Hva skjer'a?

Sverre Bergh, forskningsleder

AFS, Sykehuset Innlandet

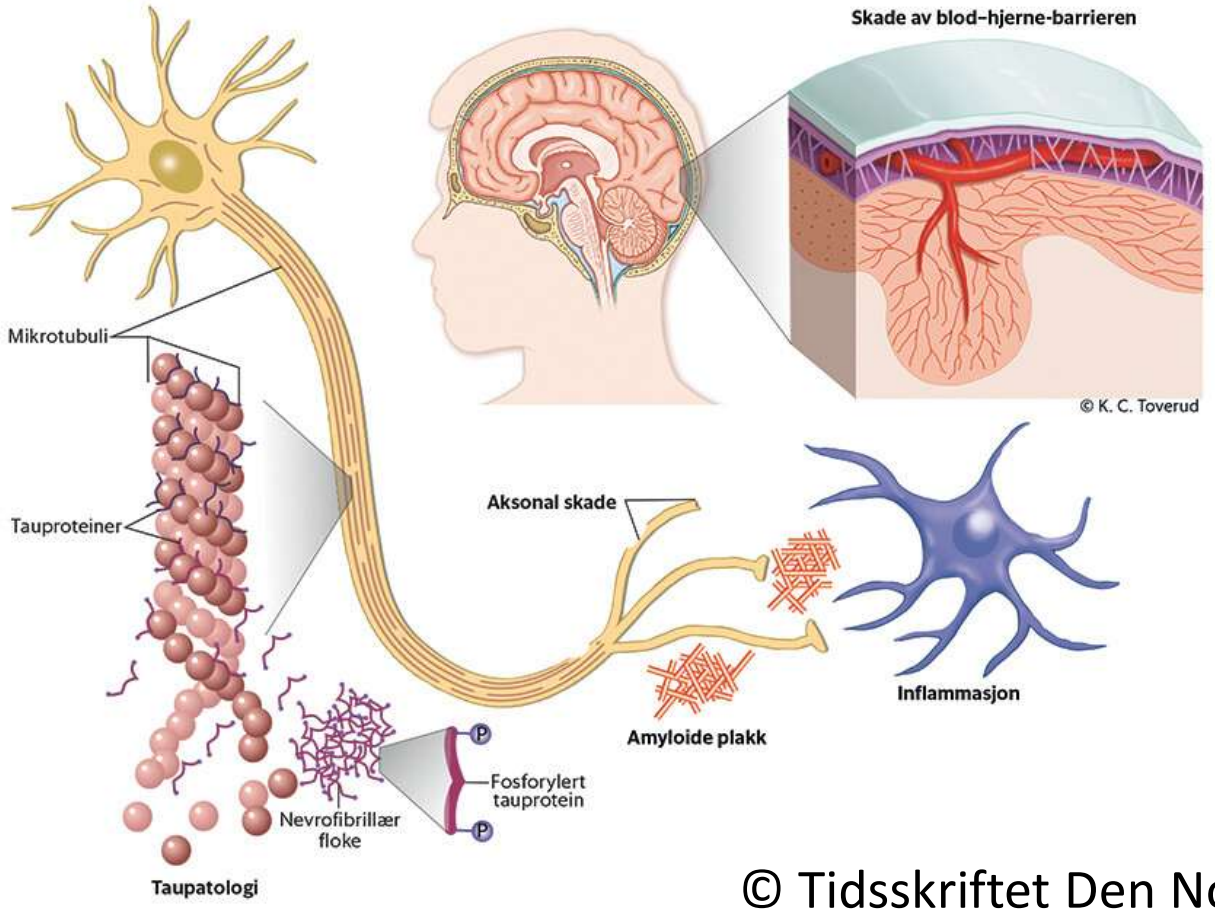
Norsk statistikk

- Dirty Oppland "Norsk Statistikk" [OFFISIELL MUSIKKVIDEO]

