

Is Alzheimer's disease transmissible?

Five people treated for pituitary dwarfism decades ago with human growth hormone (hGH) pooled from cadavers have shown cognitive decline reminiscent of early-onset Alzheimer's disease. Their dementia likely arose from transmission of the bits of amyloid-beta protein that lie behind Alzheimer's delivered along with the needed hormone, initiating a molecular chain reaction that led to brain effects decades later. Recombinant DNA technology has since provided a pure source of the hormone.

The cognition decline in these people is iatrogenic – caused by a medical procedure. The pooled hGH included infectious proteins, called prions (pronounced “pree-ons”), short for “proteinaceous infectious agent.” The research appears in *Nature Medicine* from long-time prion researcher John Collinge, director of the University College London Institute of Prion Diseases, and colleagues.

The team followed 8 patients. Two of the five with clinical signs of Alzheimer's died during the investigation, and autopsy revealed the telltale brain changes. Two other patients had mild cognitive impairment, and the eighth had no symptoms. None had mutations that cause Alzheimer's disease, ruling out genetics as a cause.

Although the study shows that Alzheimer's disease is potentially transmissible, the researchers urge that the findings be interpreted with caution and in context.

“We have found that it is possible for amyloid-beta pathology to be transmitted and contribute to the development of Alzheimer's disease. This transmission occurred following treatment with a now obsolete form of growth hormone, and involved repeated treatments with contaminated material, often over several years. There is no indication that Alzheimer's disease can be acquired from close contact, or during the provision of routine care,” said first author Gargi Banerjee.

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A primer on prions

A prion disease arises from a protein that can twist, loop, and fold into more than one three-dimensional shape (conformation), even though the underlying amino acid sequences are identical – like pop-beads of 20 colors bent into different shapes. Rarely, one guise of such an acrobatic protein becomes infectious, a prion. It gloms onto others, forcing them to fold in the infectious pattern and propagate it. Prions also clog the tiny cell components that serve as trash receptacles (proteosomes), spreading devastation and enabling cellular debris to accumulate, like New York City during a trash collection strike. In this manner, the nefarious prions “seed” organs, turning an organ like the brain into something resembling a Swiss cheese.

Prions can pass to individuals beyond the ones in which they originate under special circumstances –

such as injecting hGH extracted and pooled from cadavers.

Transmission of Alzheimer's is a first, albeit under extraordinary circumstances. Protein misfolding and spread in several other conditions suggest a prion-like mechanism at work. These include alpha synuclein deposits in some cases of Parkinson's disease, forms of ALS and Huntington's disease, Creutzfeldt–Jakob disease (CJD), and Gerstmann–Straussler syndrome. These conditions also have inherited forms.

More than 85 animal species develop prion diseases. The first to be described was in sheep that rubbed themselves raw against fences to relieve itch – their condition was named “scrapie.” Their brains became riddled with holes. Scrapie spread as the animals were fed mashed brain matter from their fellow ovines.

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A stylized slice through an Alzheimer's brain depicts amyloid-beta plaques in brown and tau tangles in blue.

From cannibalism to mad cows to CJD

The first prion disease of humans, kuru, was discovered among people living in the remote mountains of Papua New Guinea in the 1950s. The story is one of my favorites in all of biology; I've told it in several books.

“Kuru” means “to shake.” The disease began with wobbly legs, then trembling and whole-body shaking.

Uncontrollable laughter led to the name “laughing disease.” Speech slurred, thinking slowed, and walking and eating became impossible. Death came within a year.

The disease was traced to a ritual in which the people ate their war heroes to honor them. When women and children prepared the brains for consumption, prions entered cuts and abrasions and they became infected. At first, the disease was thought to be inherited because it affected relatives. But the women and their children fell ill from a shared environmental exposure, not a mutation.

The new report in Nature Medicine fleetingly mentions kuru and the cannibalism route to prion disease. “Since the cessation of this practice in the late 1950s, kuru gradually disappeared but enabled documentation of the range of incubation periods of human prion infection; the mean incubation period is approximately 12 years but can exceed 50 years,” the researchers write.

Back in the 1950s, not many people knew about the plight of the remote Fore people. Then in the mid-1990s, a similar prion disease dominated headlines in the UK when more than 120 people ate infectious prions in beef, and died. Popularly called “mad cow disease” after the effects on the unfortunate bovines, the condition was found to be a variant of a rare and horrific brain condition, Creutzfeldt-Jakob disease (vCJD).

Less well recognized was vCJD among people who’d received hGH pooled from the pituitary glands of cadavers, between 1959 and 1985, when recombinant hGH took over treatment. The dose was massive – typically 50 or more cadavers per year of treatment per patient, most of them children or teens with time for the infectious prions to spread. At least 1,848 patients with short stature developed vCJD, mostly in the UK. A history of human growth hormone therapy is here.

Autopsies of some of the patients revealed the classic amyloid-beta pathology of Alzheimer’s disease, but the CJD would likely have killed these patients long before the cognitive symptoms of Alzheimer’s had had time to manifest. But when researchers injected mice with saved samples of the tainted hGH, the rodents, which develop much faster, indeed showed the classic amyloid-beta plaques of Alzheimer’s.

Experts urge not to worry

When recombinant DNA technology was invented in the 1970s, it instantly provided a way to mass-produce a medically important protein without the taint of passage through an organism – like the gunky brain of an itchy sheep or a staggering, sick cannibal. Recombinant human growth hormone (rhGH) became available in 1985. And so the researchers urge that their report consider the history and not be taken out of context.

“There is no suggestion whatsoever that Alzheimer’s disease can be transmitted between individuals during activities of daily life or routine medical care. The patients we have described were given a specific and long-discontinued medical treatment, which involved injecting patients with material now known to have been contaminated with disease-related proteins,” explained Collinge. The possibility of transmitting amyloid-beta pathology should lead to ensuring that measures to prevent accidental transmission via other medical or surgical procedures are in place, he added.

Co-author Jonathan Schott stressed that although the circumstances that inspired the study are highly unusual, the findings “provide potentially valuable insights into disease mechanisms, and pave the way for further research, which we hope will further our understanding of the causes of more typical, late-onset Alzheimer’s disease.”

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