not vary among repair groups (DRM: $50.5\% \pm 13.7\%$ vs DRM/SFS: $37.5\% \pm 12.6\%$). Neither group was significantly different from the control.

In the stress-strain curve, load to failure correlated to the applied force at which the tissue lost its inherent strength. This correlated to a visual point at which the tissue repair split apart. In human abdominal tissue, DRM/SFS repair had a significantly higher load to failure than DRM (401.4 ± 44.9 N vs 260.6 ± 70.4 N; $n = 6$, $P < .05$). Both types of repair were significantly weaker than the control tissue (556.6 ± 187.2 N), and significantly stronger than SFS repair alone (105.8 ± 29.2 N) (Figure 7). In human back tissue, DRM/SFS repair had a significantly higher load to failure than DRM alone (524.2 ± 145.5 N vs 342 ± 71.3 N; $n = 5$, $P < .05$). Both types of repair were significantly weaker than the control tissue but significantly stronger than SFS repair alone (102.6 ± 74.4 N). Overall, there was a mean 53% greater force required to disrupt repair when the SFS was sutured along with the dermis.

Tissue stiffness was quantified by measuring the maximum slope of the stress-strain curve. Steepness of the curve is positively correlated to tissue stiffness. In the human abdominal tissue, there was no significant difference in tissue stiffness between DRM/SFS and SFS repair alone (13.2 ± 6.0 N/mm vs 12.9 ± 3.1 N/mm). Tissue stiffness did not vary significantly from control (18.7 ± 18.7 N/mm; $n = 6$). In human back tissue, tissue stiffness varied significantly between DRM/SFS and DRM repair alone (10.5 ± 1.0 N/mm vs 20.4 ± 3.0 N/mm). However, neither type of repair resulted in stiffness that differed

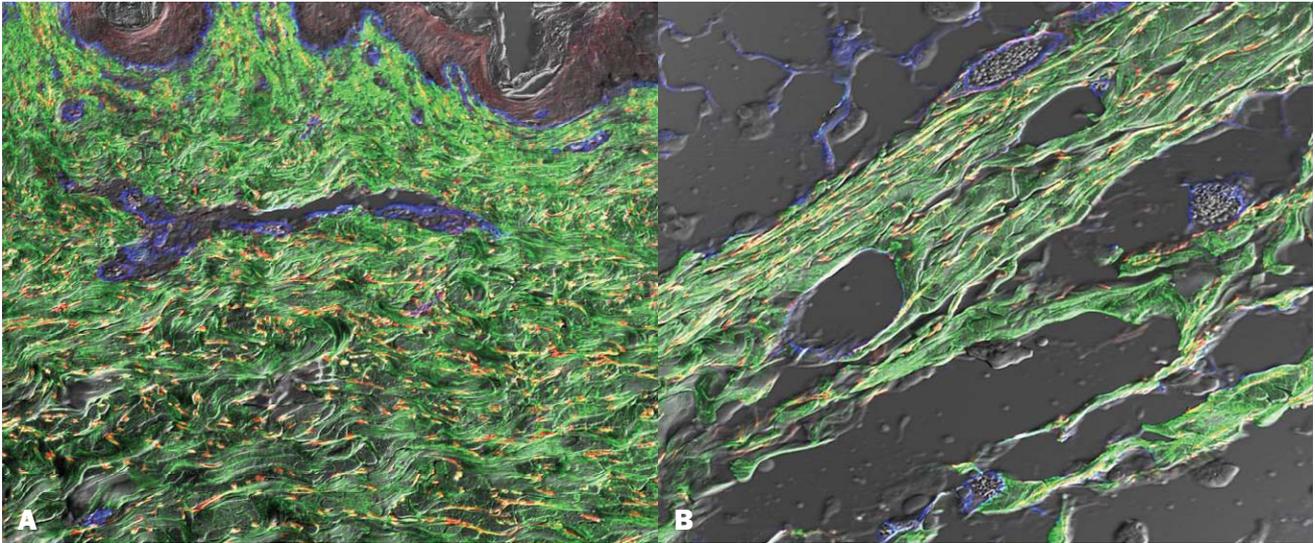


Figure 6. Immunofluorescence staining showing **A**, the dermis and **B**, an SFS fibril. The connective tissue composition of the dermis and SFS appears homologous under confocal microscopy. Green, Collagen III; red, elastin; blue, collagen IV.