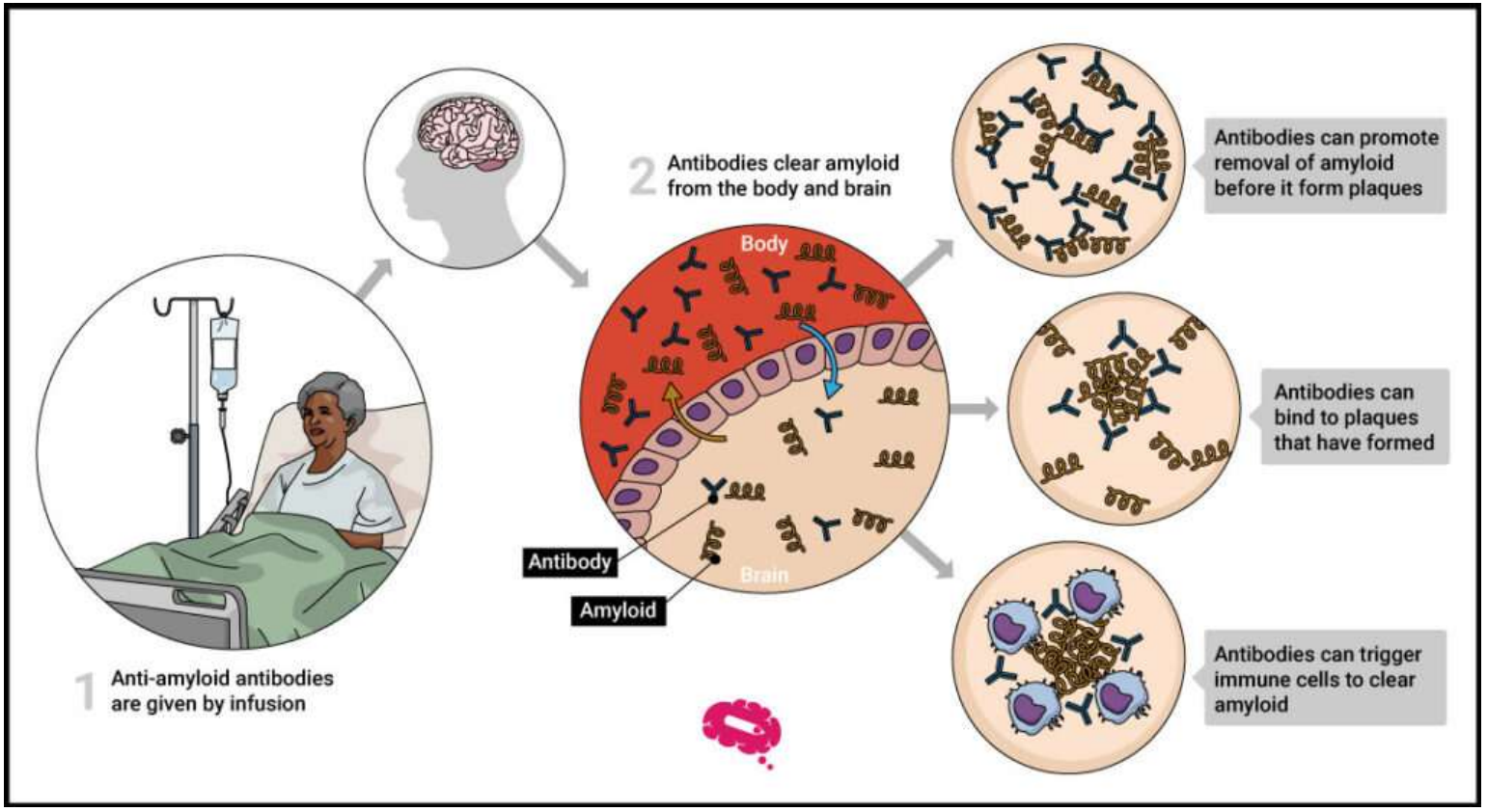
Disposisjon

- Hvordan virker de nye medisinene mot Alzheimer Sykdom (AD)?
- Hvor stor andel av eldre over 70 år i Norge ville fått medisinene?
- Hva er sammenhengen mellom vaksinasjon mot helvetesild og demens?

Nye medisiner mot Alzheimer sykdom

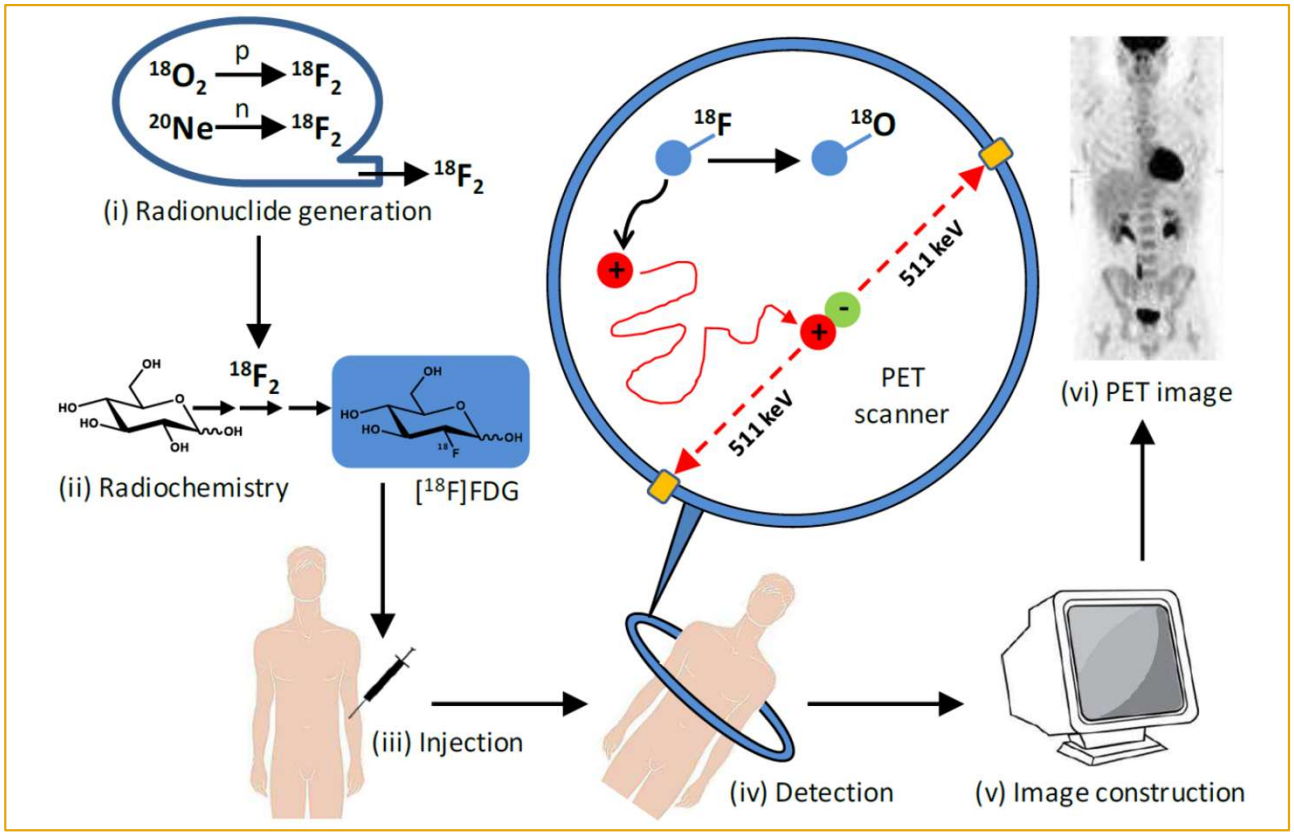


Behandling med Lecanemab



Mind the Graph

Amyloid PET



Patching, J of Diag Imag Therapy, 2015

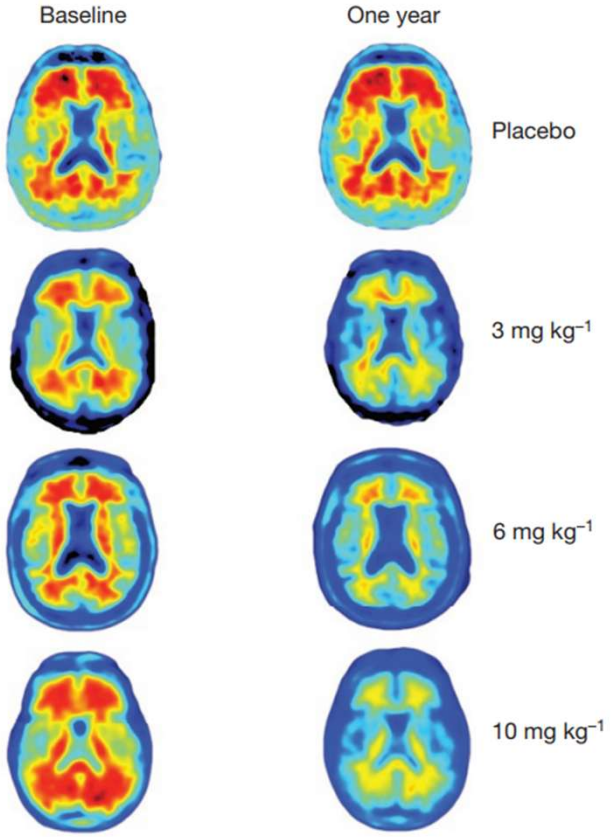


Figure 1 | Amyloid plaque reduction with aducanumab: example amyloid PET images at baseline and week 54. Individuals were chosen based on visual impression and SUVR change relative to average one-year response for each treatment group ($n = 40$, 32, 30 and 32, respectively). Axial slice shows anatomical regions in posterior brain putatively related to AD pathology. SUVR, standard uptake value ratio.

