infections can result in the Trichuris dysentery syndrome (TDS). This syndrome includes chronic dysentery, rectal prolapse, anaemia, poor growth, and clubbing of the fingers. The severe stunting in TDS now appears likely to be a reaction at least in part to a chronic inflammatory response and concomitant decreases in plasma insulin, plasma insulin-like growth factor-1 (IGF-1), increases in tumor necrosis factor-α(TNF-α) in the lamina propria of the colonic mucosa and peripheral blood (which likely decreases appetite and intake of all nutrients) and a decrease in collagen synthesis. Improvements in cognitive performance have been found after treatment for relatively heavy infections in school age children. Synergistic associations between hookworm and other helminths has been described. In a recent study from Brazil, 61% of individuals harbored mixed helminth infections. Multivariate analysis indicated significant positive associations for co-infection with hookworm and *S. mansoni* and for co-infection with hookworm and *A. lumbricoides*. Co-infection with hookworm and Ascaris resulted in higher egg counts for both, suggesting a synergistic relationship between these species, although, the intensity of *S. mansoni* or *A. lumbricoides* co-infection did not differ from that of mono-infection. Another study from from Brazil looking at Hookworm and Ascaris infection and the impact of polyparasitism on cognitive performance in Brazilian schoolchildren suggested that hookworm may be associated with poorer concentration and information processing skills while *A. lumbricoides* infection may be associated with poorer general intelligence. Polyparasitized children seem to experience worse outcomes that children with only one helminth infection. In yet another study, multivariate analysis revealed that stunting was significantly associated with ascaris infection among children and adolescents, whereas low body mass was significantly associated with hookworm infection among adults and the elderly.

Strongyloides stercoralis can cause acute infection, chronic infection and hyperinfection syndrome. Hyperinfection syndrome has been associated with a variety of risk factors and predisposing conditions, including new immunosuppressive therapy therapies; HTLV-1 infection; cadaveric transplantation; immune reconstitution syndrome; hematological malignancies (especially lymphoma). Co-infection with with HTLV-1 results in decreases in IL-5, and parasite specific IgE responses in patients with strongyloidiasis consistent with a relative switch from Th1 to Th2 response leading to an increased risk of autoinfection resulting in hyperinfection syndrome. Co-infected patients with HTLV-1 and strongyloides may not respond as well to anti-helminth treatment. In addition to HTLV-1, corticosteroid use remains one of the most frequent risk factors for hyperinfection syndrome. Hyperinfection syndrome presents with diverse symptoms and signs often leading to misdiagnosis on the clinicians part. It is associated with a high mortality rate (15-87%). Therefore, increased recognition is important for clinicians caring for at-risk patients.

Of the five major species of Schistosomiasis pathogenic to humans the only one endemic in South America is *Schistosoma mansoni*. Despite the efforts in carrying out integrated control programs during the last 25 years, there are still regions where the prevalence of *S. mansoni* is over 50%. in *S. mansoni* is reported in acute schistosomiasis and in chronic disease. Recent studies from Brazil suggest that pulmonary hypertension may be more common than previously thought in individuals with hepatosplenic disease due to *S. mansoni*. Similarly, recent studies suggest that hepatopulmonary syndrome also occurs in patients with *S. mansoni* who also have periportal fibrosis and portal hypertension. Although CNS involvement is rare, it is well described. Transverse myelitis or seizures have been described in both acute and chronic infection.

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12.004

Food-borne Toxins

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Food-borne toxins are an important cause of morbidity in the unwary traveler. In rare situations, deaths may occur. Education is the key to prevention and a careful history is usually the key to diagnosis.

Ingestion of contaminated fish and shellfish is one of the commonest causes of poisoning and the risk from marine toxins appears to be increasing as a result of multiple factors such as global warming, coral reef damage and spread of toxic algal blooms. Important examples include ciguatera poisoning from ingestion of large carnivorous coral reef fish, puffer fish poisoning and various shellfish poisonings such as paralytic shellfish poisoning. Scombroid poisoning occurs in open ocean fish such as tuna and mahi mahi that contain histidine in the flesh. Inadequate chilling after capture results in conversion of histidine to histamine and symptoms that resemble an acute allergic reaction.

In most cases the presence of toxin does not affect the appearance, smell or taste of seafood and it is not destroyed by cooking, smoking, freezing or drying. Onset of illness typically occurs soon after ingestion of contaminated food and produces gastrointestinal symptoms such as diarrhea, nausea, vomiting and abdominal pain often followed by a variety of neurological and cardio respiratory symptoms. Paradoxical dysesthesiae such as temperature reversal (hot objects feel cold and cold objects feel hot) are very characteristic of ciguatera and neurotoxic shellfish poisoning.

Treatment is usually symptomatic and supportive. In the case of scombroid poisoning antihistamines provide specific treatment and in the case of ciguatera poisoning intravenous mannitol may reduce the severity and duration of some of the neurological features. Diagnosis is usually based on a careful history. Test kits that detect ciguatoxin in contaminated fish are commercially available.

