agent of liver cancer, particularly in high prevalence areas for liver cancer, while HCC is one the five major cancers globally. The world first universal HBV vaccination program was launched in Taiwan in 1984. The prevalence of HBV infection has been reduced remarkably to approximately one tenth of the original prevalence after the vaccination program. Further more, reduction of the HCC incidence in children aged 6–14 years have been demonstrated in the vaccinated birth cohorts. Recently we have further provided evidence that the effect of HCC prevention by universal HBV immunization program has been extended from childhood to early adulthood. The risk of developing HCC in HBV vaccinees was associated with incomplete HBV vaccination; prenatal maternal HBsAg seropositivity and HBeAg seropositivity. Failure to prevent HCC results mostly from unsuccessful control of HBV infection by highly infectious mothers. Future strategies to increase the global coverage rate of HBV immunization and to interrupt mother-to-infant transmission may further enhance the cancer prevention effect of HBV immunization. Successful prevention of chronic hepatitis B virus (HBV) infection can reduce the incidence of liver cancer. It is the first example of cancer preventive vaccine in human, which proves that prevention of the infection of an infectious agent can prevent its related cancer.

S9

Prevention of hepatocellular carcinoma in chronic hepatitis C infection

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Chronic infection with the hepatitis C virus (HCV) can lead to cirrhosis and hepatocellular carcinoma (HCC). HCV infection is found worldwide, with a prevalence of approximately 1-3% and an estimated total disease burden of more than 150 million persons. Studies in Asia, Europe and North America report a 1–4% annual incidence of HCC among subjects with HCV cirrhosis. In the USA, the incidence of hepatocellular carcinoma has doubled in the past 30 years and is expected to increase further in the next decade. Progression of liver disease among subjects infected with HCV in the 1960s to 1990s as a result of percutaneous exposure is the leading reason for the increase in HCC. Prevention of HCV-induced HCC could occur by: (1) prevention of HCV infection, (2) curative treatment of patients infected with HCV, and (3) chemoprevention among patients in whom HCV infection could not be cured. At present, there is no vaccine to prevent HCV infection. Fortunately, the incidence of HCV in the United States has decreased by more than 90% over the past 2 decades. Also, during the past 20 years scientists have found treatments to cure chronic hepatitis C infection. Studies in Japan, Europe and North America have reported that curing HCV infection through treatment with interferon and ribavirin reduces the risk of HCC by 70–90%. Regrettably, many patients with HCV infection are not candidates for treatment, or have failed to be cured with interferon/ribavirin. Several large clinical trials have tested the efficacy of chronic, low-dose interferon treatment in the prevention of HCC in the belief that suppression of HCV may reduce HCC risk; unfortunately, the results of these studies have been negative. Non-antiviral chemopreventive trials are in progress. Based on animal studies showing efficacy in preventing chemical hepatocarcinogenesis, the National Cancer Institute (USA) has funded a Phase IIa trial to determine whether S-adenosylmethionine (SAMe, AdoMet) is effective in reducing alpha fetoprotein (AFP; a tumor marker for HCC) in patients with HCV cirrhosis. The future of HCC prevention in HCV will likely test HCV protease inhibitors and HCV polymerase inhibitors, two classes of drugs which are remarkably effective in curing HCV infection in Phase II trials. Because of the cost and potential side effects of HCV protease and polymerase inhibitors, it is hoped that non-antiviral drugs which are safe, inexpensive and effective in preventing HCC can be found.

S10

Studies on *H. pylori* infection and risk for gastric cancer in Latin-American countries

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Inflammation as a driving force for the development of preneoplastic lesion is clearly exemplified in the case of gastric cancer and infection with *Helicobacter pylori*. *H. pylori* colonizes the gastric mucosa of humans early in life establishing an unusual long-lasting chronic inflammation of the mucosa. After decades of continuous dialogue bacteria-host, in some cases balance is broken leading to diseases, the more severe being gastric cancer. Aggressive bacterial products add to the potentially harmful uncontrolled inflammatory mediators to damage epithelia. Reactive oxygen and nitrate compounds produced by activated inflammatory cells would also threaten DNA stability. Risk to develop gastric cancer is multifactorial and genetics of both, the host and the bacteria are important, as are other environmental factors such as diet or habits. All this factors vary from country to country and between different ethnic groups. Latin-American countries constitute a mosaic of humans groups with different genetic background and in consequence a mosaic of genetically diverse *H. pylori* strains. Thus, it is not surprising to observe Latin-American countries with the highest world mortality rates like Chile or Colombia and countries with low rates such as Mexico or Argentina. Differences in rates of *H. pylori* infection does not explain the differences in GC mortality as in most Latin-American countries prevalence in adults is over 80%, including Mexico or Colombia. We have found sequence differences in genes associated with virulence such as cagA or vacA that may partially explain their differential carcinogenic potential. Regarding areas of high risk (mountains) versus low risk (coasts) in countries such as Colombia, the presence of cagA gene and of the vacA s1m1 isotype have been found more frequently in areas with high GC risk. *H. pylori* strains from high risk areas have also shown to induce higher levels of oxidative and nitrosative reactive compounds from inflammatory cells. Regarding human genetics, our studies in Mexico and Paraguay suggest polymorphisms in genes of the innate and inflammatory responses such TLRs, HLA or TNF may increase the risk for gastric cancer. Polymorphisms in IL-1 have been reported associated with GC in some Latin-American countries but not in others. As for environmental factors, whereas consumption of cola-soda and smoking increase risk, eating vegetables or fruits rich in antioxidants have shown some protection and regression of early precancerous lesions. Whereas *H. pylori* infection is considered the principal cause for GC, this infection is not enough to cause disease and studies on the host and his environment are also necessary to better define groups at risk for GC.

Session 4. Prevention of Prostate Cancer: PSA-Screening and beyond

S11

Nutritional aspects of primary prostate cancer prevention

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There are three well-known and indisputable risk factors for development of prostate cancer, namely heredity, ethnic origin and increasing age. Geographic variations in incidence rates are considerable and, therefore, it has been suggested that environmental factors may also play a role. Data from