Sevigny, Nature 2016

AFS FORSKNINGSSENTER

Sykehuset Innlandet HF

Leqembi (lecanemab) i Norge

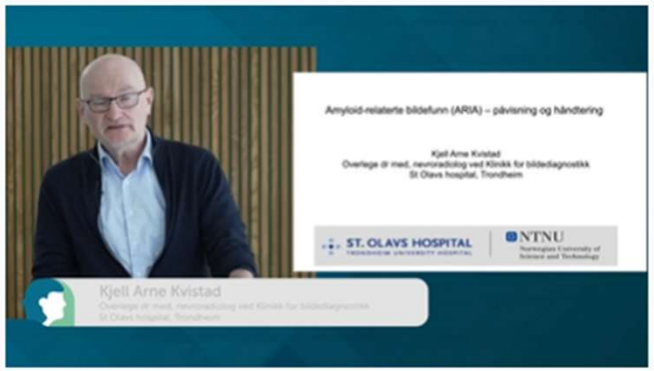
- Godkjent av Direktoratet for Medisinske Produkter (DMP)
- Metodevurdering pågår nå (Nye metoder)
  - Effekt, bivirkninger, kostnader
  - Nyttekriteriet, ressurskriteriet, alvorlighetskriteriet
- Selskapet Bioarctic planlegger lansering i år
- Beregnet kostnad 1 mill. per år inkludert oppfølging

Om aldring – Aldring og helse

2025

Ny behandling av Alzheimers sykdom – muligheter og utfordringer

V/ Geir Selbæk (Forskningssjef, Aldring og helse), Karin Persson (Spesialrådgiver Aldring og helse, Overlege geriatri, OUS), Stine Hynne (Erfaringsrepresentant, Nasjonalforeningen for folkehelsen) og Marianne Næss (Rådgiver, demens i Nasjonalforeningen for folkehelsen)

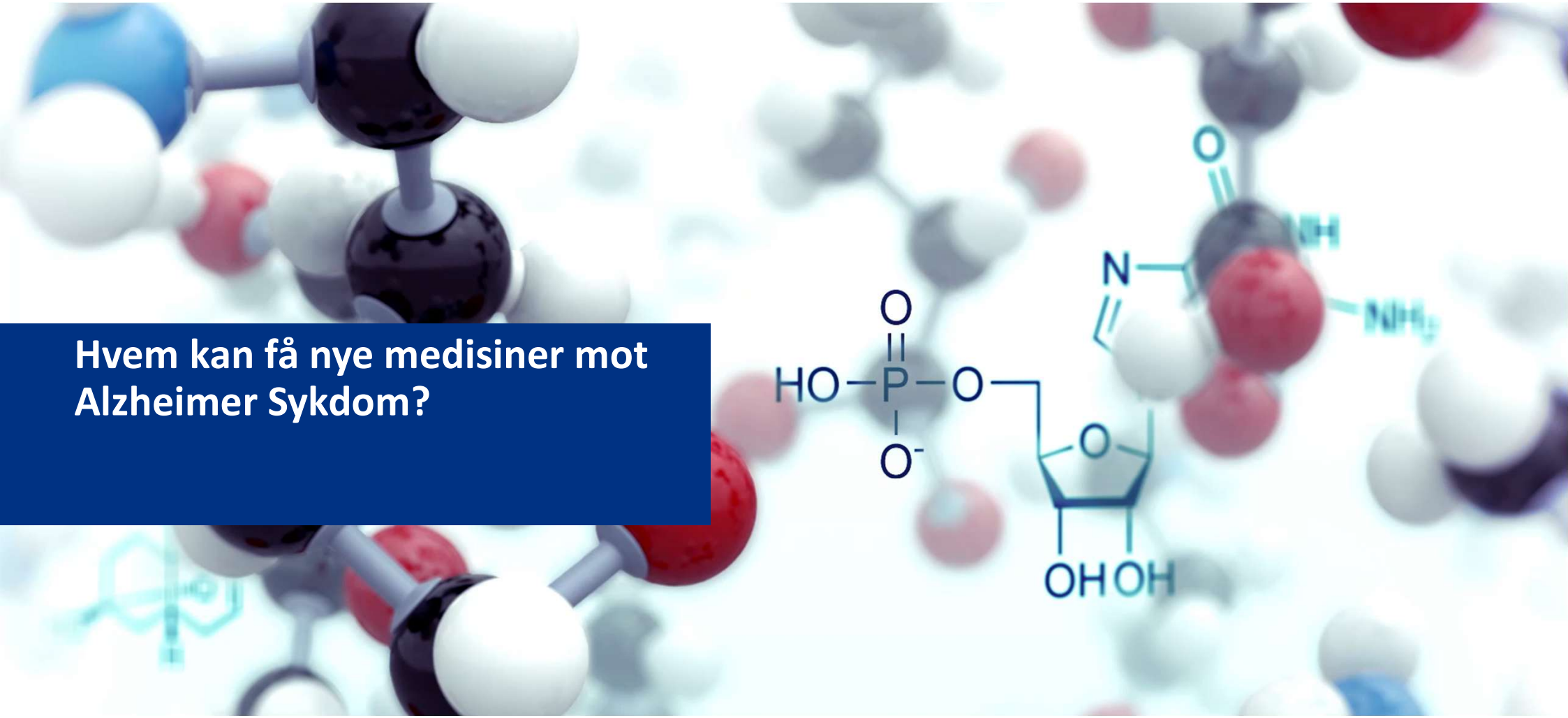


Amyloid-relaterte bildefunn ARIA – påvisning og håndtering

v/ Kjell Arne Kvistad, dr.med., spesialist i radiologi og overlege ved Klinikk for bildediagnostikk, St. Olavs hospital.

Sykehuset Innlandet HF | HELSE SØR-ØST AFS FORSKNINGSSENTER

Hvem kan få nye medisiner mot Alzheimer Sykdom?