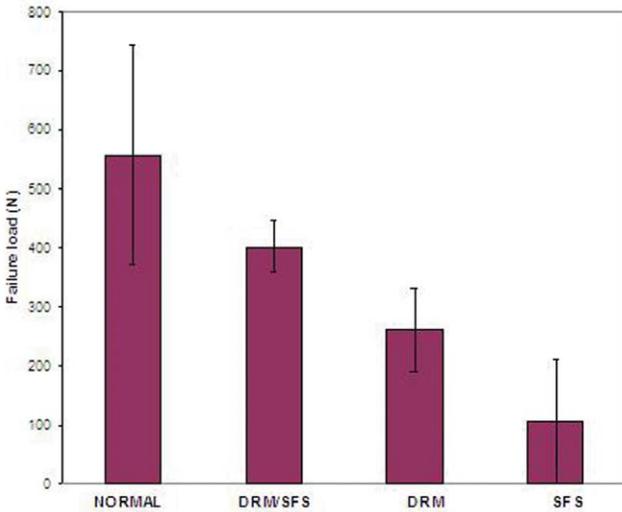


Figure 7. Load to failure of human abdominal tissue. DRM/SFS had a significantly higher load to failure than DRM alone. SFS repair had the lowest load to failure. Control tissue had the highest load to failure. All results were statistically significant ($P < .05$, $n = 5$)

significantly from control tissue stiffness (26.8 ± 9.5 N/mm).

Validation of in vivo model

To validate a swine model for application of swine data to human tissue, freshly harvested human abdominal tissue was compared to freshly harvested swine tissue using load to failure, stiffness, initial stiffness, and cyclic creep data. Figure 8 contains the results of failure load

comparison of control tissue, DRM, DRM/SFS, and SFS only repair between human and swine tissue. For both types of tissue, the “normal” (nonincised) tissue had the largest failure load, followed by DRM/SFS, DRM, and SFS, respectively. Using a paired *t* test, no significant difference was detected between human and swine tissue except for the control group ($P > .05$), indicating a significantly greater failure load for swine tissue. Significant difference was noted among the control, DRM, DRM/SFS tissue for both human and pig tissue. Failure load of SFS tissue was not different between human and pig tissue. Similarly, with the exception of the control tissue, the degree of stiffness and the amount of creep were not different between human and pig tissue for each of the repairs.

In vivo model

Tissue harvested from the pigs after 6 weeks and 12 weeks of repair were tested and compared (Figure 9). At both time points, normal tissue had the highest failure load followed by DRM/SFS/N, DRM/SFS/V, and DRM, respectively. The failure loads were higher at 12 weeks compared to 6 weeks. Using a paired *t* test, a significant difference was noted between normal tissue and all the repair groups at 6 weeks. However, no difference was found between groups in which the SFS had been repaired (DRM/SFS/V and DRM/SFS/N) versus not repaired (DRM). Similarly, no statistically significant difference was found between the absorbable versus nonabsorbable sutures in closing the SFS. By 12

