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MORPHO-FUNCTIONAL CHARACTERISTICS OF ENDOCRINE CELL DIFFERON OF THE INTESTINAL EPITHELIUM IN INFLAMMATORY BOWEL DISEASES

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Annotation: Inflammatory bowel diseases (IBD), which include chronic disorders such as Ulcerative Colitis (UC) and Crohn's Disease (CD), are characterised by persistent inflammation of the intestinal mucosa and disruption of epithelial homeostasis. One of the lesser-explored aspects of IBD pathophysiology involves the endocrine (enteroendocrine) cell complement of the intestinal epithelium. These endocrine cells, dispersed among other epithelial lineages, play critical roles in gut motility, secretion, nutrient absorption, epithelial repair, and immunomodulation. In IBD, morpho-functional changes occur within the intestinal endocrine cell population: alterations in cell density, distribution, hormonal secretion profiles, and ultrastructural features have been documented. These changes appear to correlate with immune cell infiltration, epithelial barrier disruption, and mucosal healing responses. A deeper understanding of the morphologic (cellular localisation, ultrastructure, granule content) and functional (hormone output, receptor interactions, autocrine/paracrine signalling) characteristics of the endocrine cell differon in IBD may open new avenues for diagnostics and therapeutic modulation. This paper presents a comprehensive review of the current knowledge on endocrine cell alterations in the intestinal epithelium in IBD, integrating histological observations, immunohistochemical data, experimental animal model findings, and potential mechanistic links to mucosal immunity and barrier function. The goal is to emphasise the interplay between endocrine-epithelial-immune systems in the inflamed gut and to propose directions for future research in this niche domain.

Keywords: inflammatory bowel disease, enteroendocrine cells, intestinal epithelium, endocrine cell differon, morpho-functional characteristics, mucosal immunity, epithelial barrier, hormone secretion, gut neuroendocrine system

Intestinal epithelium is a dynamic tissue composed of several specialised cell types derived from stem/progenitor cells located in crypts. Among these lineages, enteroendocrine cells represent the endocrine arm of the epithelium, responding to luminal, neural and immune cues by releasing peptide hormones and bioactive amines that regulate motility, secretion, absorption, vascular flow and immune responses. In the setting of IBD, the intestinal environment is characterised by epithelial injury, increased cell turnover, tight junction disruption, immune cell infiltration, cytokine up-regulation, and altered microbiota. Under such pathological stress, the endocrine cell population undergoes both quantitative and qualitative modifications. Histologically, studies have found altered densities of chromogranin A-positive endocrine cells in IBD. For example, in colitis



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models, increased densities of chromogranin A, serotonin, peptide YY and oxyntomodulin-immunoreactive cells were observed, while pancreatic peptide and somatostatin-positive cells showed decreased densities. These findings suggest a shift in endocrine cell composition and a possible compensatory endocrine response to inflammation. There is also evidence that in ileal segments of Crohn's disease patients, endocrine cell numbers are increased when assessed via chromogranin immunostaining. On the morphological level, endocrine cell ultrastructure may show altered granule size, shape, or distribution, reflecting changed secretory behaviour in the inflamed mucosa.

Functionally, endocrine cells in the gut contribute to mucosal defence and repair through hormone secretion that influences epithelial renewal, immune cell behaviour, and barrier properties. Gut hormones such as GLP-1, PYY, GIP, CCK and others are secreted by discrete enteroendocrine subpopulations (for example L-cells, K-cells, I-cells) and have been shown to modulate epithelial integrity and mucosal immunity, thereby limiting the development or severity of IBD. The altered endocrine cell differon in IBD may thus reflect an adaptive or maladaptive response: elevated secretion of certain peptides may aim to slow motility or reduce secretion to limit further epithelial damage, while loss of other hormones may impair mucosal repair or immunoregulation.

The morpho-functional alterations of endocrine cells in IBD must be considered in the context of epithelial-immune cross-talk. In inflamed mucosa, increased densities of leukocytes (T and B lymphocytes, macrophages/monocytes, mast cells) correlate with changes in endocrine cell phenotypes. For instance, the increased densities of PYY and oxyntomodulin cells correlated with immune cell infiltration in a DSS-colitis rat model. The mechanistic underpinning may involve cytokine-mediated modulation of endocrine progenitor differentiation, or endocrine cell responses to inflammatory mediators as part of local immunological feedback loops.

From a morphologic viewpoint, endocrine cells in IBD may also display altered localisation within the crypt-villus axis, changes in apical vs basal processes, modifications in the expression of granin proteins, and changes in the ratio between “open” (luminal sensing) and “closed” (paracrine) endocrine cell types. These morphological adaptations may have functional consequences: for example, increased apical processes may heighten luminal sensing and hormone release in response to inflammation or microbial products; conversely, basal process alterations may affect communication with immune or vascular compartments.

Functionally, altered hormone output from endocrine cells may influence motility (increased or decreased transit), secretion (mucus, fluids, ions), absorption, appetite and systemic metabolic responses. In IBD, where motility patterns, secretion profiles and nutritional status are frequently disrupted, endocrine cell changes may contribute to symptomatology (diarrhea, malabsorption, weight loss) and disease progression (flares, remission, healing). Moreover, therapeutic approaches targeting gut hormones—such as GLP-1 agonists—are emerging not only for metabolic disease but also as potential anti-inflammatory strategies in IBD, linking endocrine cell function to clinical translation.

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Importantly, the endocrine cell compartment may serve as an indicator of mucosal health and healing: changes in endocrine cell densities or hormone levels could act as biomarkers for epithelial recovery or persistent dysregulation. For instance, a relative increase in endocrine cells producing anti-inflammatory peptides might reflect a reparative phase, whereas persistent loss of specific endocrine populations could signal incomplete mucosal healing or risk of relapse.

In conclusion, the endocrine cell compartment of the intestinal epithelium represents a morpho-functional interface between the luminal environment, epithelial barrier and immune system. In IBD, this compartment undergoes significant alterations, both morphological and functional, which likely contribute to disease dynamics. Further research is required to map out the full spectrum of endocrine cell changes in human IBD, to determine causality vs consequence, to clarify receptor/hormone interplay in the inflamed gut, and to explore modulation of this system for therapeutic benefit. From a practical standpoint, assessment of endocrine cell densities, hormone secretion profiles and ultrastructure in biopsy specimens may become part of advanced mucosal phenotyping in IBD. Ultimately, restoring endocrine cell homeostasis may become an adjunct strategy in promoting mucosal healing and sustained remission in IBD.

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