The cestode *Echinococcus multilocularis* is widely known in the Northern hemisphere to infect mainly foxes and rodents and accidentally human. However human seems not to be equal facing the alveolar echinococcosis (AE) disease. Many factors such as environmental conditions, immunology or genetic susceptibility can explain the heterogeneity of infection in human. Few data existed concerning the influence of the *E. multilocularis* genetic diversity. Up to now, the genotracking of subspecies was impossible due to a lack of fast evolutionary markers. Recently, a tandem repeated multilocus microsatellite, named EmsB, was discovered in the *E. multilocularis* genome. Our objective was to assess the limit of EmsB discriminatory power at three geographical scales: (i) at a world scale, by studying Alaskan, European and Asian endemic areas, with parasitic lesions from definitive and intermediate hosts, (ii) at a continental scale by studying adult worms circulating in so-called old and new endemic areas in eight European countries and (iii) at a local scale (n x 100km^2) in the French region of the Ardennes. The hierarchical clustering analysis of the results helped us to explore relevant questions as the spatial dynamic, the circulation of the parasite between different hosts, the emergence or the re-emergence of AE in human, to increase our knowledge on the parasite transmission and to enhance the public health management.

(53) Identification of the iron superoxide dismutase gene repertoire in *Trypanosoma brucei gambiense*

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Superoxide dismutase (SOD) forms part of the defense mechanism that helps to protect organisms from superoxide anions. This enzyme is one of the isoenzyme systems commonly used to differentiate T. b. gambiense from T. b. brucei and T. b. rhodesiense. To understand the genetic basis of the differences observed between SOD electrophoretic profiles of T. brucei sub-species, we undertook the identification and the characterization of SOD gene repertoire in T. b. gambiense. This study was performed on 7 stocks (4 T. b. gambiense group 1 and 3 group 2) showing different SOD profiles. Four SOD genes (soda, sodb1, sodb2 and sodc) were identified in T. b. gambiense genome. These genes were cloned and their predicted amino acid sequences were deduced. Few differences were observed between nucleotide sequences of the four SOD genes of T. b. gambiense group 1 and 2 stocks. Even with T. b. brucei, few differences were observed. Several amino acids specific to FeSOD were found in the four SODs sequences of T. b. gambiense. Aligning the four T. b. gambiense protein sequences with those of others organisms, important differences were found with MnSOD and

Cu/ZnSOD, but high similarity with FeSOD; indicating that the SODs of T. b. gambiense are FeSOD. High similarity exists between the proteins sequences of T. b. gambiense and T. b. brucei. Despite the differences observed in SOD electrophoretic profiles, there is a genetic stability of the SODs genes in T. brucei sub-species.

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(54) Can the evolution of avian influenza virus (AIV) be predicted?

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Because the previous influenza pandemics were caused by either viruses of avian origin or reassortant viruses with some of their genomes derived from avian viruses, it is believed that the H5N1 AIV, which already causes limited outbreaks in human, may become the source of the next pandemic. Continuously evolving, the virus may adapt to the point that it can transmit efficiently in human population. An insight in the viral evolution may help us to estimate the risk, understand the cause, and prevent the emergence of a pandemic strain. Trying to understand the viral evolution, we need to look at the viral dynamics and selection pressures. While the evolution of human influenza viruses is driven mainly by immunological selection pressure, it is not clear what drives the evolution of AIV. The current H5N1 AIV has diverged into multiple sublineages. Because these sublineages or clades are antigenicly distinct, it is likely that immunological pressure has played a role in the divergence of H5N1 AIV. It is also likely that the immunological pressure was the result of massive immunization in poultry, especially in China. The recent emergence of the new Fijian-like strains further supports this notion. In general, genomes of most AIV are in evolutionary stasis because of they are in equilibrium with their natural host. Once transmitted to a new host species, the virus starts rapid evolution to adapt itself to the new host. Virus-host interaction is, therefore, considered an important factor in the evolution of H5N1 AIV. Genome analysis has revealed positive selection pressure on some parts of the viral genome. Some of these sites may eventually fix and become host-specific residues when the virus reaches the optimum in the new hosts. Although the avian-human inter-species barrier may involve several genetic determinants, the most important one may be the receptor binding preference. A few point mutations in the receptor-binding pocket of hemagglutinin have been shown to increase human-type receptor binding of H5. We have found such mutation in viral quasispecies from a human respiratory specimen. This suggests that there is a selection pressure driving the virus toward human-type receptor specificity in human infection. Viral genomes may be shaped not only by selection pressures but also mutational bias. Recent observation has shown that avian and human influenza viral genomes are different in their GC content. It was suggested that this indicates a difference in mutational bias between the two hosts. However, I would like to point it out that this may not be entirely correct. Genome composition may also be influenced by codon composition, which could be affected by selection pressure. We analyzed codon volatility,

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