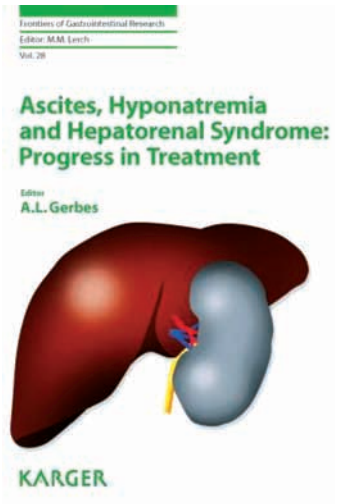
BOOK REVIEWS

Frontiers of Gastrointestinal Research (Editor: M.M. Lerch). Vol. 28: Ascites, Hyponatremia and Hepatorenal Syndrome: Progress in Treatment. A.L. GERBES (Ed). S. Karger Medical and Scientific Publishers. VIII + 212 p., 23 fig., 31 tab., hard cover, 2011. ISBN 978-3-8055-9591-9. e-ISBN 978-3-8055-9592-6. Price €135.-



Review :

Ascites is the most frequent complication of liver cirrhosis. It is estimated that nearly 60% of patients with compensated cirrhosis will develop ascites within 10 years. Hepatorenal syndrome (HRS) is the most lethal complication of cirrhosis. The volume 28 of the series 'Frontiers of Gastrointestinal Research' deals with the management of ascites, and the related complications of hyponatremia and the hepatorenal syndrome, all mainly caused by the systemic (mainly splanchnic) vasodilation in advanced cirrhosis with portal hypertension. Topics elaborated are diuretic treatment, paracentesis and transjugular intrahepatic portosystemic shunt for treatment of ascites, the use of plasma-expanders in management of ascites and renal failure in cirrhosis, the use of vaptans in the treatment of ascites and/or hyponatremia in cirrhosis. Several chapters deal with the use of vasopressin analogs in the HRS.

Each chapter is written by experts in the field, from Europe of the US, provides up-to-date references and is concluded with a bullet point summary. The book provides up-to-date information on the management of these important complications of cirrhosis for gastroenterologists dealing with these patients.

Minor drawbacks, however, are the almost inevitable overlap between the chapters dealing with the HRS. Furthermore, a list of abbreviations could have been useful. Finally, not all figure legends are self-explanatory.

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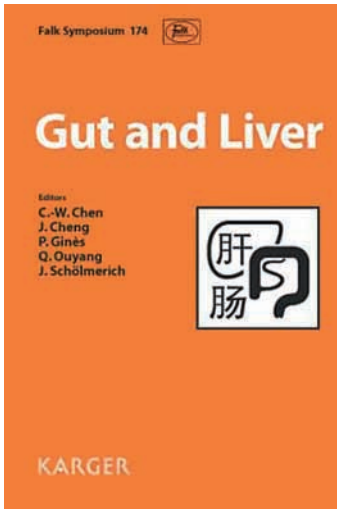
Gut and Liver, Falk Symposium 174. Basel, Karger, 2011. Chen C.-W., Cheng J., Ginès P., Ouyang Q., Schölmerich J. (editors), VIII + 142 p, 23 fig, 24 tab, soft cover 2011. ISBN 978-3-8055-9672-5.



The issue «Gut and Liver» reflects the lectures of the Falk symposium 174 held in Beijing (China) in August 2010. The title of this book suggests chapters on the interactions between those two organs. Indeed, liver diseases such as cirrhosis may induce digestive tract complications. In parallel, the gut has a role in some liver-related conditions such as iron excess, encephalopathy, toxicity of treatments of gut diseases. Furthermore, the liver, due to its anatomical position, is closely linked to the gut, being exposed to dietary substances or bacterial products of the digestive tract. In this way, the gut-liver axis and the connection between the gut microbiota and liver inflammation are increasingly recognized in the pathogenesis of e.g. alcoholic and non-alcoholic fatty liver diseases.

