The Cytokine Storm Theory of Flu Fatality:

Does it Affect Nutritional Recommendations?

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Since H5N1 has not arrived, there's still time to develop ideas about how to deal with it, in the event it does arrive. I am limiting nutritional recommendations detailed in Flu Report 1 – "Facts about the Flu, How to Prepare for it, and How to Fight it" to conventional flu. I am reserving nutritional recommendations as to H5N1, as more information is obtained from the limited human cases that have been identified to date.

A central question is in what ways is H5N1 or "super flu" different from ordinary flu. The similarities are more apparent, especially as we move down the ladder from H5N1 to less virulent bird flu strains, which many commentators optimistically forecast are more likely than H5N1 to come our way. We may not know what we're dealing with until such time as it gets here, and even then we may not know for sure whether we're contending with a super flu or simply a new conventional flu strain to which we have no preexisting immunity. As we'll discuss in a moment, the distinction between "super" and "conventional" may make a difference nutrition-wise.

One difference discussed in Flu Report 1 is that whereas millions of people are exempt, by way of prior exposure and acquired immunity, from a given human flu strain, bird flu is unrecognizable to virtually all human immune systems when initially crossing the species barrier. This fact alone, at such time as it becomes readily communicable among humans, can generate much higher flu infection rates and with it more secondary infections and deaths. It is believed that all forms of human influenza originated in animals, and human flu was first identified when animals were domesticated and people began establishing permanent settlements. The question of greatest potential consequence is to what extent H5N1 or a similar super flu is qualitatively different from ordinary flu, because this could mean that remedies helpful against one may be counterproductive against the other.

About the flu generally and cold viruses we know this: a strong, efficient immune system works to the advantage of resolving infection. Conversely, with nearly every form of infectious disease, and flu in particular, an immune-compromised state is associated with greater severity of infection and higher death rates. Epidemiological evidence from the bird flu pandemic of 1918 suggests that young adults were disproportionately afflicted, but were they not also disproportionately exposed relative to the aged who tend to live more in isolation? The deadly 1918 flu strain arrived as troops were returning from WW1 battlefields, and mirrored troop movements with the military port cities of Brest, France and Boston, Massachusetts most heavily affected. Massive troop movements, crowds, and congestion associated with demobilization of soldiers, many of whom were returning home to a spouse of similar age, may be the underlying reason why the young adult population was hardest hit by the post-war pandemic.

The Cytokine Storm Theory maintains that H5N1 behaves differently from ordinary flu, turning the immune system against itself, via

pro-inflammation proteins called "cytokines." Cytokines are produced by the body as a result of certain types of stress. Cytokines can generally be thought of as markers of temporary biochemical disequilibrium caused, for example, by infection or the stress of intense exercise. They are associated with tissue microtrauma, and are involved in the positive adaptations induced by exercise. One thing we know about avian flu is that it causes extensive respiratory tissue damage, more and deeper than conventional flu, and this may explain why a cytokine storm is observed. Another view is that the tissue damage is caused by cytokines and cytokines are overstimulated by H5N1.

I would caution against mistaking correlation for causation, by positing that cytokines are the cause of bird flu fatality, when the cause is bird flu itself. A "cytokine storm" may be the body's blunt-instrument device for dealing with extremely virulent infection, whereby it destroys its own cells in hopes of killing enough of the virus to turn the course of the infection toward recovery. It seems likely that anything that attacks cells also attacks viruses attacking cells. I would further caution against a corollary to the cytokine storm theory that we should take measures to suppress immune function during bird flu infection. This is the pinnacle of risky treatments, and I question the scientific premise upon which it is based.

The cytokine storm theory is vaguely reminiscent of the AIDS theory holding that the HIV virus kills by attacking immune cells preferentially, when in fact, the syndrome known as AIDS is largely enabled by, and symptomatic of, a compromised immune system. The AIDS theory asserted the disease is caused by a virus that disables the immune system, while the cytokine storm theory of avian flu suggests that the immune response itself is an attack upon the body. If the cytokine storm theory were accurate, its implications would require nutritional recommendations applicable to colds and conventional flu be modified. For the reasons stated in this article, I question the theory, at least insofar as it implies that immune suppression is a viable means of reducing avian flu mortality. However, the issue of the applicability of nutritional approaches to treating ordinary flu, to treating avian flu, remains unresolved.