# FOREWORD

## Enterochromaffin-Like Cell (ECL) Meeting—February 1-2, 1997

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The manuscripts collected in this volume represent the collated presentations made by national and internationally recognized authorities in the field of gastric biology and particularly neuroendocrine cell function of the stomach. The meeting was devoted to the enterochromaffin-like (ECL) cell and is the first meeting held that has been entirely devoted to this subject. Prior to the recognition of the ECL cell as the pivotal regulator of gastric acid secretion, there was considerable confusion in regard to the mechanism by which the parietal cell was stimulated to secrete acid. The identification of the ECL cell and the recognition of its primary secretory product, histamine, dispelled the previous notion that antral gastrin directly regulated parietal acid secretion. Indeed, the subsequent demonstration of a gastrin CCKB receptor on the ECL cell and the elucidation of the role of gastrin as an agonist of ECL cell function effected a major alteration in the conceptual understanding of acid secretion and the trophic regulation of the gastric mucosa. The more recent unraveling of the effects of hypergastrinemia on the regulation of ECL proliferation has shed considerable light on the genesis of gastric carcinoid tumor formation and provided fundamental biological insight into the regulation of endocrine cell proliferation. Similarly, the further delineation of the cellular mechanisms by which histamine secretion and acid secretion are intertwined has provided important insights into the biology of the gastric mucosa and the pathobiology of low acid and hypergastrinemia states. Clearly, the ECL cell is a pivotal structure both in the physiology and pathophysiology of gastric function about which much remains to be learned. The current status of knowledge in this area has been collated in this volume and should allow for an overview of current concepts in ECL physiology and biology.

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