Why Specialists Are Missing the Point on Norwegian H1N1 Mutation

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Nov. 27, 2009—A tiny mutation detected in one of the RNA strands of the AH1N1 virus has led to worldwide speculation as to whether this means the virus is about to turn into another 1918 pandemic. The flaw in nearly all reported thinking on the subject is the reductionist assumption, pervasive in modern molecular biology, that the behavior of an organism is completely determined by its genetic composition.

Now, the eight strands of RNA in the H1N1 virus, with their strings of thousands of nucleotide base trios (which will ultimately code for amino acid building blocks of proteins), are being endlessly gone over by virologists like penitents counting prayer beads.

It began with the discovery recently of a mutation in the virus's RNA strand coding for hemagglutinin (one of the two glycoproteins on the surface of the viral envelope responsible for the specificity of viral binding to host cell membrane surfaces) found in two dead flu victims in Norway. In both, probably independently, there was a change at prayer bead number 225 from a D to a G (a change in coding from amino acid aspartic acid to amino acid glycine). Laboratory studies have given some evidence that this amino acid change can lead to a change of binding specificity from alpha(2-6)- to alpha(2-3) sialic acid containing receptors in the human respiratory tissues. The fear is, that if this mutation proliferates and becomes widespread, it can lead to a new wave of more lethal flu outbreaks--similar to what happened in 1918.

It should be noted, however, that both forms of the gene have been found throughout the H1N1 virus's history, from 1918 up to today. In 1918, both forms were found separately in dead soldier tissues. Throughout the intervening years, both have been found. Therefore it is not surprising that both exist today. In fact, a study has shown that flu virus passed through egg—as is done in the lab to grow viruses, and also for vaccine production, the proportion of G to D variants at 225 grows. This is not surprising, because eggs would have avian-type alpha(2-3) sialic acid receptors, so there

would be a selection for that variant in grooming the virus to grow on the eggs.

The point is, we still do not know what makes the H1N1 pandemic virus kill certain persons, and we certainly don't know what genetic changes, if any, are necessary to turn the 2009 virus into a monster like the 1918 virus. It is very likely that it would take many factors totally outside of the virus's "control" to lead to a 1918-like pandemic. In this sense, the virus is merely hitchhiking on the opportunities presented through the large-scale actions of man upon the biosphere (just like HIV and just like the bacterium causing bubonic plague). Large-scale war, famine, and social disruption in the Fourteenth Century enabled the bubonic plague, a rodent disease, to begin spreading like wildfire among humans. It was not a random point mutation in the plague bacterium's genome that caused the death of one-third of the people in Europe. It was man's actions as society collapsed, and as the noösphere contracted sharply, which brought on the horror.

The best thing societies can do to avoid a plague is to keep moving forward, not regressing, in all scientific and social endeavors. It is through man's reason and reasoned action that he gains control over the biosphere, including the parts of the biosphere we call diseases. Wherever human existence is degraded through poverty, ignorance, and lack of access to clean water, adequate living space, adequate food, and proper sanitary infrastructure, disease will advance. If such collapse becomes widespread, a plague will be ignited, and even the rich will not be spared. If you force humans to live like rats, you die like a rat.