UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, DC 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934

Date of Report: May 29, 2015
(Date of earliest event reported)

BIORESTORATIVE THERAPIES, INC.
(Exact Name of Registrant as Specified in Charter)

Delaware	000-54402	91-1835664
(State or Other Jurisdiction of Incorporation)	(Commission File No.)	(IRS Employer Identification Number)

40 Marcus Drive, Melville, NY	11747
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code: (631) 760-8100

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

____ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
____ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
____ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
____ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01 Regulation FD Disclosure.

BioRestorative Therapies, Inc. (the "Company") has prepared presentation materials (the "Presentation Materials") that management intends to use from time to time on and after May 29, 2015 in presentations about the Company's business. The Company intends to use the Presentation Materials, possibly with modification, at the 21st Annual Meeting of the International Society for Cellular Therapy being held between May 27, 2015 and May 30, 2015 and may use the Presentation Materials in other presentations to current and potential investors, lenders, creditors, insurers, vendors, customers, employees and others with an interest in the Company and its business.

The information contained in the Presentation Materials is summary information that should be considered in the context of the Company's filings with the Securities and Exchange Commission and other public announcements that the Company may make by press release or otherwise from time to time. The Presentation Materials speak as of the date of this Current Report on Form 8-K. While the Company may elect to update the Presentation Materials in the future to reflect events and circumstances occurring or existing after the date of this Current Report on Form 8-K, the Company specifically disclaims any obligation to do so. The Presentation Materials are furnished as Exhibit 99.1 to this Current Report on Form 8-K and are incorporated herein by reference. The presentation materials will also be posted in the Investor Relations section of the Company's website, www.biorestorative.com for 90 days.

The information referenced under Item 7.01 (including Exhibit 99.1 referenced in Item 9.01 below) of this Current Report on Form 8-K is being "furnished" under "Item 7.01. Regulation FD Disclosure" and, as such, shall not be deemed to be "filed" for the purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section. The information set forth in this Current Report on Form 8-K (including Exhibit 99.1 referenced in Item 9.01 below) shall not be incorporated by reference into any registration statement, report or other document filed by the Company pursuant to the Securities Act of 1933, as amended, except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

99.1 Presentation Materials.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

BIORESTORATIVE THERAPIES, INC.

Dated: May 29, 2015

By: /s/ Mark Weinreb
Mark Weinreb
Chief Executive Officer

Tissue Engineered Brown Adipocyte Constructs

Francisco Silva[1], Dolly Holt[2], Vanessa Vargas[1], Christian Elabd[1], David Bull[2], Amit N Patel[2]

BioRestorative Therapies Inc, NY, USA[1], Division of Cardiothoracic Surgery, [2] University of Utah, Salt Lake City, UT , USA 84132

Abstract

Many treatment modalities exist including reducing calorific intake, exercise, and medicaments [1, 2]. However, these methods substantially depend upon patient compliance and often fail. We explored a surgical option independent of patient compliance that may work to reduce the adipose burden upon patients. This strategy involves the use of brown adipose tissue, which has been shown increase energy intake and metabolize fat [3, 4]. Brown adipose tissue is gaining increased focus and has been identified as a novel target to treat obesity [5]. Brown adipose tissue is relatively prevalent in newborns, but decreases with age leaving only small depots in the cervical, supraclavicular, mediastinal, paravertebral and suprarenal regions in adults [6].

We have developed a strategy to isolate human neonatal brown adipose precursor cells and differentiate them into brown adipose-like cells. Brown adipose cells produce many intracellular lipid vesicles making them too fragile to passage, inject or implant [6]. We therefore developed a tissue engineering strategy utilizing human adipose tissue scaffolds to house differentiated brown adipose cells that can subsequently be implanted. Many current tissue engineered scaffolds that have small pores and limit cell infiltration, diffusion and viability. However, we have created a novel tissue engineered scaffold that possesses large homogenous pores that readily enables cell colonization and viability and provides a relevant and sustainable environment for brown adipose cells.

The surgical implantation of brown adipose tissue has been shown to reverse diabetes and decrease fat mass. However, tissue implantation has major immune rejection problems. Our tissue-engineered strategy can allow autografted or allografted cells to be implanted with low immune rejection risk.

Methods

Human brown adipose derived stem cells were seeded onto scaffolds derived from adult human adipose tissue and subsequently differentiated into brown adipocytes. Scaffolds were implanted in the dorsal fat pad of adult NOD-SCID mice over the course of 6 weeks. White fat-seeded and empty scaffolds were concurrently implanted in separate mice from brown fat-seeded scaffolds to serve as controls. Brown adipocytes within scaffolds remained viable over the course of the 6 weeks and were shown to decrease size and weight of dorsal fat pads within implanted mice. Interestingly, only scaffolds with cells (either brown or white) remained, while empty scaffold controls were completely or mostly degraded by the end of the 6-week study.

Results

A resident stem cell population within depots of brown adipose tissue from adult humans has been isol differentiation into brown adipocytes demonstrate functional metabolic activity characteristic of brown ad vivo. White fat-seeded and empty scaffolds were concurrently implanted in separate mice from brown fat-s to serve as controls. Brown adipocytes within scaffolds remained viable over the course of 6 weeks and decrease size and weight of dorsal fat pads within mice. 6 weeks post-transplant brown fat se demonstrated the presence of multilocular adipocytes, characteristic of brown adipose depots.

