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# Adipose Tissue Inflammation in Diabetic versus Non-diabetic Pregnancies


Tiffany A. Moore Simas  
*University of Massachusetts Medical School*

Raziel Rojas-Rodriguez  
*University of Massachusetts Medical School*

Jacqueline Tessa Draper  
*University of Massachusetts Medical School*

*See next page for additional authors*

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**Presenter Information**

Tiffany A. Moore Simas, Raziell Rojas-Rodriguez, Jacqueline Tessa Draper, Aylin Sert, Maxwell Kruse, Crina Boeras, Brittany Rosenthal, Olga Gaelikman, So-Yun Min, Katherine G. Leung, and Silvia Corvera

**Comments**

Abstract of poster presented at the 2014 UMass Center for Clinical and Translational Science Research Retreat, held on May 20, 2014 at the University of Massachusetts Medical School, Worcester, Mass.

Jacqueline Draper participated in this study as a medical student in the Senior Scholars research program at the University of Massachusetts Medical School.

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## Adipose Tissue Inflammation in Diabetic versus Non-diabetic Pregnancies

Tiffany A. Moore Simas, MD, MPH, MEd  
UMass Medical School/UMMHC  
508-334-6678  
Tiffanya.mooresimas@umassmemorial.org

Tiffany A. Moore Simas, MD, MPH, MEd<sup>1a</sup>; Raziell Rojas-Rodriguez, BS<sup>1b</sup>; Jacqueline Tessa Draper, BS<sup>1</sup>; Aylin Sert, MEd<sup>1</sup>; Maxwell Kruse<sup>1b</sup>; Crina Boeras, MD<sup>1a</sup>; Brittany Rosenthal<sup>1b</sup>; Olga Gealekman, PhD<sup>1b</sup>; So Yun Min, MSc<sup>1b</sup>; Kathy Leung, MPH<sup>1a</sup>; Silvia Corvera, MD<sup>1b</sup>

<sup>1</sup>University of Massachusetts Medical School; <sup>a</sup> Obstetrics & Gynecology, <sup>b</sup> Program in Molecular Medicine

**Objective:** Beyond weight associated with fetus, placenta, amniotic fluid and increased blood volume, adipose tissue (AT) expansion is an accepted and expected component of pregnancy weight gain. Normal pregnancy is associated with relative insulin resistance (IR). In non-pregnant humans, AT expansion has been associated with IR and AT inflammation. However, it is not known whether AT expansion and IR in pregnancy are also associated with AT inflammation. This pilot study examined relationships between AT expansion and inflammation in control versus diabetic pregnancies.

**Methods:** Eligible subjects undergoing scheduled Cesarean delivery for obstetric indications were prospectively enrolled. Subjects provided demographic and anthropometric data, and biologic specimens. Immunofluorescence microscopy was performed on subcutaneous (SQ) and omental (OM) AT samples to evaluate macrophage infiltration. Included gravidas had paired AT samples and either negative glucola screening (controls) or gestational or pre-gestational Type 2 diabetes mellitus (T2DM).

**Results:** 13 subjects with SQ and OM AT samples were evaluated (10-controls, 3-diabetics (2-T2DM and 1-GDM)). Mean BMI and gestational weight gain of controls was 27.8 kg/m<sup>2</sup> (range 19.5-42) and 27.6 pounds (range 15-36) and of diabetics was 30.6 kg/m<sup>2</sup> (range 30-33) and 19 pounds (range -3-30), respectively. Macrophage infiltration was seen in OM AT from 2/ 3 diabetics and 0/ 10 controls (see figure).

**Conclusions:** These results indicate that AT expansion in non-diabetic pregnancies is not accompanied by macrophage infiltration. Thus, the IR of normal pregnancy is unlikely to be related to AT inflammation, and AT expansion per se does not lead to AT inflammation. However, as has been reported for T2DM in non-pregnant humans, the presence (T2DM) or development (GDM) of diabetes in pregnancy is associated with macrophage infiltration of AT. Despite the small sample size, the observed large differences in macrophage infiltration between controls and diabetics suggest that these findings will persist in a larger cohort.

