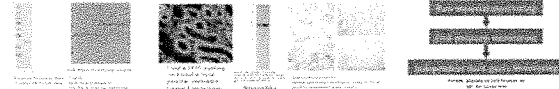


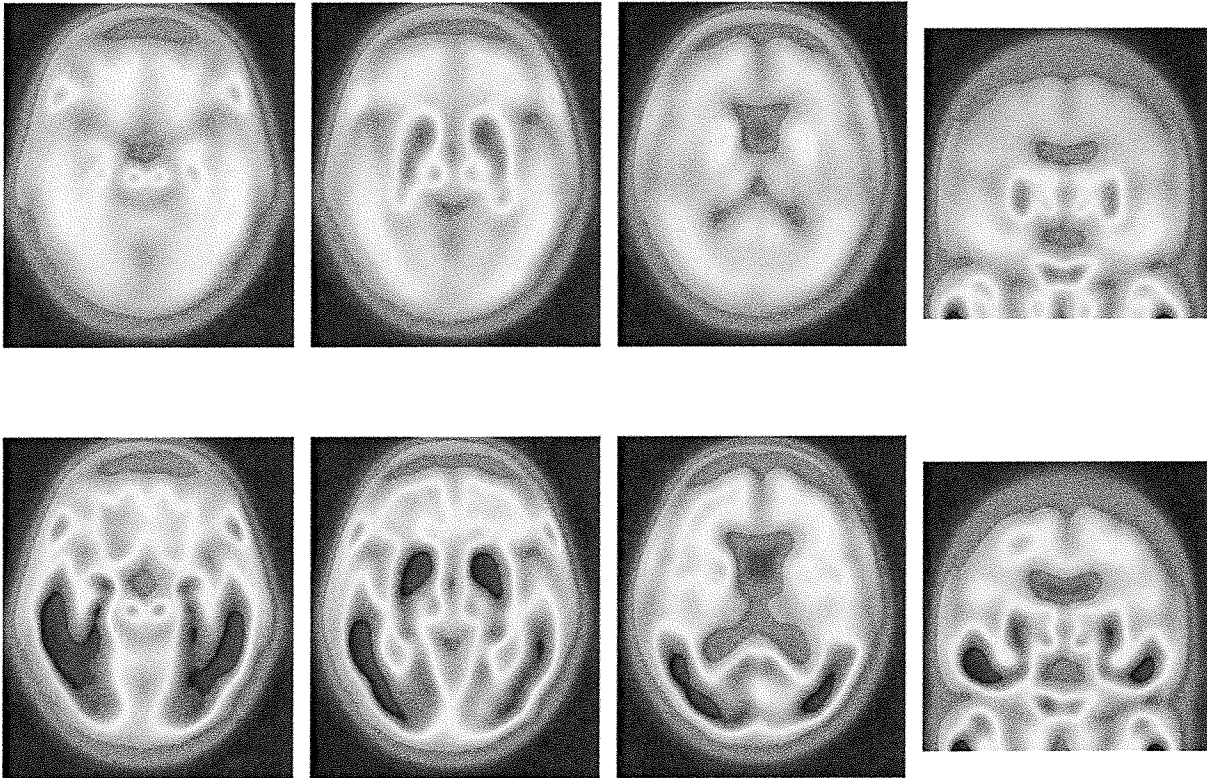
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PET scans showing tau deposits in the brains of healthy people (top) and those with Alzheimer's (bottom). The red areas indicate tau deposits.

Dr. Matthew Brier

Tau protein—not amyloid—may be key driver of Alzheimer's symptoms

By Emily Underwood | May. 11. 2016 . 2:00 PM

One of the telltale signs of Alzheimer's disease (AD) is sticky plaques of β -amyloid protein, which form around neurons and are thought by a large number of scientists to bog down information processing and kill cells. For more than a decade, however, other researchers have fingered a second protein called tau, found inside brain cells, as a possible culprit. Now, a new imaging study of 10 people with mild AD suggests that tau deposits—not amyloid—are closely linked to symptoms such as memory loss and dementia. Although this evidence won't itself resolve the amyloid-tau debate, the finding could spur more research into new, tau-targeting treatments and lead to better diagnostic tools, researchers say.

Scientists have long used an imaging technique called positron emission tomography (PET) to visualize β -amyloid deposits marked by radioactive chemical tags in the brains of people with AD. Combined with postmortem analyses of brain tissue, these studies have demonstrated that people with AD have far more β -amyloid plaques in their brains than healthy people, at least as a general rule. But they have also revealed a puzzle: Roughly 30% of people without any signs of dementia have brains "chock-full" of β -amyloid at autopsy, says neurologist Beau Ances at Washington University in St. Louis in Missouri.

