List of presentations, publications, abstracts


Aaron J. Fields, Britta Berg-Johansen, Brandon La, Ellen Liebenberg, Sabra Djomehri, Dezba Coughlin, James Graham, Lionel N. Metz, Peter J. Havel, Jeffrey C. Lotz, Effect of Type-2 Diabetes Mellitus on Endplate Microarchitecture, Marrow Cellularity and Intervertebral Disc Creep in Rats, 60th Annual Meeting of the Orthopaedic Research Society, 2014


Has the funding led to receipt of other funds?

We are currently using these data as part of a new NIH R01 submission on the topic of diabetes, obesity, and spinal disc degeneration.

Abstract

Using a rat model of polygenic obese type 2 diabetes, we demonstrated that diabetes compromises disc composition, matrix homeostasis and biomechanical behavior. Caudal motion segments were harvested from 6-month-old lean Sprague-Dawley rats, obese Sprague-Dawley rats, and diabetic obese UCD-T2DM rats (diabetic for 69 ± 7 days). Findings indicated that diabetes but not obesity reduced disc glycosaminoglycan and water contents, and these degenerative changes correlated with increased endplate thickness and decreased endplate porosity, and with higher levels of the AGE pentosidine. Consistent with their diminished composition and higher AGE levels, discs from diabetic rats were also stiffer and exhibited less creep when compressed. At the matrix level, elevated expression of hypoxia-inducible genes and catabolic markers in the discs from diabetic rats indicated a shift in matrix homeostasis that was consistent with increased oxidative stress and greater interactions between AGEs and one of their receptors (RAGE). Taken together, these findings indicate that endplate sclerosis, increased oxidative stress and AGE/RAGE-mediated interactions could be important factors for explaining the greater incidence of disc pathology in type 2 diabetes.