Article

Prevalence of Alzheimer's disease pathology in the community

https://doi.org/10.1038/s41586-025-09841-y

Receive https://doi.org/10.1038/s41586-025-09841-y

Accepted: 31 October 2025

Published online: 17 December 2025

Check for updates

Dag Aarsland[1,2] ✉, Anita Lenora Sunde[1,3] ✉, Diego A. Tovar-Rios[1,4,5,6], Antoine Leuzy[1], Tormod Fladby[7], Henrik Zetterberg[8,9,10,11,12,13], Kaj Blennow[8,13,14,15], Kübra Tan[8], Giovanni De Santis[8], Yara Yakoub[16], Burak Arslan[8], Hanna Huber[8,17], Ilaria Pola[8], Lana Grötschel[8], Guglielmo Di Molfetta[8], Håvard K. Skjellegrind[18,19], Geir Selbaek[20,21,22] & Nicholas J. Ashton[8,23,24]

The prevalence of Alzheimer's disease neuropathological changes (ADNCs), the leading cause of cognitive impairment, remains uncertain. Recent blood-based biomarkers enable scalable assessment of ADNCs[1]. Here we measured phosphorylated tau at threonine 217 in 11,486 plasma samples from a Norwegian population-based cohort of individuals over 57 years of age as a surrogate marker for ADNCs. The estimated prevalence of ADNCs increased with age, from less than 8% in people 58–69.9 years of age to 65.2% in those over 90 years of age. Among participants aged 70 years or older, 10% had preclinical Alzheimer's disease, 10.4% had prodromal Alzheimer's disease and 9.8% had Alzheimer's disease dementia. Furthermore, among those 70 years of age or older, ADNCs were present in 60% of people with dementia, in 32.6% of those with mild cognitive impairment and in 23.5% of the cognitively unimpaired group. Our findings suggest a higher prevalence of Alzheimer's disease dementia in older individuals and a lower prevalence of preclinical Alzheimer's disease in younger groups than previously estimated[2].

Brukt data fra Helseundersøkelsen i Trøndelag (HUNT)

- Målte p-tau217 hos 11.486 personer over 70 år
- Beregnet hvor mange i hver aldersgruppe som hadde amyloide forandringer i hjernen
- Beregnet hvor mange over 70 år som hadde indikasjon for å bruke sykdoms-modifiserende behandling (DMT) = lecanemab
  - Mild kognitiv svikt eller mild demens
  - Amyloid i hjernen
  - Ikke homozygot ApoE4
  - Ikke tidligere hjerneslag, hjernedrypp (TIA) eller blødningsforstyrrelser

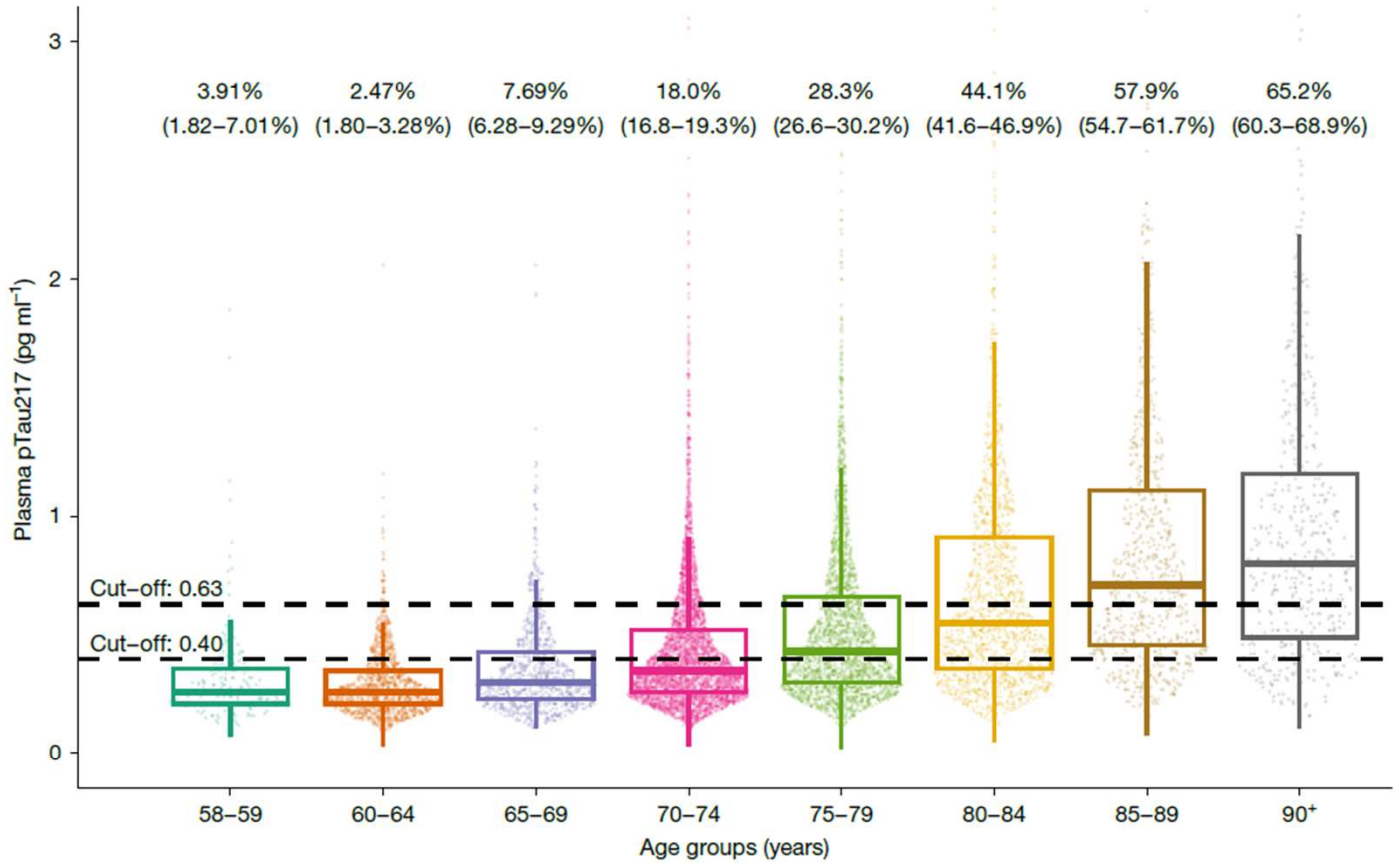


Fig. 1 | Plasma pTau217 concentrations in different age groups. Individual dots represent plasma pTau217 concentrations (n = 2,537 participants from HUNT3 and n = 8,949 participants from HUNT4 70+). Percentages (95% confidence interval) are estimates of how many in each age group have AD neuropathology, defined by plasma pTau217 concentration of 0.63 pg ml^{-1} or more. The lower cut-off of 0.40 pg ml^{-1} is also shown. The horizontal line in each box represents the median, and bottom and top edges delineate the second and third quartiles. The bottom whisker represents the first quartile, and the top whisker denotes the fourth quartile. Concentrations above 3 pg ml^{-1} are not shown.

