

Introduction

A tremendous need exists for a reliable procedure that produces lasting aesthetic and functional results for soft tissue defects of the head and neck. Autologous fat grafting has many attractive characteristics: ease of harvest with minimal donor site morbidity, non-immunogenicity, and favorable physical characteristics. However, unpredictable long-term results limit the application of autologous fat grafting.3 Autologous fat grafting typically yields 50% - 75% of the graft volume at one year, with varying results even between animals of the same strain. For this reason, the future of fat grafting is dependent on modulation of the resorption process.4

Given its efficacy in these other systems, we hypothesized that fibrin glue might enhance fat survival and for other xenografting studies, but it is not devoid of all immune function.5 Macrophage, antigen presenting cell, natural killer cell and complement function is significantly impaired in this model, and therefore, strategies that (1) enhance more rapid neovascularization, (2) transiently provide oxygen or nutrition, or (3) raise the adipocytes’ toxicity, and matrix formation which dictates cell behaviors including shape, migration, proliferation and metabolism. Although fibrin glue decreased resorption in this study, there are still limitations as to why the resorption rates are so high in this model. Inflammation may play an important role. The atherogenic murine has been widely used for testing fat graft survival and for various grafting studies, but it is not well defined as all immune functions. Macrophage, antigen presenting cell, natural killer cell and complement function is not affected in this mouse. Thus, there is a greater opportunity for immune system involvement in this model than in human autologous transplantation.

Discussion

Autologous fat grafting may be the ideal soft tissue volume restoration technique in reconstructive and cosmetic plastic surgery applications. However, its major drawback is resorption over time that leads to a primary biologic insult that most likely drives fat graft resorption is ischemia due to a disrupted vascular supply. Therefore, strategies that (1) enhance more rapid neovascularization, (2) transiently provide oxygen or nutrition, or (3) raise the adipocytes’ toxicity, and matrix formation which dictates cell behaviors including shape, migration, proliferation and metabolism. Although fibrin glue decreased resorption in this study, there are still limitations as to why the resorption rates are so high in this model. Inflammation may play an important role. The atherogenic murine has been widely used for testing fat graft survival and for various grafting studies, but it is not well defined as all immune functions. Macrophage, antigen presenting cell, natural killer cell and complement function is not affected in this mouse. Thus, there is a greater opportunity for immune system involvement in this model than in human autologous transplantation. From this and other experiments as well as clinical experience, the significant resorption associated with fat grafting is the primary limitation to consider when using fat for soft tissue volume in the head and neck. Further investigation into the pathways responsible for this resorption is necessary in order to modulate and ultimately improve graft take. The primary biologic insult that most likely drives fat graft resorption is ischemia due to a disrupted vascular supply. Therefore, strategies that (1) enhance more rapid neovascularization, (2) transiently provide oxygen or nutrition, or (3) raise the adipocytes’ toxicity, and matrix formation which dictates cell behaviors including shape, migration, proliferation and metabolism. Although fibrin glue decreased resorption in this study, there are still limitations as to why the resorption rates are so high in this model. Inflammation may play an important role. The atherogenic murine has been widely used for testing fat graft survival and for various grafting studies, but it is not well defined as all immune functions. Macrophage, antigen presenting cell, natural killer cell and complement function is not affected in this mouse. Thus, there is a greater opportunity for immune system involvement in this model than in human autologous transplantation.

References


Results

All grafts demonstrated volume resorption over time. Within fourteen weeks, approximately 87% of implanted fat graft volume resorbed in the control group, versus 75% in the treatment group (Figure 5C).

Histologic sections of the grafts revealed that the tissue remaining after fourteen weeks was composed of adipocytes with intact cellular morphology and significant in-growth of fibrous tissue.

The graft-host tissue interface was evaluated by histologic section and found to include blood vessels in-growth in both groups (Figure 6C). No fibrinoid eschares were visible, but some inflammatory infiltrate was noted in both treatment groups (Figure 6D).

In the ultrasonic evaluation of the grafts, cystic areas were more commonly found in the treatment group. Cyst volumes were measured and subtracted from the gross volume measurements yielding volumes of true remaining grafted fat.

Figure 5C: Graph of fat graft resorption over 24 weeks. Series 3 (FG treatment group, series 3 with control).