Preface

Fatty liver disease and nutrient intervention

Both alcoholic fatty liver disease (AFLD) and non-AFLD (NAFLD) are two of the major challenging public health problems in the world. Development of steatosis, steatohepatitis, fibrosis, and cirrhosis ultimately progresses towards hepatocellular carcinoma (HCC), and frequently leads into liver transplantation, which underlines the significance of these diseases with treatment and prevention. Given the high incidences of these diseases, in particular, the high mortality rate of HCC and its poor prognosis, the prevention of these diseases progression through dietary nutritional means, represent an important disease control strategy. Important aspects in the etiology, pathogenesis, complications, and treatment/prevention of AFLD and NAFLD from recent research are summarized in a series of review and original articles in this and the following issues of *HepatoBiliary Surgery and Nutrition* (*HBSN*). Leading experts in the field share their current prospective on this most important liver disease with the focus on clinical relevant issues.

The first issue will focus on molecular mechanisms in AFLD and NAFLD containing three review- and two original research articles. Sirtuin 1 (SIRT1), a NAD+-dependent protein deacetylase has been implicated as a key regulator of metabolism, inflammation, immune function, hypoxic responses, cell survival, and longevity. Growing evidence from human and rodent studies supports that both AFLD and NALFD are associated with impairment of hepatic SIRT1 signaling. The first review highlights significant role of SIRT1 signaling and its regulatory mechanisms, and indicates optimizing SIRT1 signaling by pharmacological and/or nutritional intervention as novel and effective remedies for AFLD. The significant increase in the prevalence of overweight and obesity not only in Europe and the US, but also in China resulting in diabetes mellitus (DM) and metabolic syndrome (MS) is also of major public concern. Although DM and MS are primarily associated with arteriosclerosis and coronary heart disease, another complication is NAFLD. In the second review, the associations between DM and NAFLD is discussed. The importance to consider not only the occurrence of NAFLD with DM, but also the effects of DM on fatty liver progression to NASH, as well as screening targeted to NASH patients has been highlighted. Recently, overconsumption of carbohydrates has been linked to the rise in the prevalence of obesity and associated with NAFLD. In the third review, the authors indicated that high intake of carbohydrates, especially high fructose corn syrup in the soft-drink and pre-packaged foods are more likely to directly contribute to the development of NAFLD than dietary fat intake. In addition, recent research on the generation of highly carcinogenic etheno-DNA adducts similar as in alcoholic liver disease is reported which may be an important mechanism for HCC in patients with NAFLD. Finally, an inhibition of chemical carcinogen induced alcohol promoted hepatic inflammation and precancerous lesions by the flavonoid luteolin associated with increased SIRT1 activity is reported in mice.

The second issue of *HBSN* also consists of three review articles and two original articles. Clinicians often disagree whether patients with NAFLD should drink alcohol. The paper dealing with the effect of alcohol on the development and the progression of NAFLD will give a clear answer to this important question. Furthermore, genetic variations in PNPLA3 in various liver diseases including NAFLD are summarized. Carotenoids, the pigments principally responsible for the characteristic yellow and red color of fruits and vegetables have generated research interests on their capacity to protect against fatty liver diseases. The effect of carotenoids that are associated with antioxidant and anti-inflammatory effects in NAFLD is discussed. The second issue of *HBSN* on NAFLD ends with two original reports on the effect of dietary saturated fatty acids on hepatic fat and fibrosis in rats, and on DGAT1-deficiency affecting cellular distribution of hepatic retinoids and progression of chemically induced hepatic fibrosis.

In summary the two issues focusing on fatty liver disease of *HBSN* do present not only an excellent up-to-date knowledge in this field, but also present exiting new data stimulating further research in this area.

We thank all the experts for their excellent contribution on this most important and rising topic in liver disease. We are also very grateful to the editors and staff of *HBSN*, particularly Editor-in-Chief, Prof. Yilei Mao and Editor of Science, Ms. Eunice X. Xu for their kind support in realizing this series on fatty liver disease.

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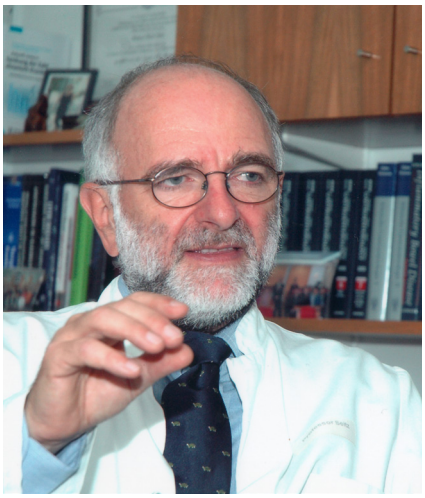

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