

No cure yet, but progress made in managing and detecting Alzheimer's

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Alzheimer's disease isn't something you must either passively accept or dread, a panel of doctors and scientific experts told an audience of more than 500.

There are new reasons to think Alzheimer's can eventually be cured or prevented, perhaps with drugs already on the market, according to the panel.

People can also strengthen their cognitive muscles in advance so their brains can better resist decline, panelists said at the annual Date With A Cure, held by Alzheimer's San Diego at Qualcomm Hall in Sorrento

Valley, Calif. And they can help research by volunteering for clinical trials. Hispanics, who are underrepresented in studies, are especially needed.

Gloom descended on the field in March, when clinical testing of a much anticipated drug called aducanumab was halted for lack of efficacy. The [drug](#) clears a toxic brain protein called [beta amyloid](#) believed to trigger the disease. But the treatment apparently was given too late. So researchers have increased efforts to find drugs to prevent the disease.

Certain HIV drugs may prevent Alzheimer's, said panelist Dr. Jerold Chun of Sanford Burnham Prebys. These drugs inhibit an enzyme called reverse transcriptase that copies HIV into the genome of host cells. HIV patients, who get these drugs, have an extremely low rate of Alzheimer's compared to the general population Chun said.

The drugs might work by preventing genetic changes from taking place in the brain of Alzheimer's patients, caused by units of DNA that jump around the molecule, changing its sequence.

These changes during the patient's lifetime may help explain why anti-amyloid drugs haven't worked, Chun said. They target a single form of amyloid, while [genetic recombination](#) might generate thousands of varieties of amyloid that current drugs can't touch.

Doctors can prescribe reverse transcriptase inhibitors now to individual patients who in their judgment are at risk for Alzheimer's as an "off-label" use, Chun said. Risk factors include genetic variants, age and symptoms of early cognitive decline. But to move from these one-off uses to large-scale adoption, controlled clinical trials are necessary.

To give Alzheimer's drugs their best chance of success, those at elevated risk should be treated before damage begins. Major progress toward this

goal has been made, said panelist Dr. Paul Aisen. He heads the University of Southern California's Alzheimer's Therapeutic Research Institute, based in San Diego.

Currently, amyloid deposits are detected in expensive PET imaging tests, Aisen said. These are useful to find appropriate subjects for clinical trials. But they're too costly for general use. So subjects are selected by pre-screening for early symptoms of potential decline.

But in a few years, an inexpensive blood test will make mass screening of cognitively normal people feasible, Aisen said. Intervening in the earliest stage of the disease process will increase the odds of success, he said.

Aisen also cautioned against relying on unproven supplements to ward off cognitive decline.

"There is no evidence that any of them work," Aisen said during a question-and-answer session with the public. "These are marketing schemes. They're not based on science, regardless of what the commercials say ... I'm quite sure none of these supplements work."

Building up brain resilience can help, Aisen said in a pre-event interview. This can be seen with people who show strong evidence of amyloid buildup but show no symptoms of impaired cognition, he said.

"That's going to involve lifestyle changes. It's going to involve cognitive exercise, social activity," Aisen said. "We need to continue to refine our understanding of brain resilience. But this is something that can be implemented during the lifespan and can really change the landscape of Alzheimer's symptomatology."

Further down the road, advances in [stem cell research](#) on Alzheimer's-

related [genetic changes](#) may lead to better drugs, said Dr. James Brewer, a neurologist at UC San Diego.

Drugs previously tested often originated in mouse models of the disease, which Brewer said have proven to be flawed. But it's now possible to take skin cells from patients and turn them into stem cells and then into brain cells, so human-pertinent models can be made.

Salk Institute scientists have helped by developing converted brain cells that retain the genetic marks of aging, Brewer said. This makes more realistic models, because Alzheimer's is an age-related disease.

UCSD's Shiley-Marcos Alzheimer's Disease Research Center is also looking to increase the diversity of study participants by reaching out to Hispanics in the South Bay. Hispanics have a different pattern of genetic risk than non-Hispanic whites, Brewer said. So more Hispanic participation is needed to ensure treatments are effective for them.

In addition, that differing risk pattern could yield clues into which combinations of factors are most important, Brewer said. For non-Hispanic whites, variations in a gene called APOE play a significant role in elevating or lowering risk of Alzheimer's. This isn't as true for Hispanics, and it is unknown why that is.

"There are different [risk factors](#) for different cultural backgrounds, and so we need more participation from across the diversity of the community," Brewer said.

Alzheimer's San Diego will present a documentary on the search for an Alzheimer's cure from 10 am. to 12:30 p.m. on Sept. 17. The documentary, "Turning Point" will take place at 8695 Spectrum Center Blvd, San Diego. Go to www.alzsd.org/turningpoint/ for more information.

Go to www.alzsd.org/ for more information from Alzheimer's San Diego on research and for support for families affected by Alzheimer's.

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