Aarsland, Nature, 2025

AFS FORSKNINGSSENTER

Sykehuset Innlandet HF

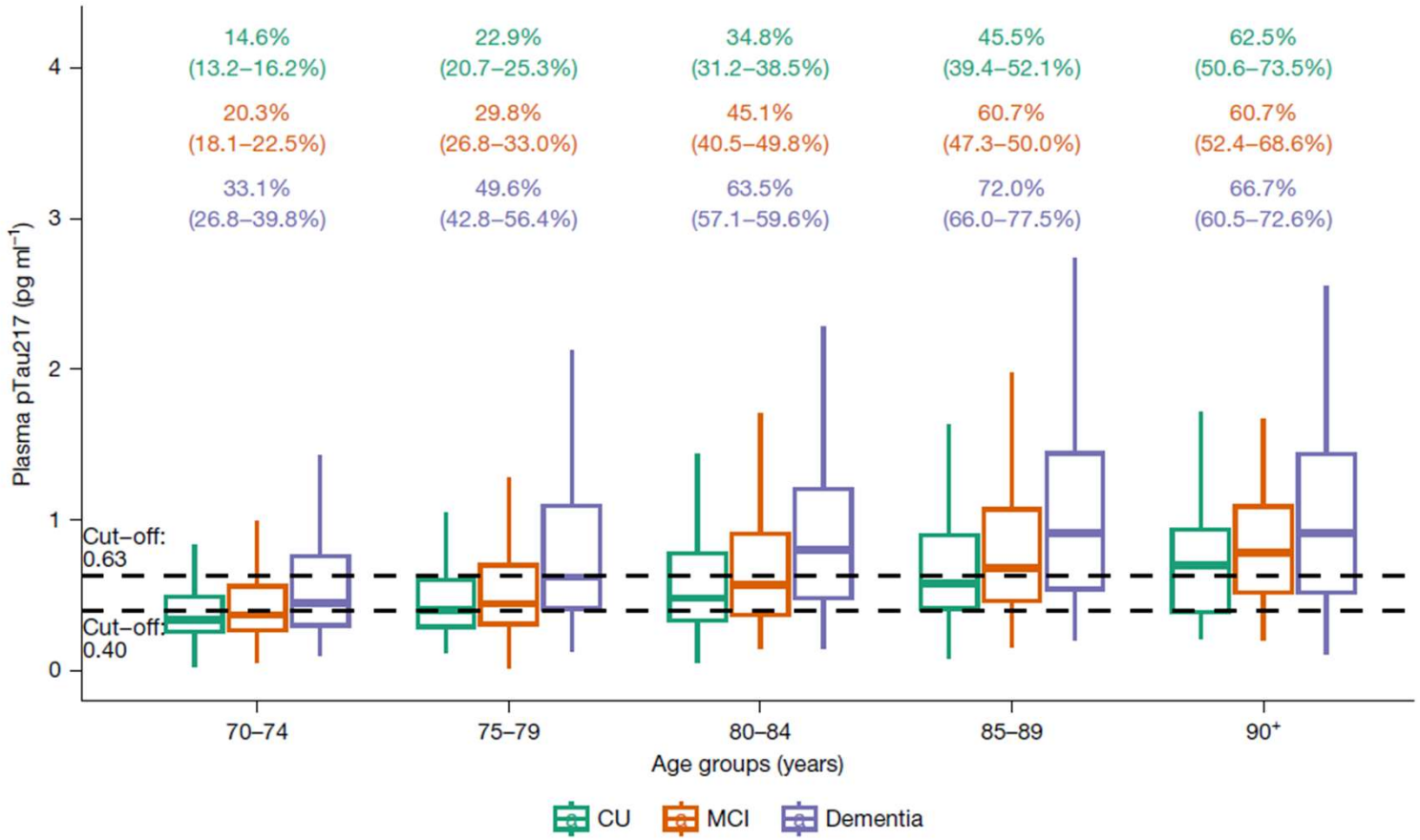


Fig. 2 | Plasma pTau217 concentrations in people 70 years of age or older who are cognitively unimpaired, have MCI or have dementia. Percentages (with 95% confidence intervals; colour-coded to match the box plots) are estimates of how many in each cognitive group have AD neuropathology, defined by plasma pTau217 concentration ≥ 0.63 pg ml^{-1}. The lower cut-off of 0.40 pg ml^{-1} is also shown. n = 8,949 participants from HUNT4 70+. The horizontal line in each box represents the median, and top and bottom edges delineate the second and third quartiles. The bottom whisker represents the first quartile, and the top whisker denotes the fourth quartile. CU, cognitively unimpaired.

Aarsland, Nature, 2025

AFS FORSKNINGSSENTER

Sykehuset Innlandet HF

Flere resultater, Aarsland et al. Nature 2025

- 11,1 % av alle over 70 år har indikasjon for å starte med DMT
  - 77.700 personer i år, 150.000 personer i 2060
  - Vil koste 78 milliarder per år

