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Titolo	Signalling pathways in acute oxygen sensing [[electronic resource]]
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Descrizione fisica	1 online resource (302 p.)
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Nota di bibliografia	Includes bibliographical references and indexes.
Nota di contenuto	Cover; Contents; Chair's introduction; Regulation of gene expression by HIF-1; DISCUSSION; Regulation of HIF: prolyl hydroxylases; DISCUSSION; General discussion I; Regulation of HIF: asparaginyl hydroxylation; DISCUSSION; Oxygen-sensing by ion channels and mitochondrial function in carotid body glomus cells; DISCUSSION; The role of TASK-like K+ channels in oxygen sensing in the carotid body; DISCUSSION; Reactive oxygen species facilitate oxygen sensing; DISCUSSION; Reactive oxygen species facilitate oxygen sensing; DISCUSSION; Oxygen sensing in neuroepithelial and adrenal chromaffin cells; DISCUSSION Hypoxic regulation of Ca2+ signalling in astrocytes and endothelial cellsDISCUSSION; General discussion II; Functional proteomics of BK potassium channels: defining the acute oxygen sensor; DISCUSSION; A central role for oxygen-sensitive K+ channels and mitochondria in the specialized oxygen-sensing system; DISCUSSION; Role for mitochondrial reactive oxygen species in hypoxic pulmonary vasoconstriction; DISCUSSION; Hypoxic pulmonary vasoconstriction- triggered by an increase in reactive oxygen species?; DISCUSSION; General discussion III The role of twin pore domain and other K+ channels in

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	hypoxicpulmonary vasoconstrictionDISCUSSION; AMP-activated protein kinase couples mitochondrial inhibition by hypoxia to cell-specific Ca2+ signalling mechanisms in oxygen-sensing cells; DISCUSSION; Role of capacitative Ca2+ entry but not Na+/Ca2+ exchange in hypoxic pulmonary vasoconstriction in rat intrapulmonary arteries; DISCUSSION; Final general discussion; Index of contributors; Subject index
Sommario/riassunto	Oxygen sensing is a key physiological function of many tissues, but the identity of the sensor, the signalling pathways linking the sensor to the effector, and the endpoint effector mechanisms are all subjects of controversy. This book evaluates the various mediators that have been proposed, including the mitochondria, NAD(P)H oxidases, cytochrome p450 enzymes, and direct effects on enzymes and ion channels. There has been a resurgence of interest in the role of mitochondria, based partly on the ability of mitochondrial inhibitors to mimic hypoxia, but there is little consensus concerning mech