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Molecular Mechanism of Rectal Insulin Signalling in Inflammatory Bowel Disease

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Deadline for manuscript submissions:
closed (20 April 2024)

Message from the Guest Editor

Intestinal epithelial cells harbor insulin receptors on their basolateral membranes. The physiological role of signaling through the insulin receptor in the intestinal epithelium remains elusive (if it exists at all). Mouse inactivation experiments have demonstrated that these insulin receptors do not play a role in intestinal development. However, under specific conditions, such as during high-fat diet-induced obesity, a phenotype can be elicited. Intriguingly, this phenotype includes changes in the number of certain enteroendocrine cells. In Ulcerative Colitis, insulin receptor mRNA can be upregulated in mucosal biopsies. Furthermore, immunohistochemistry revealed a correlation between this upregulation and increased amounts of insulin receptor immunoreactivity on the basolateral membranes of colonocytes. Subsequent experiments showed that rectally instilled insulin in mice with chemically induced colitis attenuated the inflammation. Collectively, these findings allow pharmacological targeting of epithelial insulin receptors using a local administration approach to treat inflammatory bowel diseases.

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Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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