More evidence for the reality of genetic entropy

I found the article 'More evidence for the reality of genetic entropy', by Robert Carter, in vol. **28**(1) enlightening and exciting. However, there is something I don't understand: he mentions that health authorities should be not be concerned with mutating human viruses since these are degrading—that I understand. But then he says we should be concerned with *new* viruses and mentions ones jumping from animals. But why would new viruses from animals not have the same degradation going on since they have presumably gone through many generations in the animal population? I suspect I am missing something very important which I would like to understand.

Earl Rodd
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» Robert Carter replies:

Great question. The answer lies in the origin of viruses, which is a contentious issue. There are many virus particles in your gut, more than the number of bacteria, but these are mostly normal denizens of your intestinal flora and do not cause disease. Instead, they act to regulate the bacterial flora. For flu viruses specifically, all extant strains circulate among aquatic wild fowl, and do not generally cause disease. There is something about the virus in its natural state (assuming this *is* the natural state; I do not know their function in birds) that allows them to persist apparently indefinitely. It is when a virus jumps to a new host, one without a viral control mechanism, that they burn hot, burn fast, and eventually burn out. Perhaps they will burn out in their host species, eventually, but an out-of-control virus gives us enough reproductive cycles in a short enough time to actually measure the degeneration. So, no, you did not miss anything and you are thinking straight. I just failed to provide all relevant details, and even here I was too brief as additional caveats and glosses are coming to mind as I write.

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Human genome decay

I read with interest the subject article in the current edition of *Journal of Creation*. I had one concern that popped out at me. The author makes considerable use of mutation rates which have their basis in a study involving the Hutterite community. I would question the usefulness of such data for the purpose of extrapolation over larger classes of humans over long timeframes. The Hutterites constitute an excessively inbred community which one would expect to be characterized by much higher than normal genetic entropy rates than those found in the general population. According to Steinberg (1967), the entire extant Hutterite population could be traced back to 90 ancestors who lived from the early 1700s to the early 1800s. This does not make them a good candidate population for use in such as Williams undertakes.

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» Alex Williams replies:

In-breeding may result in a higher rate of genetic injury in progeny but that has little to do with baseline mutation rates. Indeed it may result in a decrease in the long-term genetic entropy rate because it leads to a higher rate of elimination of homozygous mutants. The measured mutation rate in the Hutterite study is similar to another recent whole genome family study: Conrad, D.F. *et al.*, Variation in genome-wide mutation rates within and between human families, *Nature Genetics* **43**:712–714, 2011; doi:10.1038/ng.862. My conclusion from both studies is that our best estimate of the whole genome error rate is currently ~40 new mutations per generation.

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