Article

A natural experiment on the effect of herpes zoster vaccination on dementia

https://doi.org/10.1038/s41586-025-08800-x

Received: 4 November 2023

Accepted: 18 February 2025

Published online: 2 April 2025

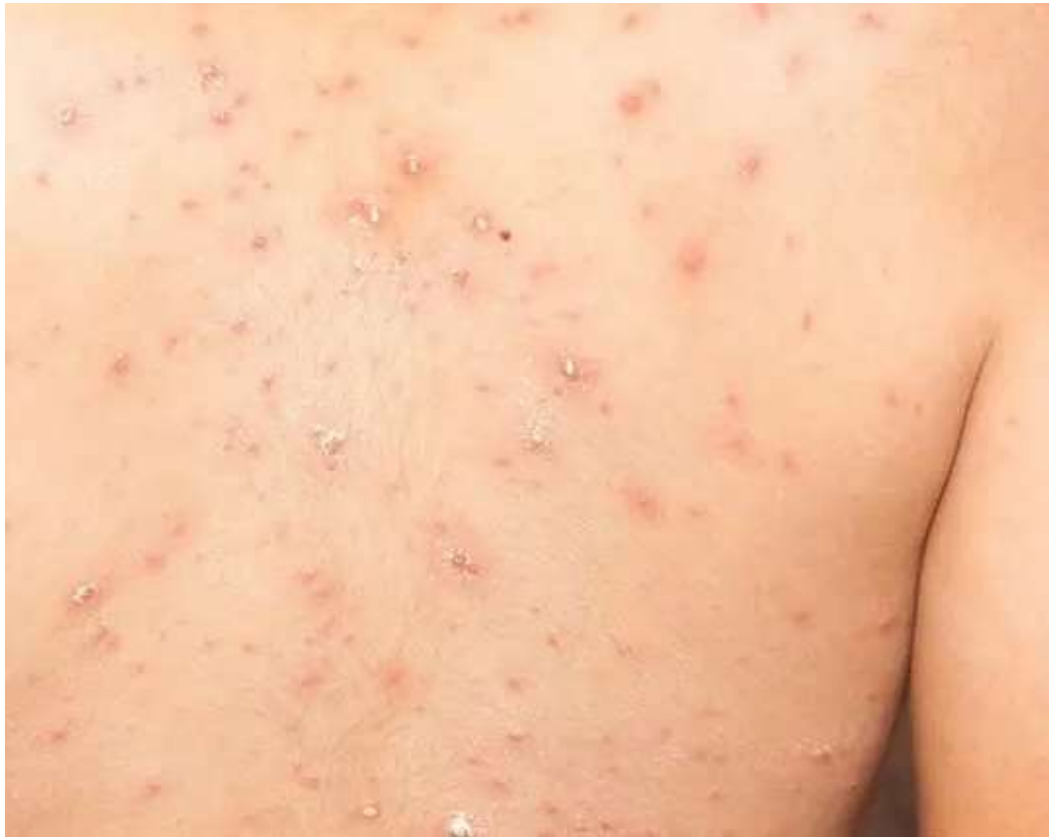
Open access

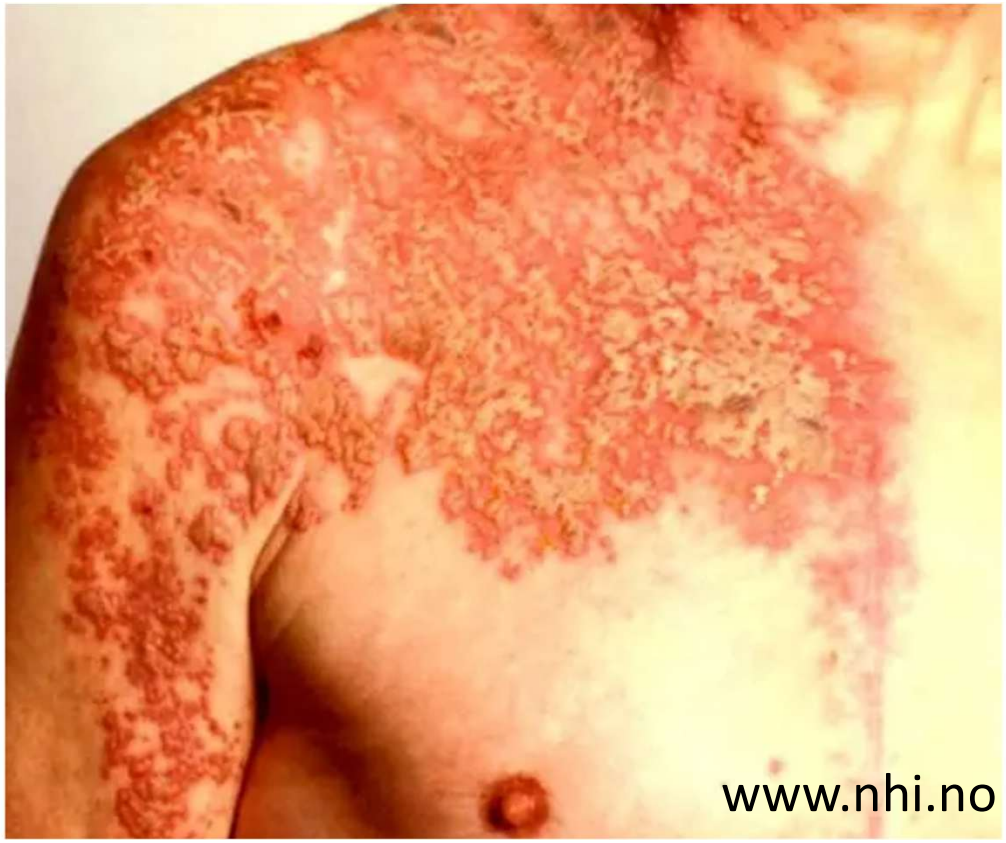
Check for updates

Markus Eyting[1,2,3,9], Min Xie[1,4,9], Felix Michalik[1,4], Simon Heß[5], Seunghun Chung[1] & Pascal Geldsetzer[1,6,7,8]

Neurotropic herpesviruses may be implicated in the development of dementia[1–5]. Moreover, vaccines may have important off-target immunological effects[6–9]. Here we aim to determine the effect of live-attenuated herpes zoster vaccination on the occurrence of dementia diagnoses. To provide causal as opposed to correlational evidence, we take advantage of the fact that, in Wales, eligibility for the zoster vaccine was determined on the basis of an individual's exact date of birth. Those born before 2 September 1933 were ineligible and remained ineligible for life, whereas those born on or after 2 September 1933 were eligible for at least 1 year to receive the vaccine. Using large-scale electronic health record data, we first show that the percentage of adults who received the vaccine increased from 0.01% among patients who were merely 1 week too old to be eligible, to 47.2% among those who were just 1 week younger. Apart from this large difference in the probability of ever receiving the zoster vaccine, individuals born just 1 week before 2 September 1933 are unlikely to differ systematically from those born 1 week later. Using these comparison groups in a regression discontinuity design, we show that receiving the zoster vaccine reduced the probability of a new dementia diagnosis over a follow-up period of 7 years by 3.5 percentage points (95% confidence interval (CI) = 0.6–7.1, $P = 0.019$), corresponding to a 20.0% (95% CI = 6.5–33.4) relative reduction. This protective effect was stronger among women than men. We successfully confirm our findings in a different population (England and Wales's combined population), with a different type of data (death certificates) and using an outcome (deaths with dementia as primary cause) that is closely related to dementia, but less reliant on a timely diagnosis of dementia by the healthcare system[10]. Through the use of a unique natural experiment, this study provides evidence of a dementia-preventing or dementia-delaying effect from zoster vaccination that is less vulnerable to confounding and bias than the existing associational evidence.

Herper Zoster infeksjon





Hva skjedde i september 1933?

