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CHANGES IN ECM DISTRIBUTION DURING SMALL INTESTINAL ADAPTATION FOLLOWING MASSIVE SMALL BOWEL RESECTION

William Hyunhak Goo

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Short gut syndrome (SGS) is defined as insufficient intestinal length to meet the nutritional and metabolic demands of a patient. Causes range from necrotizing enterocolitis and congenital malformations (children) to inflammatory diseases and cancer (adults). In these diseases, small bowel resection is performed to cut out the damaged and unrecoverable portion of the small intestine (SI), resulting in SGS. In an adaptive physiological response, the body increases the length of intestinal villi and the depth of crypts within remaining short intestine, thereby increasing absorptive mucosal surface area. This regenerative physiological response is called “intestinal adaptation,” but the genetic and molecular mechanisms that drive this adaptive response are poorly understood. Extracellular matrix (ECM) is known to play key roles in many developmental and regenerative processes. We hypothesize that the ECM plays a critical role in the intestinal adaptation response to SGS. In support of this model, our lab found that following SI cytotoxic damage and RNA-seq analysis, ECM related genes were the most highly upregulated class of genes among all gene ontology categories. My research focuses on analyzing changes in ECM during SI adaptation following massive small bowel resection (SBR). To address the role of the ECM in the regenerative response to SGS, we will track the localization of key ECM proteins before SBR and four days after SBR to compare the composition and distribution of the ECM during the adaptive response. Our approach will provide a clearer view of the role of the ECM in the intestinal adaptation following the SBR. A better understanding of SI ECM regenerative processes may inform new therapies for neonatal, pediatric, and adult patients suffering from SGS.