That mystery has inspired many in the AD field to ask whether a second misfolded protein, tau, is the real driver of the condition's neurodegeneration and symptoms, or at least an important accomplice. Until recently, the only ways to test that hypothesis were to measure tau in brain tissue after a person died, or in a sample of cerebrospinal fluid (CSF) extracted from a living person by needle. But in the past several years, researchers have developed PET imaging agents that can harmlessly bind to tau in the living brain. In the new study, Ances and colleagues used one of these tags and an amyloid-binding one to analyze deposits of both proteins in 10 people with mild AD and

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The same relationship did not apply to β -amyloid, suggesting that—although β -amyloid PET scans can detect early stages of AD—tau is a better predictor of when people transition from the early, nonsymptomatic stages of the disease into mild Alzheimer's disease, Ances says. He suspects that it is the combined insults from β -amyloid and tau that drive the often-dramatic decline: Although the brain may be able to compensate for the deficits caused by β -amyloid, once tau starts to spread, “that pushes you over,” he says.

When the team measured tau in the study participants' CSF, it found that higher levels were specifically correlated with increased tau in the temporal lobe, a region involved in memory processing, Ances says. That's important, he suggests, because it means that one could potentially use tau in CSF as a diagnostic tool.

That's still a long ways off, says Pedro Rosa-Neto, a clinical neurologist at McGill University, Montreal, in Canada. The study took only a snapshot of participants' brains at a single point in time, so it can't prove any association between increased tau and mental deterioration. Still, he says, “it is a very nice paper” that provides a “first glimpse” of how tau and amyloid contribute differently to cognitive decline.

Ances says that large, longitudinal studies tracking both tau and β -amyloid in people over time are already underway. Ultimately, he says, the “dream” is that researchers will be able to tailor which AD therapy you need, “based on what's happening in your brain.”

Posted in: [Brain & Behavior](#), [Memory Topic](#)
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**Emily Underwood**Emily is a contributing correspondent for *Science*, covering neuroscience.
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Lane Simonian • 10 months ago

The key to Alzheimer's disease is nitro-oxidative stress. It is what leads to the formation of amyloid oligomers and plaques and what causes the hyperphosphorylation and/or nitration of tau proteins. Amyloid oligomers can contribute to that stress but they are not the principal cause. Hyperphosphorylated tau can interfere with neurotransmissions, but in a relative low oxidative environment tau can be de-phosphorylated.

A recent study found that amyloid is not harmful unless protein kinase C is activated. It is the activation of protein kinase C that leads to the formation of the primary oxidant in Alzheimer's disease: peroxynitrite. Peroxynitrite interferes with the transport, synthesis, and release of acetylcholine thus causing various forms of memory loss in Alzheimer's disease. Various peroxynitrite scavengers such as aromatherapy using rosemary, lemon, orange, and lavender essential oils, Korean red ginseng, and heat processed ginseng have partially reversed memory deficits in Alzheimer's patients in small-scale clinical trials.

1 ^ v • Reply • Share ›



Erich Mielke • 6 months ago

Is there any connection between Creutzfeldt Jacob disease and Alzheimer? Can be there any connection?

^ v • Reply • Share ›



It is !! → Erich Mielke • 9 days ago

Yes, I think so. They are a misfolding of the proteins. I think many amyloid diseases are caused by this misfolding of the proteins. MS, C.Jacob, Alzheimers (a lot of them). Amyloids is in cats, dogs even the Queens swans have it. This misfolding attacks many different body organs. From the brain, liver, heart... I think even more organs. What is that sheep one?... scrapie (?).. anyway from what I gather these are environmental as well as inherited. But, I'm just an accountant that has been reading about it because it's in the OSH breed of cats (oriental short hair). Researchers don't share their work.. I know last year Siemens was working on a "light up machine" is what I called it, that if an animal was given this liquid the machine would light up this goo where it was in the body. From what I gather... proteins misfold wrongly... then they travel around the body.. some go to the brain (alzheimers) some might go to the heart others to the liver. It's a kind of goo that attaches like glue to these organs messing them up & deforming them. I could be totally wrong but that is the way it looks to me.

^ v • Reply • Share ›



Jbar • a year ago

Sure, it's harmless if you don't mind having 5 trillion bits of antimatter exploding in your body!

^ v • Reply • Share ›



Sean Thomas Swift • a year ago

Sorry...but if you didn't first see plaques, you wouldn't be seeing tangles. The battle has always been over what the genesis of the disease is...not which post-cascade mechanisms are responsible for detectable symptoms. Your title is very misleading, if not disingenuous.

^ v • Reply • Share ›



Alice • a year ago

if you know the problem, then how can you correct it? I hope that soon you can cure this horrible disease soon

^ v • Reply • Share ›



Mindbreaker → Alice • a year ago

There is increasing evidence that a bacteria is ultimately responsible. When the brain's immune system is boosted, good things happen. Google IL-33 and bacteriophage M13. IL-33 is a protein that stimulates the brain's immune response to bacterial invasion. IL-33 is a critter that just goes after bacteria and kills it. Most antibiotics do not cross the blood-brain barrier.