- Mellomkrigstid
- John Støa gikk fra Trondheim til Oslo på 4 dager, 21 timer og 3min

Bilde: Trondheim Byarkiv

Født før og etter 2. sept 1933 i Wales

- 1. september 2013 begynte Wales med vaksinasjon mot Herpes Zoster (helvetesild)
- Født 1. sept 1933 eller før => Ikke vaksine
- Født 2. sept. 1933 eller senere => vaksine
- Eyting og kollegaer undersøkte sammenhengen mellom vaksine og senere demens (alle typer demens)
  - Data fra primær og sekundærhelsetjenesten i Wales

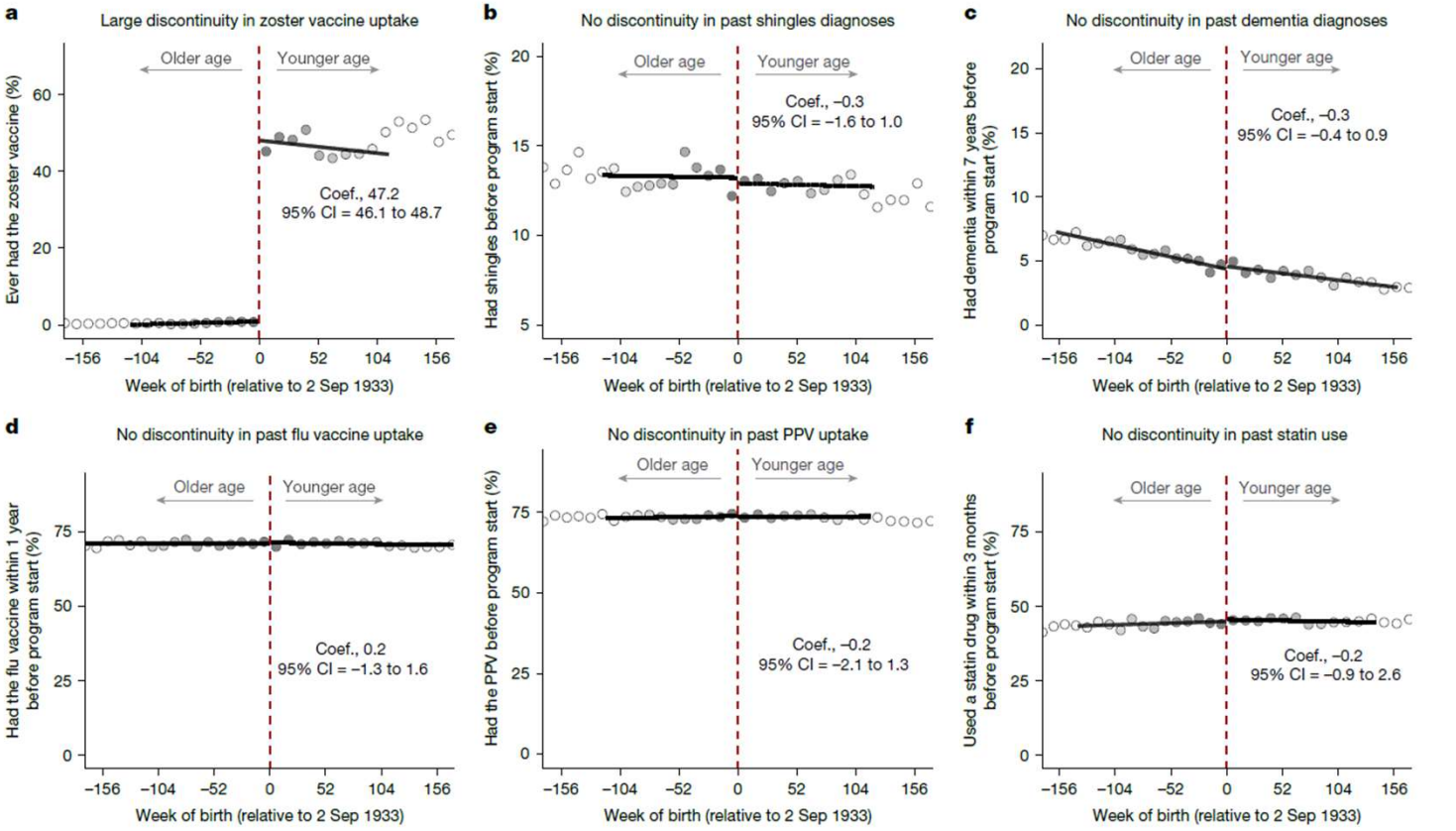


Fig. 1 | A large jump in zoster vaccine receipt at the date-of-birth eligibility threshold. a–f, The date-of-birth eligibility cut-off led to a large discontinuity in zoster vaccine receipt (**a**), but there is baseline exchangeability across the cut-off for uptake of other preventive interventions (flu vaccine (**d**), pneumococcal polysaccharide vaccine (PPV) (**e**) and statin medications (**f**)) as well as past shingles (**b**) and dementia (**c**) diagnoses. The data source for this analysis was the SAIL database for Wales. All analyses were run on the same sample as those for the effect of the zoster vaccine on dementia occurrence. The exception is **c**, for which we did not exclude individuals with a diagnosis of dementia before the start of the zoster vaccine program. The grey dots show the mean value for each 10-week increment in week of birth. The grey shading of the dots is proportionate to the weight that observations from this 10-week increment received in the analysis.

Eyting, Nature 2025

AFS FORSKNINGSSENTER

Sykehuset Innlandet HF

Resultater

- 35.307 av 282.541 delta-kere fikk demens

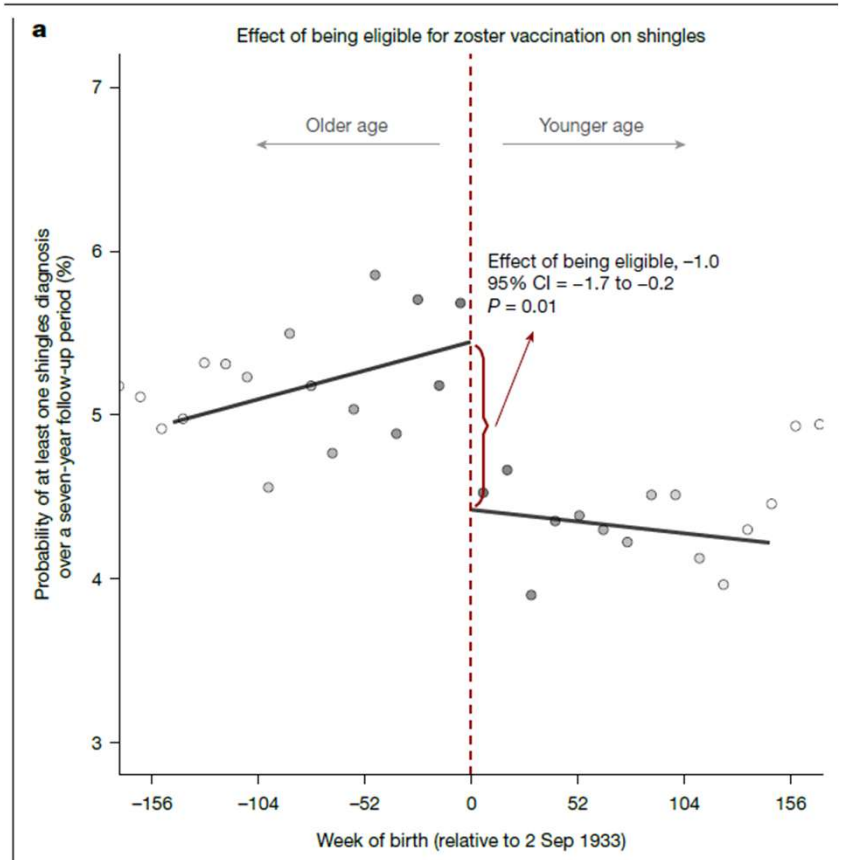


Fig. 2 | The effect of the zoster vaccine on shingles diagnoses. a–c, Effect estimates of being eligible for (**a**), and having received (across different follow-up periods (**b**) and across different grace periods (**c**)), the zoster vaccine on the probability of having at least one shingles diagnosis during the follow-up period. For **a**, the MSE-optimal bandwidth is 145.7 weeks (95,227 adults). The grey dots show the mean value for each 10-week increment in week of birth. The grey shading of the dots is proportionate to the weight that observations from

