Can You Catch Alzheimer's? Forget It

By Josh Bloom — September 10, 2015

If nothing else, Alzheimer's Disease (AD) is one wicked mystery. Its importance has grown steadily, if for no other reason that in most developed countries, people are living longer. Since AD correlates very closely with age, it is a given that as people live longer the incidence of AD will rise. The trend or increasing lifespan is not only obvious, but shows no sign of abating. Not only are people living longer, but every year, without exception, life expectancy increases even more.

(Source: World Bank)

AD has been the black hole of biomedical research. Despite an enormous effort from drug companies, the NIH, and universities, there is nothing out there even remotely close to even stop or slow progression, let alone reverse it.
The number of failed clinical trials for AD drugs and vaccines at least 225 to date is staggering. There are a few drugs[1] that mitigate some of its symptoms (barely), but also carry with them some fairly nasty side effects.

By any measure, the therapeutic options for AD are suboptimal at best. Not only are there no good treatments, but the cause of AD is unknown, which further hampers research efforts. The hallmark of AD is the presence of beta-amyloid plaques in the brain, but it's unclear if these plaques are a cause of AD, or a result of it.

Many drug companies have had long-running, enormous projects focused on inhibiting the enzyme (beta-secretase) that is thought to cause the plaques. But inhibitors of beta-secretase have not been useful, despite a number of trials focused on this.

Uber-blogger Derek Lowe, the founder of the "In the Pipeline" blog, summarizes some of the approaches[2] that have completely bombed in the clinic. It's not pretty.

And, Matt Herper, writing in Forbes[3], provides an overview of some of the frustration that is associated with AD research.

A new study[4] that was recently published in Nature, discusses an intriguing, but very speculative slant on AD the possibility that it could be transmitted in a similar manner to that of infectious diseases.

Lead author Zane Jaunmuktane, of the Division of Neuropathology at The National Hospital for Neurology and Neurosurgery in London, and colleagues postulate that the very rare Creutzfeldt Jakob disease[5] (CJD), which has shown to be transmissible via medical and surgical procedures and cannibalism, may play a role in the development of AD.

CJD is nothing if not strange. Fortunately it is rare about one person in a million will get it. This is fortunate, since it is invariably fatal, usually within a year.

What makes CJD especially interesting is that it is caused by prions, the strangest infectious agent you will ever see. Prions are unique because they are the only pathogen without any genetic components. No DNA, no RNA, just a protein that has the terrible property of interfering with the shape of functional proteins in the brain, which renders them non-functional. This makes prions unique in the world of infectious pathogens.
There are other very rare, exotic diseases in the same family as CJD. They are all part of a family that is called transmissible spongiform encephalopathies (TSEs), and are, thankfully, uncommon. The term spongiform refers to the appearance of brains upon autopsy. They are full of holes. The best known member of the family is bovine spongiform encephalopathy, aka "Mad Cow Disease."

Prior to 1985, 30,000 people worldwide mostly children who were not growing at a normal rate were treated with hGH that was obtained from pooled extracts from the pituitary glands of thousands of cadavers. It was subsequently found that some of the extracts were contaminated with CJD prions.

By 2012, there were 226 people in Europe and the U.S. who died from prion infection. (That number is certain to rise, since the incubation period from this infection is about three decades.)

This is where it gets interesting.

The Nature researchers examined the brains of eight people who died from CJD after becoming infected by the "old" hGH when they were young. Prior to their death, none of the had clinical symptoms of AD. But, of the eight cases where CJD was confirmed, six of the brains had plaque deposition which looked just like brains from AD patients.

The authors proposed that there may be an infectious component of not just CJD, but AD as well. Although this is plausible, it would be a mistake to make too much of this study.

Even if their theory is found to be true, there is no way to treat CJD, so there is virtually no chance that these findings even if verified will impact AD anytime soon, if at all. Rather, the study is an intriguing (but probably small) piece of a very complex puzzle that, despite thousands of researchers trying to solve it, is still mostly a black box. This is unlikely to change anytime soon.

This study should be viewed as interesting, but a very long way from contributing to a therapy against AD.