In stem cell-based strategies for disc regeneration, the potential rejection of the implanted cells into the host's body remains a concern. Adipose tissue represents an abundant and easily accessible source of adult stem cells with the ability to differentiate along multiple lineage pathways and potentially able to facilitate treatment of an early degenerating disc. Rejection of implanted stem cells by host immune system has been a major concern in the field of stem cell-based therapies and is the focus of this study. The specific purpose of this study was to determine if ADHSC express major histocompatibility complexes (MHC 1 and 2) and if so whether ADHSC show an immune response after co-culture with T-cells.

Methods: Adipose-derived stem cells (ADSCs) were obtained from a commercial source (ZenBio, Research Triangle Park, NC). ADSCs were characterized for both stem cell and immune surface markers using flow cytometry. Briefly, ADSCs were incubated with fluorochrome conjugated antibodies for 15 minutes, washed 3 times, and fixed with 1% paraformaldehyde. Peripheral blood mononuclear cells (PBMCs) were isolated from heparinized whole blood using Lymphocyte Separation Medium (Mediatech, Manassas, VA). ADSCs were plated 4 hours prior to the plating of PBMCs to allow for adherence. PBMCs are counted prior to plating and after 3 days in culture using hemocytometer. PBMCs are activated using 0.1ug/ml of phorbol-12-myristate-13-acetate (PMA) (Sigma-Aldrich, St. Louis, MO).

Results: The ADSCs were determined by flow to be CD11c-, CD29+, CD44+, CD49+, CD90+, MHC I+, MHC II-, CD80-. Co-culturing of ADSCs with PBMCs yielded no PBMC expansion after 3 days in culture. Proliferation of PBMCs is observed after 3 days when stimulated with 0.1 ug/ml of PMA, but the PMA-induced proliferation is reduced when PBMC is co-cultured with ADSCs.

Discussion and conclusion: ADSCs have surface expressions of MHC class I molecules but not MHC class II or its co-stimulator molecule CD80. Co-culture of ADSCs together with PBMCs have demonstrated ADSCs alone will not cause the proliferation of the T-cell population within PBMCs. This suggests that implantation of ADSCs will not cause rejection by the host. More surprisingly, ADSCs were able to modulate the stimulation of T-cell proliferation by PMA. This suggests an immunomodulatory role for ADSCs, and that they may be a potential therapeutic agent with autoimmune disease. In vivo experiments are needed and are ongoing to confirm the in vitro findings in this abstract. Allografts of ADSCs may be particularly interesting in cell-based treatment of intervertebral disc disease.