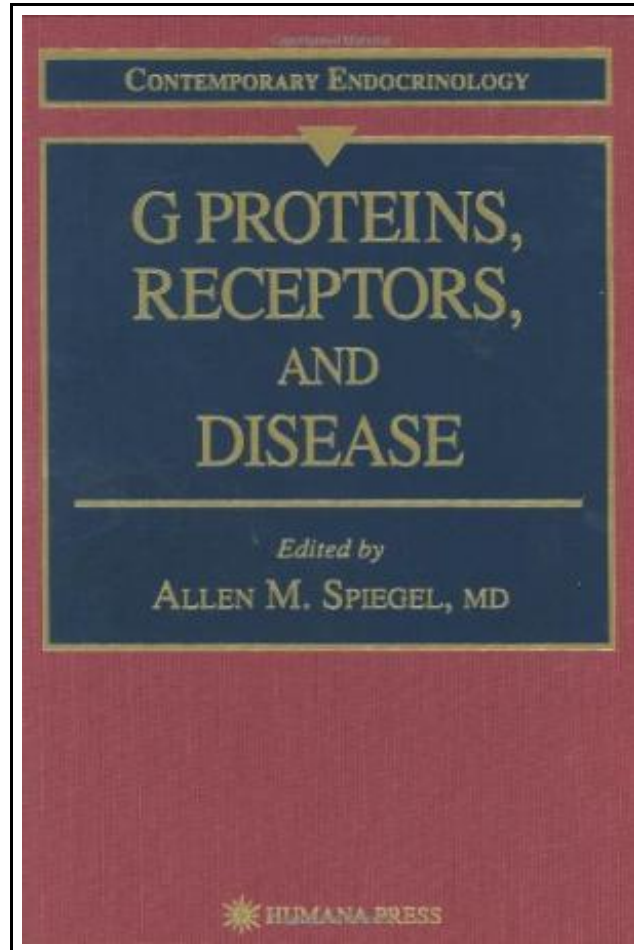


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Humana Press Inc., United States, 1998. Hardback. Book Condition: New. 1998 ed.. 258 x 182 mm. Language: English . Brand New Book ***** Print on Demand *****. Theoretically, one should obtain essentially the same clinical picture from failure of an end-organ to respond to a hormone as from a decreased production or absence of said hormone. With these words, Fuller Albright began his now classic paper describing a novel disease, pseudo hypoparathyroidism (PHP), and a novel concept, hormone resistance as a cause of disease. Soon, other hormone resistance disorders such as nephrogenic diabetes insipidus (NDI) were recognized, and the concept was extended to resistance to other substances such as calcium ions in familial hypocalciuric hypercalcemia (FHH). Later, diseases characterized by excess rather than deficient hormone action such as McCune-Albright syndrome (MAS) and familial male precocious puberty (FMPP) were recognized to be caused by autonomous endocrine hyperfunction. Although many investigators provided careful and detailed descriptions of the clinical features of these and other related endocrine disorders, an understanding of pathogenesis proved elusive for many years. In just the past few years, we have gone from clinical description to a molecular understanding of these interesting disorders. This remarkable progress reflects a synthesis of three distinct, but now overlapping, areas of biomedical research: the aforementioned recognition and careful clinical description of specific diseases, the elucidation of the basic mechanisms of signal transduction, and the application of the powerful tools of molecular biology and genetics. Fundamental studies on the mechanisms of hormone action by Rodbell and colleagues at NIH culminated in the discovery of a major signal transduction pathway involving heterotrimeric G proteins.



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