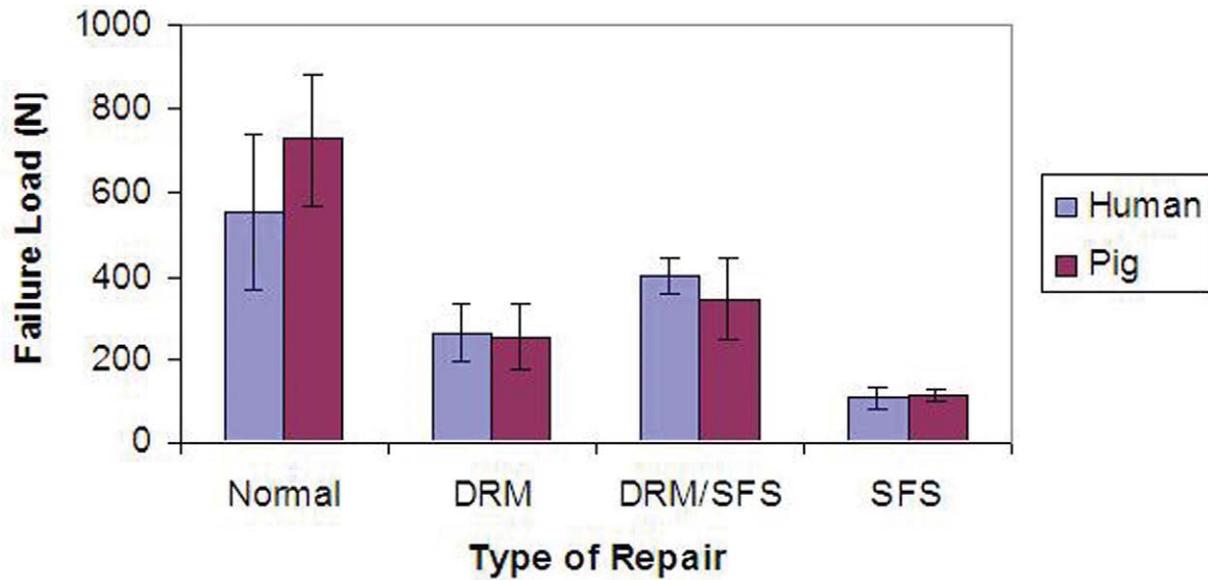


Figure 8. Comparison of failure loads between human and pig tissue. To assess the validity of the animal model, the load to failure of pig tissue was compared to that of human tissue repaired using same closure. Normal, Intact tissue (no incision); DRM, incision repaired at dermis only; DRM/SFS, dermis repaired plus SFS with nonabsorbable sutures; SFS, SFS repaired with nonabsorbable suture.

weeks post-repair, despite relative increase in failure of the repair groups, no statistically significant difference was detected among any of the groups, including the control.

Discussion

Our observations verify what Lockwood and others have described to be the biomechanical role of SFS.^{3-7,12} Repair of the SFS transfers tension from the dermis to the deeper tissues, minimizing tension to the skin flap. This biomechanical energy transfer is enhanced by the dermis-SFS junctional architecture. The continuity of the dermis to the SFS ensures a direct energy transfer, and the oblique and vertical orientation of the SFS septae disperses the energy in a direction perpendicular to the wound tension.

In our ex vivo study, repair of the SFS in addition to the dermis had a significant impact on wound tensile strength. The SFS layer alone withstood biomechanical testing, demonstrating a viscoelastic response pattern.¹¹ In addition, we were able to note that SFS sutures to a wound site did not necessarily impact other inherent biomechanical properties of a tissue, including tissue stiffness and creep. Our ex vivo results are most applicable to the very early postoperative period, prior to significant wound healing.

We then validated a swine model by testing similarly harvested tissue samples from human abdomen to that of

swine at time zero using intact and repaired samples. A similar pattern of biomechanical behavior was seen for both types of tissue, with the combination of DRM/SFS adding more tensile strength and stiffness compared to DRM alone. We thus concluded that the use of a swine model for a longitudinal study of SFS closure would be appropriate and applicable to human tissue.