Ackee poisoning and cassava poisoning are examples of food poisoning from non-marine sources. Ackee poisoning occurs after eating unripe ackee fruit and results in vomiting and life threatening hypoglycemia. Acute and chronic cyanide poisoning may occur after ingesting cassava root products containing cyanogenic glycosides. Acute poisoning causes diarrhea, vomiting, mental confusion and death.

Chronic intoxication causes abnormal thyroid function and various neurological disorders.

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Viral hepatitis (Invited Presentation)

13.001

Epidemiology of Chronic Viral Hepatitis in Latin America

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Chronic viral hepatitis caused by Hepatitis B or C viruses are major health problems in this beginning of the 21st century. Estimated prevalence in the world population in different regions range between less than 1% to more than 3% for HCV and between less than 2% to more than 8% for HBV, affecting more than 400 million people in the world. In Latin America, prevalence estimates are flawed. For HCV it varies from less than 1% to 2%, and for HBV from less than 1% to more than 8%. Numbers can be as high as 15% in the Amazon region.

In Latin America, some surveys report HBV prevalence as high as 21.4% in Dominican Republic and 7.9% in Brazil, followed by 3.2% in Venezuela and 2.1% in Argentina. Low prevalence was found in Mexico (1.4%) and Chile (0.6%). For HCV, rough estimates project more than 10 million infected people. Many surveys were conducted by blood banks, but results are biased by sampling problems.

In Brazil, HCV prevalence studies estimates had found a wide range, varying from 0.4% to 5.9%. A population based study in 2007 found a HCV antibodies prevalence of 0.28% to 2.61% and a HCV-RNA from 0.02% to 0.9% in different regions of the country. From 1994 to 2005, the Ministry of Health database has registered 52,440 HCV cases. Recently, a national survey was conducted by the Ministry of Health, but results are not published yet.

In São Paulo state, there were 30,299 HCV cases registered from 2002 to 2008 and 14,810 HBV cases in the same period. In the city of São Paulo it is estimated a mean prevalence of 1.42% (95% confidence interval 0.7 — 2.12%). Diagnosis can be done with blood tests, but availability is a concern in poor countries.

Treatment is expensive and fairly effective, implying in high morbidity, mortality and costs. Chronic hepatitis is a great challenge for the health systems in Latin America.

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13.002

Update on Hepatits B Therapy

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Tha main goal for the treatment of chronic hepatitis B (CHB) is to prevent advanced hepatic disease: cirrosis,hepatic failure and hepatocellular carcinoma (HCC). The first aim of treatment is to achieve sustained suppresion of HBV replication as well as the remission of liver disease.The sustained supression of virological replication varies widely,depending on the population on treatment, therapeutic agent s, treatment duration and less clearly from genotype. Since 1992,eigth therapeutic agents have been approved worlwide (INF alfa, lamivudine, adefovir, entecavir, PegINF alfa-2ª thymosin alfa1, ,telvibudine and tenofovir) but only some of them are used in different countries according to national regulation.. When and how to treat an CHB depends on the HBV DNA levels, ALT and status of HBeAg. For HBeAg(+) patients,the endpoint of treatment is HBeAg seroconvertion. Therapy is considered in GHB with HBV DNA leves of 20.000 IU/ml or higer (HBeAg positive patient) or 2.000 IU/ml (HBeAg negative), although lower HBV DNA levels migth be selected when evidences of progresive disease are identified. ALT normalization and HBV DNA suppression are the measures of reponse to therapy. Oral nucleoside analogs (NA) is a significant contribution for treatment in the last years,but a major concern with this agents is the selection of antiviral resistant mutations. This may be identified prior to virological breakthrough or at the same time. Peginterferon alfa-2ª, entecavir and tenofovir are currently included in the first-line treatment choice on the basis of their potency as well as the low rate of antiviral drug resistance. The strategy of drugs combination in CHB treatment for achieving a sustained virological response and some end points has beeen explored and the level of HBV DNA suppression. This combination theraphy is encouraging in some clinical trials.

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13.003

Hepatitis C Treatment Today and the Future

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Therapy of chronic HCV infection is based on the use of the combination of pegylated interferon and ribavirin. Sustained virological response (SVR), a negative HCV RNA 24 weeks following discontinuation of therapy, is the most important surrogate parameter to achieve. Actually, SVR is obtained in about 50% of patients with genotypes 1/4 and in 80% of the patients with genotypes 2/3.

Patients infected with genotypes 1 or 4 must be treated for 48 weeks. But, if the patient achieves a rapid virological response (RVR), defined as a negative HCV RNA at week 4, we can consider a shortening of treatment. In patients with a slow response to treatment (HCV RNA only negative between weeks 12 and 24) the length of therapy must be extended to 72 weeks. For patients infected with genotypes 2 or 3 treatment should be planned for 24 weeks.

New drugs are needed for non-responders and for those who are not good candidates to treatment.

Several new oral agents, more potent, less toxic and allowing for shorter duration of treatment are being developed. These new drugs are designed to inhibit several viral enzymes. Results of recent clinical trials using inhibitors of NS3/4A protease or inhibitors of NS5B polymerase in combination with peginterferon/ribavirin are promising. These studies demonstrated that adding telaprevir or boceprevir (the protease inhibitors in the most advanced phases of evaluation) to peginterferon/ribavirin improved the rates of SVR