I think the next test needs to be trying strong antibiotics that can cross the blood-brain barrier and can also make there way through the plaques.

At least a fraction...perhaps 30% of Alzheimer's cases are likely to be brain diabetes. And there is also interesting evidence that diabetes is caused by staph bacteria. When rabbits were exposed to staph...they got diabetes. Staph is not obvious unless the infection is large. Why might larger people get diabetes more often than skinny ones? One possibility is that skin folds either make cleaning skin more difficult, cleaning feet more difficult, or just make an environment that is more friendly.

IL-33 normally initiates an attack on E.Coli. So it is possible E.Coli is somehow getting in the brain and causing the condition or it is staph and IL-33 is provoking an attack on that too. There are any number of possible agents. The medical community is content to believe that there are many benign organisms/viruses in our systems...despite the fact that they know many cause harm in development or immune compromised individuals. It is looking increasingly likely that these "benign" organisms are harmful when carried for decades. Aids takes some time to develop after exposure to HIV, why do they persist in the dogma that none of these other infections cause problems?

The bacterias: Chlamydomphila pneumoniae, and Helicobacter pylori, have already been implicated in Alzheimer's disease as has the parasite:

[see more](#)

1 ^ v • Reply • Share ›



Be they profitable or not, organizations such as the Cure Alzheimer's fund are supporting any and all avenues toward treating and/or eradicating this disease. But across the spectrum of neuro- degenerative diseases (Alz., HD. PD etc), researchers are finding that post insult responses in the brain (be they trauma, bacteria, inflammation) often result in an increased production of beta amyloid- now widely seen as an auto-immune like response. Clinical trails aimed at shutting down its production have had little or no success, but have resulted in an increased understanding of the role of amyloid in the brain. It is thought by many that it is when the system becomes overwhelmed, and amyloid (and other proteins) begin to form plaques and conglomerate (as in Alzheimer's, eventually leading to the formation of Tau tangles), that the cascade accelerates...and cell death occurs unabated. A key component in many of these conditions appears to be the presence of and interactions with metals (copper, iron, zinc) stuck within these accumulating plaques which may circumvent the brains ability to clear these otherwise naturally occurring substances. There are trials going on with metal chelating agents (such as PBT2) which can cross the blood-brain barrier...target these metals...remove and redistribute them in the brain...and allow for the brain to recover its ability to clear these plaque causing substances.

1 ^ v · Reply · Share ›



It is !! ➡ Mindbreaker · 9 days ago

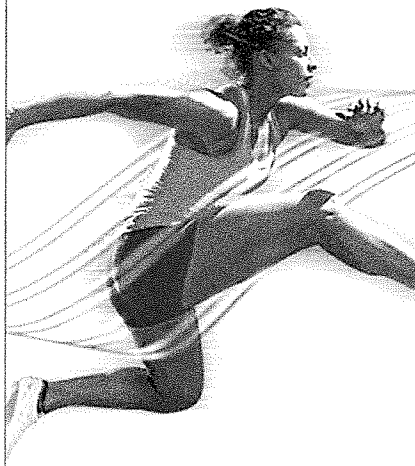
YES, excellent post. You're onto something... I also think some kind of antibiotic... I've been looking at this stupid amyloids because it is now running rampant in the OSH cat breed. Soon to cross over to the Siamese then my Russian breed of cat. So, I wanted to figure it out! I contacted several researchers and shouldn't have been at all surprised they do NOT share their research! So in the cats it is caught from nature OR it is also gotten via being inherited. In cats the liver explodes. What I was thinking (and I could be wrong on this) but, this amyloids is in a LOT of diseases (more than known). Now, I know at the U.of T a researcher there has made some kind of goo that when given to an animal they have a machine that can light it up (so you know where it is in the body). Siemens makes this light up machine. I think England (the Queens swans also got this disease)... so I think they have more research on it. Now there is an antibiotic (azithromycin) which I got for the cats.... this "toxoplasma" and the staph are in cats... so you might be onto something. Also, one of the bigger researchers (I won't name) didn't know it was in CATS! I said yes cats, dogs too (not so much) but even the Queens swans get this misfolding of proteins. I was wondering if it came from ticks (?)... something environmental (so if the animals got this... then man either eating or being around animals might pick it up?). I think cats get this chlamydia as well... How it got into man? If you want more info try to talk to researches in the Ukraine if possible (they are having a hard time now because of the problems w/Russia) but, a microbiologist there showed me a lot... PCR Testing....UV lighting. BUT, you have something about it being a bug that needs antibiotics.

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