In our in vivo study, the DRM/SFS group exhibited an increased tensile strength at 6 weeks. Among these, the DRM/SFS/N group exhibited greater tensile strength compared to the DRM/SFS/V group. By 12 weeks, the tensile strength in all wounds had increased significantly compared to 6 weeks, as would be expected from the normal wound healing process.¹² Again, DRM/SFS was shown to result in better tensile strength compared to DRM alone. Interestingly, the increase in tensile strength of DRM/SFS/N compared to DRM/SFS/V was more dramatic. This was likely due to complete absorption of vicryl sutures by this time point. Yet the use of absorbable suture in SFS still resulted in better tensile strength as opposed to dermis alone; this may be a result of better SFS approximation at the time of closure. Unfortunately, due the small sample size in this population, statistical significance was not achieved. Only a similar study with a larger number of animals could give a more definitive conclusion on the longitudinal effect of SFS repair versus no repair and use of absorbable versus nonabsorbable suture.

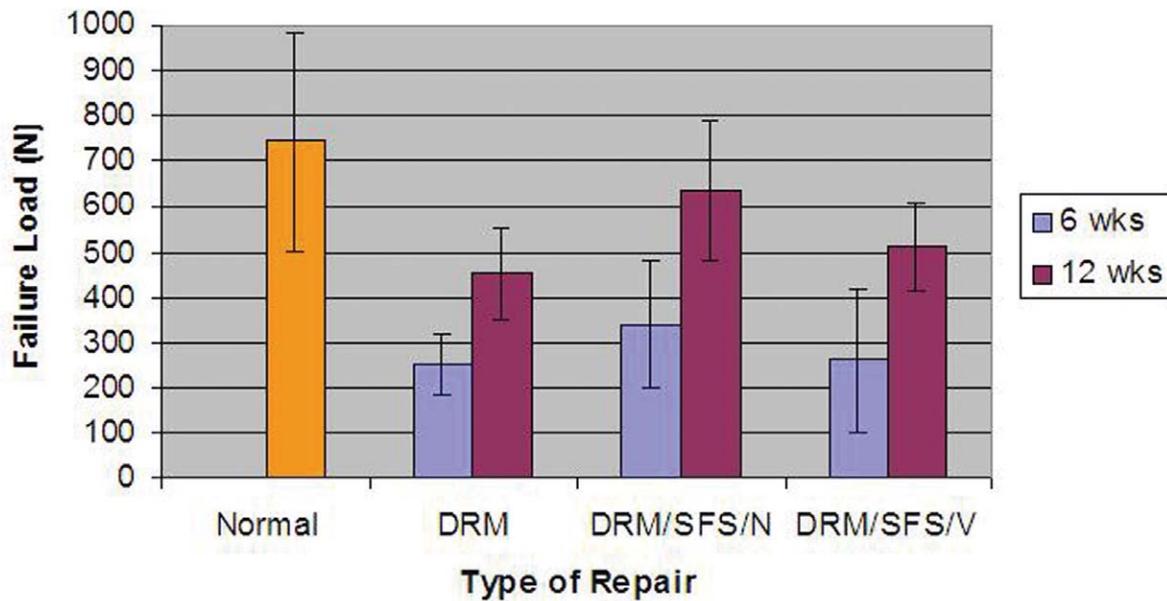


Figure 9. Failure loads at 6 and 12 weeks post-repair. The amount of force (load) that results in failure of each type of tissue is shown. For both time points, the largest failure load was required for normal tissue followed by DRM/SFS/N, DRM/SFS/V, and DRM, respectively. Normal, Intact tissue (no incision); DRM, incision repaired at dermis only; DRM/SFS/N, dermis repaired plus SFS with nonabsorbable sutures; DRM/SFS/V, dermis repaired plus SFS with absorbable sutures.

In the initial phases of healing, the wound has virtually no intrinsic strength. In the first 48 hours, the wound enters an inflammatory phase that consists of platelet formation, growth factor and chemoattractant release, and recruitment of granulocytes. Only after day 3 does the proliferative phase begin, when collagen and extracellular matrix components are secreted by the fibroblasts. At 6 weeks, 50% of the initial wound strength has returned, and the surgical site can withstand most moderate forces.¹⁴ We were interested in the early post-operative state, when wound strength is dependent on the quality of the wound closure. Increasing the wound strength during this phase could significantly decrease the incidence of wound dehiscence.

