conferenceseries.com

WORLD SUMMIT ON DIABETES

June 16-17, 2021 | Webinar

Hyperglycemia in Type 2 Diabetes Mellitus is Associated with Imbalanced Bone Remodeling by Osteocytes

Rachana Vaidya

University of Massachusetts Dartmouth,MA 02747, USA

Patients with type 2 diabetes mellitus (T2D) have a 3-fold increase in skeletal fragility compared to healthy adults despite having normal or high bone density. One possible contributing factor is the response of bone cells to the presence of high sugar in the body, known as hyperglycemia. Specifically, hyperglycemia may lead to alterations in the behavior of osteocytes, which are bone cells embedded in the bone matrix that play a key role in regulating the bone remodelling process. Osteocytes release two key proteins sclerostin (encoded by SOST gene) and receptor activator of nuclear factor-κB (RANKL) that play a role in the regulation of bone formation and resorption, respectively. To test this hypothesis, Ocy454 osteocytes were cultured in hyperglycemic conditions to investigate their response to a simulated diabetic environment. We hypothesized that exposure of osteocytes to high glucose will result in changes in SOST and RANKL expression to indicate an imbalance in remodeling. Our results show significant increase in sclerostin gene (10-fold) and protein expression (4-fold) in 22mM glucose ($p \leq 0.05$) relative to control (5.56mM glucose). RANKL gene and protein expression remained unaffected in 22mM glucose compared to control. Sclerostin is an inhibitor of bone formation and RANKL stimulates bone resorption. Thus, our results indicate hyperglycemia triggers osteocytes to inhibit bone formation but does not affect bone resorption. Together these results suggest that hyperglycemia as present in T2D causes imbalanced bone remodeling, potentially compromising bone's overall strength and increasing fracture risk in diabetic patients.

Biography

Rachana Vaidya is currently a PhD Candidate in Bioengineering and Biotechnology program at University of Massachusetts Dartmouth, MA, USA and has completed her Master's in Biotechnology from St Xavier's College, Mumbai. Her investigation focuses on how hyperglycemia and advance glycation end products(AGEs), such as pentosidine and CML alter bone cell behavior, to understand deterioration of bone quality and increased bone fragility in Type 2 diabetic patients. She will be graduating in summer 2021 and wants to continue working in diabetes/aging research. She has published 2 papers and 2 book chapters since 2018.

rvaidya@umassd.edu