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Nota di bibliografia	Includes bibliographical references at the end of each chapters and index.
Nota di contenuto	A brief introduction to extrasynaptic GABAA receptors and ‘tonic’ GABAA receptor mediated inhibition in physiology and disease -- Extrasynaptic GABAA receptors: subunit composition, distribution, and regulation -- Biophysical properties of recombinant 2- AND - subunit containing GABAA receptors -- The pharmacology of extrasynaptic GABAA receptors -- Neurosteroids and extrasynaptic GABAA receptors -- Sources of GABA that activate extrasynaptic GABAA receptors -- Modulation of Extrasynaptic GABAA Receptors by G-protein-coupled Receptors -- Extrasynaptic GABAA receptors and tonic inhibition in spinal cord -- The role of peri-synaptic GABA receptors after stroke -- The role of extrasynaptic GABAA receptors in focal epilepsy -- Gain-of-Function of Thalamic Extrasynaptic GABA-A Receptors in Typical Absence Seizures -- GABAergic control of the hypothalamic-pituitary-adrenal (HPA) axis: role of extrasynaptic GABAA receptors -- Tonic GABAA receptor mediated inhibition in Fragile-X Syndrome: A cause of dysfunction or a pathway for a cure?
Sommario/riassunto	GABA is the principal inhibitory neurotransmitter in the CNS and acts via GABAA and GABAB receptors. Recently, a novel form of GABAA

receptor-mediated inhibition, termed “tonic” inhibition, has been described. Whereas synaptic GABAA receptors underlie classical “phasic” GABAA receptor-mediated inhibition (inhibitory postsynaptic currents), tonic GABAA receptor-mediated inhibition results from the activation of extrasynaptic receptors by low concentrations of ambient GABA. Extrasynaptic GABAA receptors are composed of receptor subunits that convey biophysical properties ideally suited to the generation of persistent inhibition and are pharmacologically and functionally distinct from their synaptic counterparts. This book highlights ongoing work examining the properties of recombinant and native extrasynaptic GABAA receptors and their preferential targeting by endogenous and clinically relevant agents. In addition, it emphasizes the important role of extrasynaptic GABAA receptors in GABAergic inhibition throughout the CNS and identifies them as a major player in both physiological and pathophysiological processes.