Sildenafil for Cognitive Decline?

Upon retrospective analysis of insurance records, Viagra use was associated with a 69% reduced risk of Alzheimer's disease over six years of follow-up.

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A great deal of media attention was garnered by a recent study reporting that the use of sildenafil (Viagra) was associated with a reduction in risk for Alzheimer's disease. The study was an elegant combination of informatics, epidemiology, and cell biology. Networks of genes and proteins associated with the two hallmarks of Alzheimer's — beta-amyloid and tau — were evaluated in an effort to identify drugs that impact both, with the hypothesis that these might represent candidates for treating Alzheimer's.

Four candidates were identified: one, sildenafil, turned out to be associated with reduced risk for Alzheimer's disease (about 70% reduction in men and 30% in women). Another one, lansoprazole (aka Prevacid), was associated with increased risk for Alzheimer's. The other two, dantrolene/Revontro (which prevents calcium release inside muscle cells, thus a sort of muscle relaxer) and deferoxamine/Desferal (which binds iron), had too little data to determine whether or not their use may be associated with any change in the risk for Alzheimer's.

So of the four drugs identified-- one good, one bad, and two equivocal-- sounds almost random. But then anything that might be helpful here would be welcome. The researchers went on to show that sildenafil, when applied to neurons or microglia (immune cells in the brain) that had been generated from Alzheimer stem cells, increased neuronal process growth and reduced the phospho-tau that is found in the tangles in Alzheimer's. That, if nothing, else is intriguing.
It is also possible that, being a drug of vasodilation, chronic sildenafil use (or tadalafil or vardenafil for that matter) might prove helpful in dementia types rooted more in vascular insufficiency but prove unhelpful or less helpful in those rooted in toxic exposure, inflammation, oral/sinus/gut infections, insulin insensitivity, and hormone and/or nutritional deficiencies.

Overall though, at least these two fundamental questions remain, and perhaps even a third: #1 Does sildenafil actually cause the reduced risk, or is this simply a non-causal association? It is well-documented that the brain changes of Alzheimer's begin about two decades (yes, you read right) before an actual diagnosis. So perhaps the people who had "pre-Alzheimer's" prior to frank diagnosis had less interest in or need for using sildenafil. Perhaps too, they had more balanced hormone levels and more robust social connections-- and thus better brain support. #3 What if any implications might this finding have for the relative dementia newcomer, HIV-associated neurocognitive decline (aka "HAND")?

#2 The study only addressed risk-- not treatment. And, needless to say, it is a big leap from risk reduction to reversal of cognitive decline. The only sustained reversals of cognitive decline in persons with Alzheimer's or "pre-Alzheimer's" have been with multi-pronged nutritional and lifestyle interventions such as is being offered at George Washington University, via Buck Institute on Aging's Dale Bredesen (of "The End of Alzheimer's" renown)'s "ReCODE" program, and various collaborative private clinics sprinkled across the country. It seems quite unlikely that a PDE-5 inhibitor will end up showing clinical utility against dementia, but clinical trials are underway to try to find out.
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