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Optimal Fat Graft Sizing with the 'Micronizer' System

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INTRODUCTION: It has been shown that to ensure fat graft survival, it is important that fat cells do not exceed 1mm in diameter, but at the same time contain a high concentration of stromal vascular fraction. A method of harvesting large sized fat grafts and reducing them in size to less than 1mm is described.

METHODS: The Micronizer system
Fat is harvested with a 4mm cannula

1. The lipo-blade attaches to the canister.
It has a 4mm inner diameter containing a blade that cuts the fat to 2mm particles .

2. The 2.4mm micronizer cuts 2 mm particles to 1mm particles (Microfat) .
Microfat can be injected atraumatically through a 1mm cannula.
3. The 1.4 micronizer cuts 1mm particles to 0.5 mm Fat particles, which can then be injected atraumatically through a 0.7 mm cannula. The fat can be further reduced in size by passing the fat back and forth several times. (Ultra micro fat)
4. To obtain so called nano fat , the fat is passed through multiple times and then strained. Depending on the strainer size, the fluid can be injected through a 27g or 30 g needle.

RESULTS: Using the micronizer system, it is possible to harvest fat with a 4-5 mm cannula and by means of the micronizer system reducing the size of the fat grafts to 2mm, 1mm and 0.5mm and even smaller if required. As the grafts are cut from 4-5 mm particles they contain a higher SVF content than fat harvested with a 1mm cannula. The micronizer is the only system having a single ultra sharp blade that actually cuts the fat particles with minimal trauma. Furthermore the fat particles can be cut to the specific size that will passthrough the selected cannula or needle atraumatically.

CONCLUSION: 4mm fat particles containing a higher concentration of SVF than 1mm particles ,can be reduced in size to less than 1mm using the Micronizer system.

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Syngeneic Adipose-Derived Stromal Cells Modulate the Immune Response within Decellularized Adipose Tissue Scaffolds in a Murine Subcutaneous Implant Model

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INTRODUCTION: Despite a remarkable capacity to expand and regress throughout mammalian life, adipose tissue typically fails to regenerate following reconstructive surgeries or tissue injury and is often replaced by scar tissue. Biomaterials, including decellularized adipose tissue (DAT), have shown promise for stimulating adipose regeneration in pre-clinical models, with an enhanced response often reported when the scaffolds are seeded with adipose-derived stromal cells (ASCs). While ASC-based therapies have shown promise for a range of applications, the extent and mechanisms through which ASCs support tissue regeneration in vivo are not well characterized.

METHODS: An established decellularized adipose tissue (DAT) delivery platform was employed to investigate the pro-angiogenic and immunomodulatory effects of transgenic mouse syngeneic DsRED+ donor ASCs within a subcutaneous implant model in immunocompetent C57BL/6 mice, with endpoints up to 8 weeks post-implantation. It was hypothesized that ASC seeding would promote a more regenerative macrophage phenotype and enhance localized vascular perfusion, resulting in improved scaffold integration compared to unseeded scaffolds. Flow cytometry was used to assess macrophage phenotype and track ASCs, and fluorescence-activated cell sorting enabled isolation of these populations for proteomics assessment via biological mass spectrometry. The integration of scaffolds with surrounding tissue was assessed histologically, and vascular perfusion was measured using a μCT- based angiography approach.

RESULTS: Initial proteomics data indicate that ASCs modulate cell surface receptor and extracellular matrix protein expression over time; however, few ASCs persisted in the tissue beyond 2 weeks post- implantation. Histological assessment showed qualitatively similar tissue integration between seeded and unseeded scaffolds over time. While vascular perfusion was not affected by ASC seeding, a more regenerative macrophage phenotype was noted in the seeded group, but only up to 2 weeks post- implantation while ASCs were most abundant. Interestingly, DsRED fluorescence was detected in a small subset of macrophages, indicative of a population that had phagocytosed ASC-derived protein. Notably, these macrophages displayed a more regenerative phenotype than their DsRED- counterparts.

CONCLUSION: ASCs modulated macrophage phenotype within the DAT scaffolds at early time points, potentially mediated in part through their phagocytosis. Investigation of strategies to increase the localized persistence of ASCs in vivo may help to augment cell-mediated regeneration.