1. Record Nr. UNIORUON00025181

Titolo	MANAGING health systems in developing areas : Experiences from Afghanistan / edited by Ronald W. O'Connor
Pubbl/distr/stampa	Toronto, : Lexington Books, 1980 xvii, 314 p. ; 23 cm
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Lingua di pubblicazione	Inglese
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Autore	Kim Sang Geon
Titolo	AMPK-S6K1 signaling pathway as a target for treating hepatic insulin resistance / / Sang Geon Kim, Il Je Cho and Hee Yeon Kay
Pubbl/distr/stampa	New York, : Nova Science Publishers, c2010
ISBN	1-61761-457-2
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Descrizione fisica	1 online resource (69 p.)
Collana	Hepatology research and clinical developments series
Altri autori (Persone)	CholI Je KayHee Yeon
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Note generali	Description based upon print version of record.
Nota di bibliografia	Includes bibliographical references (p. [33]-49) and index.
Nota di contenuto	Introduction: Insulin signaling in hepatocytes -- Clinical and preclinical drugs for the treatment of hepatic insulin resistance.
Sommario/riassunto	Hepatic insulin resistance and altered insulin metabolism, as characterized by the desensitization of hepatic parenchymal cells to insulin, play a role in the pathogenesis of liver disease, particularly

resulting in steatosis and steatohepatitis. By the same token, type II diabetic patients are at higher risk for developing liver diseases, including steatosis, hepatitis, cirrhosis, and hepatocellular carcinoma. On the other hand, established liver disease from any cause leads to glucose intolerance and peripheral insulin resistance systemically. The link between insulin resistance and liver pathology reviewed in this book suggests that insulin resistance is closely related with a variety of liver diseases. Recent evidence indicates that the AMP activating protein kinase (AMPK) in conjunction with p70 ribosomal S6 kinase 1 (S6K1) serves as a key signaling pathway regulating insulin-dependent physiological functions; thus, this pathway serves as a target for the therapy of diseases associated with insulin resistance. In this chapter, the regulatory role of the AMPK-S6K1 pathway is discussed in terms of enhancing insulin receptor signaling with insulin receptor substrate-1/2 and phosphatidylinositol phosphate kinase activity, which may contribute to preventing and/or treating insulin resistance in the liver.