

PANEL DISCUSSION QUESTIONS

Questions answered by Nancy Barbas, MD, MSW

1. My brother was put on Aricept. When we gave him the medication he was unable to function, not even eat because he slept until the drug wore off. He is 69-70. We decided to discontinue the drug, then he was alert. We did check with the doctor but he encouraged giving him the Aricept. What else should we/can we do to help keep him alert?

It is difficult to comment on the specifics of a situation. Some individuals do experience side effects associated with Aricept which can include sleepiness, worsened confusion, vivid dreams, restless legs, and most commonly, upset stomach. Interestingly, even though Aricept (Donepezil), Rivastigmine, and Galantamine are all in the same class, sometimes an individual tolerates one better than another. I recommend discussing any changes with his treating doctor.

2. My mom has been on Aricept for four years, went quickly from diagnosis of AD five years ago to being completely immobile and unable to talk. Is this unusual? Is there therapy available to help her talk?

There is a wide range of course of progression of symptoms in individuals with Alzheimer's disease. Aricept will not reverse or stop the progression. It might be worthwhile discussing her course of symptoms with her treating physician to consider whether there may be other medical factors contributing to her symptoms.

3. For those living at home, can pharmaceutical companies come up with weekly rather than daily medications?

This is a great idea. Weekly medication administration would be very helpful for many patients and families. I don't know if any companies are in the process of developing this type of preparation--medication delivery would have to be through a very slow and steady route. I will certainly spread the word about the need for such a preparation.

Question answered by Jeffrey M. Burns, MD

1. In your studies, how do you control for other interventions? i.e. medications, dietary, etc.

We use a stretching program as a control group for comparing to our aerobic exercise group. The stretching program is conducted the same number of times a week and for the same length of time per day as the aerobic exercise program. This allows us to assess the effect of aerobic exercise rather than potential confounding benefits of increased social activities, attention from exercise instructors, etc.

Questions answered by Nikos Scarmeas, MD

1. "Is there any difference between olive oil and virgin olive oil in the benefits we receive from them?"

Virgin olive oil has lower acidity and is not chemically processed. In theory it may be better but we have no true evidence for this, in particular for neurological disease.

2. "What amounts of vitamin E and C are required for a measurable improvement?"

There is no evidence that vitamin E or vitamin C supplementation offer any benefit in terms of Alzheimer's disease.

3. "Is the MeDi prolonging death in AD, do patients progress more slowly (have prolonged functionality) thru GDS-FAST stages when diet is adherent to MeDi guidelines while alive?"

There have been no formal studies on this. A preliminary look into our data (not published yet) did not show any MeDi effect on rates of cognitive or functional decline (after AD onset) but this could be a methodological limitation of our ability to reliably measure this decline.

Questions answered by Roger Albin, MD

1. What is the link if any between DLB and Parkinson's disease?

We think they are part of a spectrum of Lewy body disease.

2. What is the frequency of autopsy? (I think they mean of our research subjects, but not sure).

Around 90%.

3. a) Is it possible to biopsy brain tissue if PET scan is not nearby? b) If so, what are the chances to see DLB in tissue biopsy?

Brain biopsy is almost never done as it doesn't change management.

4. I am taking part in a UM research study, brain imaging. Have signed and returned all paperwork to donate my brain to UM, but how can I MAKE SURE this happens?

Contact Kris Wernette at 734 936 5894

Questions answered by Sid Gilman, MD

1. Does UM offer specialized EEG testing for MCI/Alzheimer's?

EEG testing is generally not helpful in the diagnosis of Mild Cognitive Impairment and Alzheimer's disease.

2. What is the connection between caffeine intake and onset of AD?

As far as we know, there is no relationship between caffeine intake and Alzheimer's disease

3. Is there research that supports the use of Enbrel in slowing the progression of AD?

Enbrel (Etanercept) is an immunosuppressant drug used to treat rheumatoid arthritis, psoriasis, ankylosing spondylitis, psoriatic arthritis and juvenile rheumatoid arthritis. A physician in Los Angeles has demonstrated Enbrel by injecting the drug into the muscles at the back of the neck and has claimed that there is slowing of disease in patients with Alzheimer's disease. This work has not been published in reputable journals and the claim lacks credible evidence.

4. What is the source/sources of funding for your research?

Our research is funded almost fully by competitive grants from the National Institutes of Health (NIH)

5. Are there any toxins identified as precursors besides lead? (pesticides, volatile cleaning agents)

No toxins have been identified that are linked to the development of Alzheimer's disease. The linkage of lead to Alzheimer's disease is a hypothesis only and not a proven toxin with a causative role in the disease.