Biomechanics of the subdermal tissues have previously been studied using the superficial musculoaponeurotic system (SMAS). Skin and SMAS were determined to be viscoelastic, exhibiting the typical phenomena of creep, defined as stretching of tissue under constant tension, and stress relaxation, defined as gradual decrease in intrinsic tensile force of a tissue under constant stretch.¹⁵ Although the authors were focusing on the biomechanical role that the SMAS plays in facial rejuvenation, this study prompted us to further consider the SFS as a SMAS equivalent in trunk and extremities. As much as the SMAS plays a biomechanical role in maintaining the contour of facial structures and the integrity of wound

repair, the SFS may perform similar duties on trunk and extremities.

The strength of the early wound is dependent on the interplay and collaboration between sutures and tissues.¹⁶ The tissues must have intrinsic strength not only against gravitational and movement-related strains, but also against the strain of the sutures themselves. The placement of sutures in the SFS should incorporate a large number of individual SFS fibers. Our current investigation of SFS anatomy showed that the SFS fibrils emerge from the dermis somewhat irregularly, with gaps ranging from 4 to 9 mm between the fibrils. Therefore, wide bites of subcutaneous tissue with a large needle are necessary to capture multiple SFS fibers. Currently, there is no consensus as to the best type of sutures to place in the SFS, best direction of suture placement, or number of sutures that should be placed to optimize both appearance and wound strength. In our study, we standardized our SFS sutures to include 1.5 cm of tissue from the wound edge, with a slightly oblique placement. This is similar to Lockwood's¹ clinical description. To decrease experimental variability, a single surgeon performed all our repairs.

We have further affirmed that the SFS is not a passive collagenous scaffold that holds the fat. It is a dynamic, viscoelastic strength layer that plays a variety of roles in body contouring. First and foremost, the SFS maintains

body surface contours.¹⁷ Stretching of this viscoelastic layer is most likely responsible for cellulite, sagging, and much of post-bariatric weight-loss deformity. While the sagging skin in post-bariatric weight-loss patients is a frequent topic of discussion, the accompanying stretching of SFS is infrequently noted. The direct continuity of the dermal-SFS junction and the nearly identical connective tissue content make the SFS an extension of the dermis. When the SFS is incorporated into surgical repair, it enhances the initial biomechanical strength while effectively closing the dead space. This may serve to decrease the incidence of mechanical wound dehiscence compared to dermal sutures alone. A randomized prospective clinical trial would be necessary to confirm this clinical correlation. However, wide clinical experience favoring SFS sutures, combined with data in our study, supports the continued use of this technique.

In our paradigm, the biomechanical testing was performed in a manner that was largely parallel to the direction of the dermal collagen fibrils, but largely perpendicular to the direction of the SFS fibrils. We tested in this manner because it best simulated the clinical scenario. While it would be of interest to perform biomechanical testing on individual fibers of the SFS to assess their maximal viscoelastic potential, this is an extremely difficult pursuit. First, there was tremendous variation in the diameter of the fibers. Additionally, the multiple branching points on each SFS fiber present another biomechanical variable.

Conclusion

We have determined using an *ex vivo* model, that repair of the SFS layer in addition to dermis repair significantly increases the initial biomechanical strength of wound repair. This has the potential to decrease early wound dehiscence. In our *in vivo* model, the use of a nonabsorbable suture to approximate the SFS demonstrated a trend toward increased long-term wound strength. This suggests the possibility of enhanced long-term tensile strength and, thus, more consistent and lasting aesthetic results in clinical body contouring. We believe our studies provide scientific data that SFS is a key contributory strength layer in the early postoperative period, and is likely to be a strength layer even in the later stages of wound healing. ■

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