A family of unpredictable viruses

The large H.N. family

The avian influenza viruses are programmed to make use of cells in the respiratory and digestive tracts to multiply. They provoke epizootic outbreaks that are well known to veterinarians. Apart from the H9 subtype virulent in poultry, the H5 and H7 subtypes currently are host to the most aggressive viral strains.

Highly pathogenic avian influenza outbreaks in the world

1959	Scotland	H5N1	
1963	England		H7N3
1966	Ontario (Canada)	H5N9	
1976	Victoria (Australia)		H7N7
1979	Germany		H7N7
1979	England		H7N7
1983	Pennsylvania (USA)	H5N2	
1983	Ireland	H5N8	
1985	Victoria (Australia)		H7N3
1994	Queensland (Australia)		H7N3
1994	Mexico City (Mexico)	H5N2	
1994	Pakistan		H7N3
1997	New S. Wales (Australia)		H7N4
1997	Hong Kong	H5N1	
1997	Italy	H5N2	
1999	Italy		H7N1
2002	Hong Kong	H5N1	
2002	Chile		H7N3
2003	Netherlands		H7N7
2004	Pakistan		H7N3
2004	Texas (USA)	H5N2	
2004	British Columbia (Canada)		H7N3
2004	South Africa	H5N2	
2005	Southeast Asia	H5N1	
2005	China	H5N1	
2005	Romania	H5N1	
2006	Turkey	H5N1	
2006	50 countries infected by	H5N1	

The genetic drift strategy

Type A avian influenza viruses are varied and unpredictable because they use a short term survival tactic. Through slight genetic modifications, they escape the immune defenses of a host while exploiting for as long as possible the population vulnerable to the infection.

Two mechanisms at play

Genetic drift occurs through an accumulation of replication errors in a slow process to which organisms may adapt themselves. It is responsible for an antigen shift that allows a virus to partially escape a host's immune response or to progressively adapt to a new host species.

In contrast, genetic reassortment occurs when two viruses trade genetic material, which is only possible if the same cell is infected simultaneously by these two viruses.

Sacred ibis flying in the sky over Chad, 2006 – Alexandre Caron, © Cirad

Feared combinations

Genetic reassortment can have two consequences: a major antigenic modification and the acquisition of new characteristics of virulence or adaptation to a host species.

The combination of these two consequences resulted in the emergence of the viruses responsible for the Asian (1957) and Hong Kong (1968) flu in humans, springing from a recombination of a human and an avian virus. Three of the genes in the 1957 H2N2 virus came from an avian influenza virus and the 5 other segments came from a H1N1 virus derived from the virus responsible for the Spanish flu (1918-1919).