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## **Inflammatory Patterns and Cellular and Vascular Permeation in Rat Abdominal Wall Defects Using Alloderm, Permacol and CollaMend for Repair**

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**Division of Plastic Surgery**

**Inflammatory Patterns and Cellular and Vascular Permeation in  
Rat Abdominal Wall Defects Using Alloderm, Permacol and CollaMend for Repair**

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**BACKGROUND:** Abdominal wall defects secondary to hernias, trauma or disease are a common problem in surgery. Complications of closing these defects with synthetic meshes include foreign body reactions, infection, and adhesions to abdominal viscera. To minimize these problems, several acellular dermis derived collagen products are being used clinically for tissue replacement/reconstruction and repair of abdominal wall defects, including both animal (bovine, porcine) and human derived products, chemically crosslinked or non-crosslinked. While these implants are in use clinically, the inflammatory response each causes, cellular and vascular permeation into the implant, and relative amount of implant remodeling have not been completely characterized.

**PURPOSE:** The purpose of this study is to compare Alloderm (LifeCell Corporation, Branchburg NJ), Permacol (Tissue Science Laboratories, Andover, Ma) and CollaMend (C.R. Bard, Murray Hill, NJ) in a rat model, using histologic staining to evaluate the patterns of inflammatory response, cellular and vascular permeation into implant, and relative amount of implant remodeling.

**METHODS:** Abdominal wall defects were created in 127 Sprague Dawley rats by removing a one centimeter by three centimeter portion of rectus muscles lateral to the midline. Rats were randomized into four groups, and the defect was repaired with an inlay graft of one centimeter by three centimeter sections of Alloderm, Permacol and CollaMend; the fourth group served as the control and the defect was closed primarily. 4.0 Prolene was used to close the abdominal wall defect in all rats; the skin was closed in a running subcuticular fashion with 4.0 PDS. Rats were sacrificed at time intervals of three days, one week, two weeks, one month, three months and six months. The defect area was explanted, including the implants with a surrounding muscle strip, placed into formalin, and two representative samples from each group at each time point were sectioned. Sections were stained with Hemotoxylin and Eosin (H&E), Mason's Trichrome (stains cell nuclei and cytoplasm red, collagen blue), and Verhoeff stain (stains mature elastin black.)

**RESULTS:** Three distinct inflammation patterns were noted in the Permacol, CollaMend and Alloderm groups, compared to the control group and to one another. Cellular infiltration peaked much earlier in the Alloderm implants with blood vessel formation in the implant identifiable by 14 days and evidence of thick channels of cellular infiltrate pushing apart the implant matrix. Both the Permacol and CollaMend implants developed distinct patterns of inflammation on their ventral and dorsal portions, but these thick cellular coats did not appear to "send" cells into the implant. By one month in the CollaMend implants, the ventral coat of cells was totally disconnected from the implant itself, and the implant as a whole was only sparsely infiltrated by cells. The Permacol group at one month had

only a few cells at the middle of implant, though several strong channels of cellular penetration at the lateral edges of the implant were apparent, as well as a few blood vessels. In all three experimental groups, copious inflammation was seen at the junction between the native rectus fascia and implant. Infiltration of the implant originating from this large collection of cells was seen, in varying degrees, in all three implant groups. Side by side histologic photographs of the implant groups at various time points will be included in the presentation.

**CONCLUSIONS:** Alloderm, Permacol and CollaMend implants have all been described for use in abdominal wall reconstruction, but the materials have very different inflammatory patterns as well as rates and types of cellular and vascular permeation in a rat model.