In fact, this book contains twenty-two topics on diseases of the gut (ulcerative colitis and Crohn disease only) and on liver diseases (hepatitis B and C, liver failure and liver cancer), respectively. The title of the book is somehow misleading, as the links between the gut and the liver are not evident or are not discussed in the different book chapters. However, each chapter is very well written by recognized experts in the field from Asia, Europe and America starting with an abstract, and containing up-to-date references. Despite the fact that some articles could benefit from some tables or some graphs for clarity, this book is recommendable to readers seeking for good review papers on inflammatory bowel diseases (diagnosis, treatment) and on hepatic diseases.

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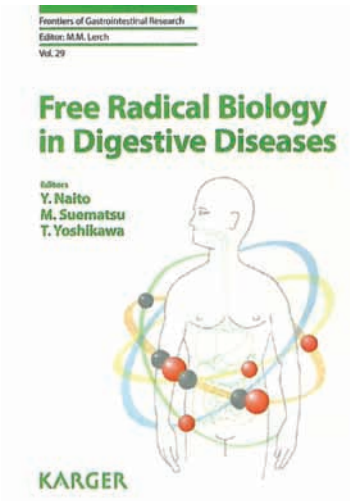
Free radical biology in digestive diseases. Editors : Y. Naito, M. Suematsu, T. Yoshikawa. Frontiers of Gastrointestinal Research, Vol. 29. VIII + 176 p., 36 fig., 3 in color, 9 tab., hard cover 2011. Karger Publishers, Basel. ISBN 978-3-8055-9609-1



The goal of this book on «Free radical biology in digestive diseases» is twofold : First, to review the main mechanisms of free radical production and their consequences for the biology of the cell or tissue, and second, to evaluate whether and how reactive oxygen species, free radicals and/or oxidative alteration of macromolecules interfere with the pathogenesis of specific diseases of the digestive tract such as H. Pylori infection, gastrointestinal tract lesions induced by nonsteroidal anti-inflammatory drugs, inflammatory bowel diseases, chronic liver diseases and pancreatitis.

The sources for free radicals are, on one hand, the mitochondria and, on the other hand the NADPH oxidase of inflammatory cells. Thus, ROS are elicited by cells experiencing a constraint on mitochondrial function and in which energy metabolism is perturbed (ROS are regarded as a harmful by-product of the aerobic metabolism) and in cells rendered susceptible by mtDNA mutations in relation to aging or to prior exposure to oxidative stress. Also, ROS are deliberately produced by NADPH oxidase, expressed in numerous cell types but in particular by inflammatory cells and epithelial cells of the gastrointestinal tract. In this case ROS production is bactericidal and participates to the host defense. The attention is drawn on neutrophil-dependent oxidative stress in gastrointestinal diseases such as infection, ischemia, inflammatory bowel diseases with in perspective, interruption of neutrophils recruitment as possible therapeutic targets. This however has to be balanced against anti-infectious consequences of such an action.

The toxic effectors of the oxidative stress are the modified macromolecules : lipoperoxides, oxidative DNA and protein damage leading to altered functions of biological membranes, activation of pro-inflammatory signals and mutagenicity. Some chapters are dedicated to the practical evaluation of oxidative markers whether in circulation or tissues. Those are deceitful as the reading does not provide a clear idea of what to look for, in which circumstances and how to interpret it.

The last part of the book is dedicated to the role of oxidative stress and injury in specific pathologies of the digestive tract. It appears clearly that free radicals and oxidative damage take a large part in diseased processes, exacerbated by alteration in the anti-oxidant defenses (whether by overwhelming stress, genetic determination or disease specific mechanisms) as well as auto-perpetuating and auto-amplifying positive feed-back loops. Indeed, oxidative stress damages the cells and activates pro-inflammatory pathways which recruit inflammatory cells, causing more damages, decreasing defenses and generating more ROS.

Anti-oxidant strategies may thus be considered in the therapeutic arsenal of digestive diseases using tools to quench ROS (free radial scavengers, inhibition of ROS generating enzymes), to stimulate anti-oxidant defense, to interrupt inflammatory recruitment, … Specific strategies are under evaluation for several diseases (inflammatory bowel diseases, chronic liver disease, pancreatitis, …) Despite the strong rational, in practice, anti-oxidant strategies are deceitful and probably better adapted for adjuvant therapy and prevention.

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