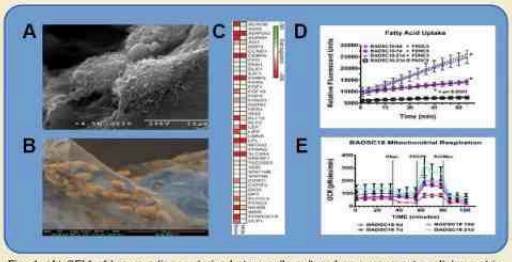

Fig. 1. (A) SEM of brown adipose derived stem cells cultured on porous extracellular matrix scaffolds. (B) SEM of directionally differentiated brown adipocytes on scaffolds.(C) Transcriptional profile of brown adipose derived stem cells differentiated into brown and white adipocytes. (D) Fatty acid uptake of brown fat differentiated brown adipose derived stem cells at 7, 14 and 21 days post differentiation. (E) Functional mitochondrial respiration assay of brown adipose derived stem cells differentiated into brown adipocytes at 7, 14 and 21 days post differentiation.

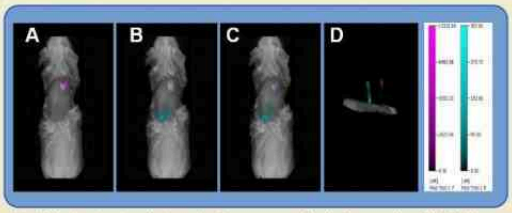

Fig. 2. In vivo imaging of mice using fluorescence molecular tomography with (A-D) brown adipose implanted scaffolds after 3 weeks. Purple indicates scaffold only and Turquoise indicates cells only. Three scaffolds were implanted subcutaneously on the dorsum of the mice. The two lower left scaffolds in each mouse indicate not labeled scaffolds with labeled cells. The upper right scaffolds shows a labeled scaffold with labeled cells.

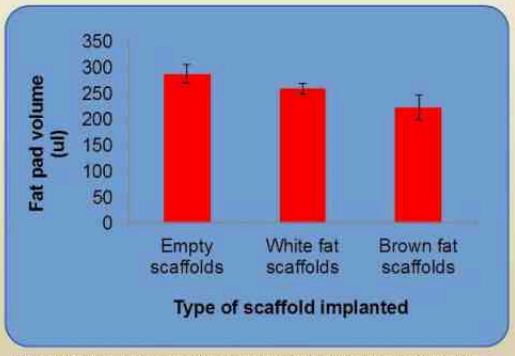

Fig. 3. After 6 weeks, mice were sacrificed and the fat pad volumes were measured. Mice with brown fat scaffold implants had significantly reduced fat pad volumes (n=9 mice per group). * P< 0.5.

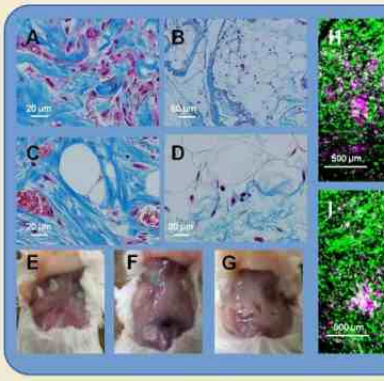

Fig. 4. Histology images of explanted scaffolds after 6 weeks showing, (A) fat scaffold, and (B) white fat cells within white fat scaffolds. Thes vascularization and (D) integration of host fat with scaffold. Photographi implantation, mice dorsal fur and skin were removed to indicate implanted fat, (F) white fat, and (G) empty scaffolds. Importantly, all mice receiv resorbed the scaffolds by 6 weeks. Representative images from mice seeded scaffolds, white fat-seeded scaffolds, empty scaffolds are sho Confocal images showing viability of (H) brown fat and (I) white fat cells in 6 weeks. Viable cells were labeled with Calcein AM (Green). Pre-labeled purple. White shoes co-localization of viable and pre-labeled cells, indic many host cells migrated into the scaffolds and 2) implanted cells remain for 6 weeks. Representative images were taken from 3 different brown or w

Conclusion

These results uniquely demonstrate a resi population within depots of brown adipos adult humans. These cells were capa seeded onto human derived scaffolds human 3D tissue engineered tissue mimics naturally occurring brown adipose scaffolds were shown to provide an effe modality that retained cells and enabled t vivo. The delivery of brown adipocytes engineered scaffolds may provide a via reducing white fat burden and ultimately harmful effects of obesity and metabolic di

References

[1] Barlow SE, Dietz WH. Obesity evaluation and treatment: Expert Committee Maternal and Child Health Bureau, Health Resources and Services Administrati Health and Human Services. Pediatrics 1998;102:E29.
[2] Halpern B, Halpern A. Why are anti-obesity drugs stigmatized? Expert opinion on d
[3] Lidell ME, Enerback S. Brown adipose tissue--a new role in humans? Nature 2010;6:319-25.
[4] Cannon B, Nedergaard J. Brown adipose tissue: function and physiological s reviews 2004;84:277-359.
[5] Castillo-Quan JI. From white to brown fat through the PGC-1alpha-dependent m for diabetes and obesity. Disease models & mechanisms 2012;5:293-5.
[6] Nicholls DG, Locke RM. Thermogenic mechanisms in brown fat. Physiological revi