6. Is chelation treatment tried?

Yes. A company in Australia, Prana Biotechnology, tried using Clioquinol, a chelating agent, but it proved to be toxic to humans and the trial was stopped. They have a new chelating agent, PBT2, which is in clinical trials currently. The idea is to chelate heavy metals from the brain, as heavy metals are needed to form fibrillar beta-amyloid deposits.

Questions answered by Sid Gilman, MD, CON'T

7. What methods are there of detoxifying?

Immunotherapy is being studied as a means of removing beta-amyloid from the brain. Currently there are several approaches in clinical trials, including Bapineuzumab from Janssen and Wyeth pharmaceuticals. There is another trial under way by Lilly CO. and multiple other companies have clinical trials in progress. This approach is not a true detoxification process, but rather a means of reducing the amounts of a damaging protein in the brain.

Questions answered by Judith Heidebrink, MD

1. Could you discuss the hypothesized relationship/association between the herpes simplex virus I and AD? Is this a valid association? How might HSVI vaccines help prevent AD? HSVI co-localizes with amyloid - is this investigated in amyloid related treatment studies?

Herpes simplex virus I is the virus that causes cold sores. Most people have been exposed to this virus, even if they have no symptoms. The virus typically lies dormant within the nervous system. In brain tissue from persons with Alzheimer's disease, HSV 1 viral DNA has been discovered within amyloid plaques. However, it is not known whether HSV 1 plays a causal role in the development of Alzheimer's disease. Thus, it is unknown whether antiviral therapy or an HSV1 vaccine would help prevent AD. I am not aware of HSV 1 being investigated in amyloid treatment studies.

2. In an article I recently read, it indicated that "Benadryl," an antehistamine, can interfere with the cells ability to transmit signals. However, the Russian study in your presentation indicated that antehistamines may help treating cognitive disorders. Can these contradictions be explained?

Both Benadryl and the Russian medication Dimebon are antihistamines, but they have other chemical effects that differ. Benadryl is anticholinergic (blocks acetylcholine, a neurochemical that is important in cognitive function). Dimebon is not anticholinergic. It may actually have slight cholinergic properties (facilitates acetylcholine).

3. What % of AD patients become immobilized? How common is it four years into the disease?

I don't know a precise %, but immobility typically occurs very late in the course of Alzheimer's disease, at a time when the cognitive impairment is very severe. Immobility within the first few years of AD is uncommon. Other medical conditions (e.g. musculoskeletal problems) or a dementia other than AD (e.g. Dementia with Lewy Bodies) might be contributing if an individual has early immobility.

Questions answered by Bruno Giordani, Ph.D

1. What are the neuropsych tests that used to determine Mild Cognitive Impairment?

Same tests as for dementia. You look at whether the person is oriented to person, place, and time to a sufficient degree (MMSE), then you look at each of the cognitive areas to fit MCI (memory, attention, visual-spatial, etc.).

2. Will you accept individuals with an autoimmune disease as a research volunteer?

Depends on the study, but most likely not, due to the possible cognitive compromise.

3. How is it possible for a person in stage 5 or moderately severe decline to consistently say they have no memory problem and get angry if it is suggested that there is an issue?

It is not at all uncommon. Some patients have very little insight into their difficulties. It is hard to judge which patients. Perhaps those with the most frontal involvement end up with the least insight, as we use our frontal lobes to better understand our position in terms of others.

4. Do alcoholics (both active drinking and in recovery) have a higher risk of AD?

Research is not clear. There is a dementia associated with drinking and there is definite brain damage associated with severe drinking. In some cases, though, you see a patient who has stopped drinking heavily for some time and suddenly develops a dementia and gradual decline. I think anything that affects the integrity of the brain increases our risk to at least some degree. Also, those who are significant drinkers also often have other associated medical conditions or problems that increase their risk (e.g., head injuries, metabolic disorders, poor eating habits).

5. Is it typical for a person to make up an answer to a question (when they cannot remember) and they really think they are telling the truth? What makes them do this?

This is confabulation, in which the patient comes up with an associated response that seems to be triggered in the brain and not inhibited by the frontal lobes, almost as if there are poor connections and associations are not made well. At other times, though, some patients appear to make up responses that they hope will not be noted by others and they appear aware that they are incorrect, but are trying to cover their lack of knowledge.

6. Is there a study planned to evaluate/follow children of parents with AD to determine hereditary impact?

There are several underway. We don't have one here, but NIH.gov probably lists some or the Alzheimer's Association website, www.alz.org