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Adult stem cell derived enteroids as a model of intestinal pathologies in obesity

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Statement of the Problem: The prevalence of obesity in the U.S. population has increased steadily since the 1960s—from 3.4 % of adults in 1962 to 39.8 % in 2016. In 2016, ~100.3 million U.S. residents had obesity and another ~80.2 million were overweight. Chronic diseases driven by obesity accounted for $480.7 billion in direct health care costs in the U.S., with an additional $1.24 trillion in indirect costs due to lost economic productivity. However, the pathogenesis of obesity and the intestinal contribution to metabolic syndrome remains poorly understood. The purpose of this study is to determine the possible differences in dietary carbohydrate absorption by intestinal epithelium from lean vs. obese subjects. Methodology & Theoretical Orientation: Stem cell derived enteroid cultures that represent jejunal epithelial were generated from biopsies obtained from consented lean (BMI≤25) and obese (BMI≥30) subjects. Dietary glucose absorption and gluconeogenesis in enteroids were measured. Expression of carbohydrate transporters and gluconeogenic enzymes was assessed and pharmacological approach was used to dissect the specific contribution of each transporter or enzyme to carbohydrate absorption and metabolism, respectively. Findings: Four phenotypes representing the relationship between patients' BMI and intestinal dietary sugar absorption were found, suggesting that human enteroids retain the obese patient phenotype heterogeneity. Intestinal glucose absorption and gluconeogenesis were significantly elevated in enteroids from a cohort of obese patients. Elevated glucose absorption was associated with increased expression of SGLT1 and GLUT2, whereas elevated gluconeogenesis was associated with increased expression of GLUT5, PEPCK1 and G6Pase. Conclusion & Significance: Obesity phenotypes preserved in human enteroids provide a mechanistic link to aberrant dietary carbohydrate absorption and metabolism. Enteroids can be used as a preclinical platform to understand the pathophysiology of obesity, study the heterogeneity of obesity mechanisms and identify novel therapeutics.

Biography

Olga Kovbasnjuk is working as an Associate Professor in University of Maryland School of Medicine in USA