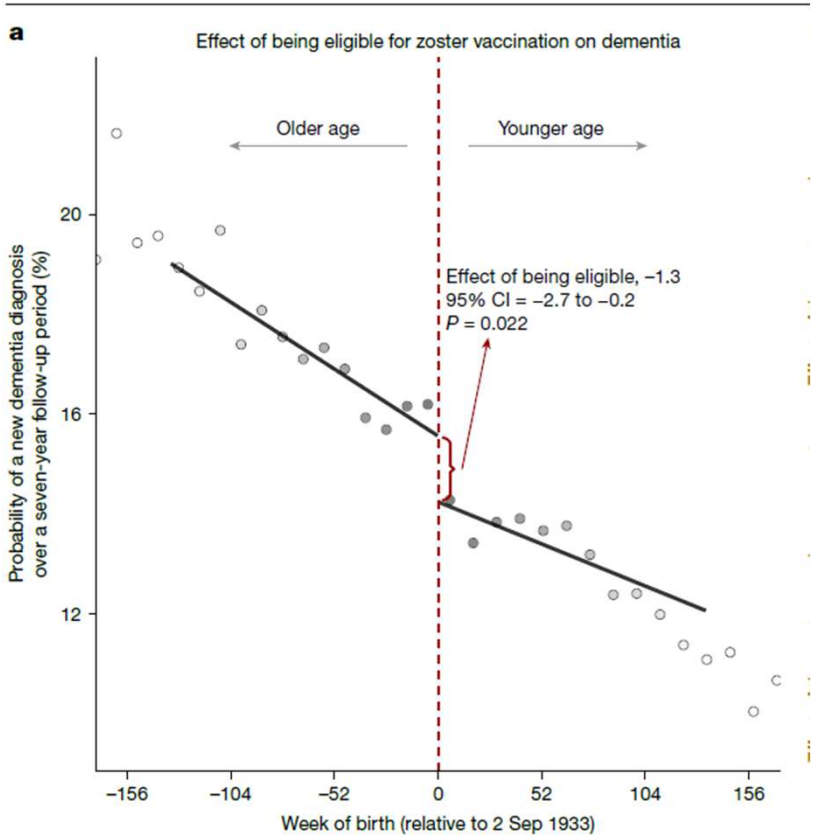


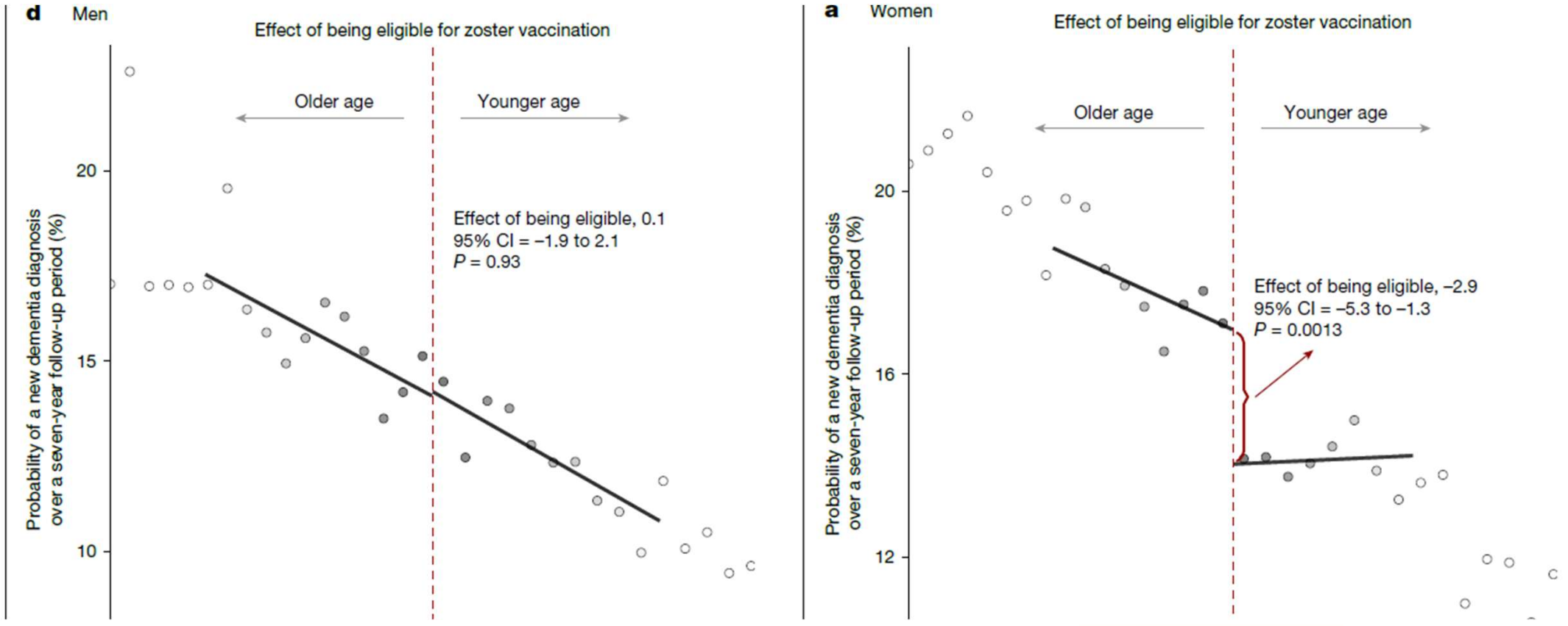
Fig. 3 | The effect of the zoster vaccine on new diagnoses of dementia. **a–c**, Effect estimates of being eligible for (**a**), and having received (across different follow-up periods (**b**) and across different grace periods (**c**)), the zoster vaccine on new diagnoses of dementia. For **a**, the MSE-optimal bandwidth is 134.4 weeks (83,167 adults). The grey dots show the mean value for each 10-week increment in week of birth. The grey shading of the dots is proportionate to the weight that observations from this 10-week increment received in the

Eyting, Nature 2025

AFS FORSKNINGSSENTER

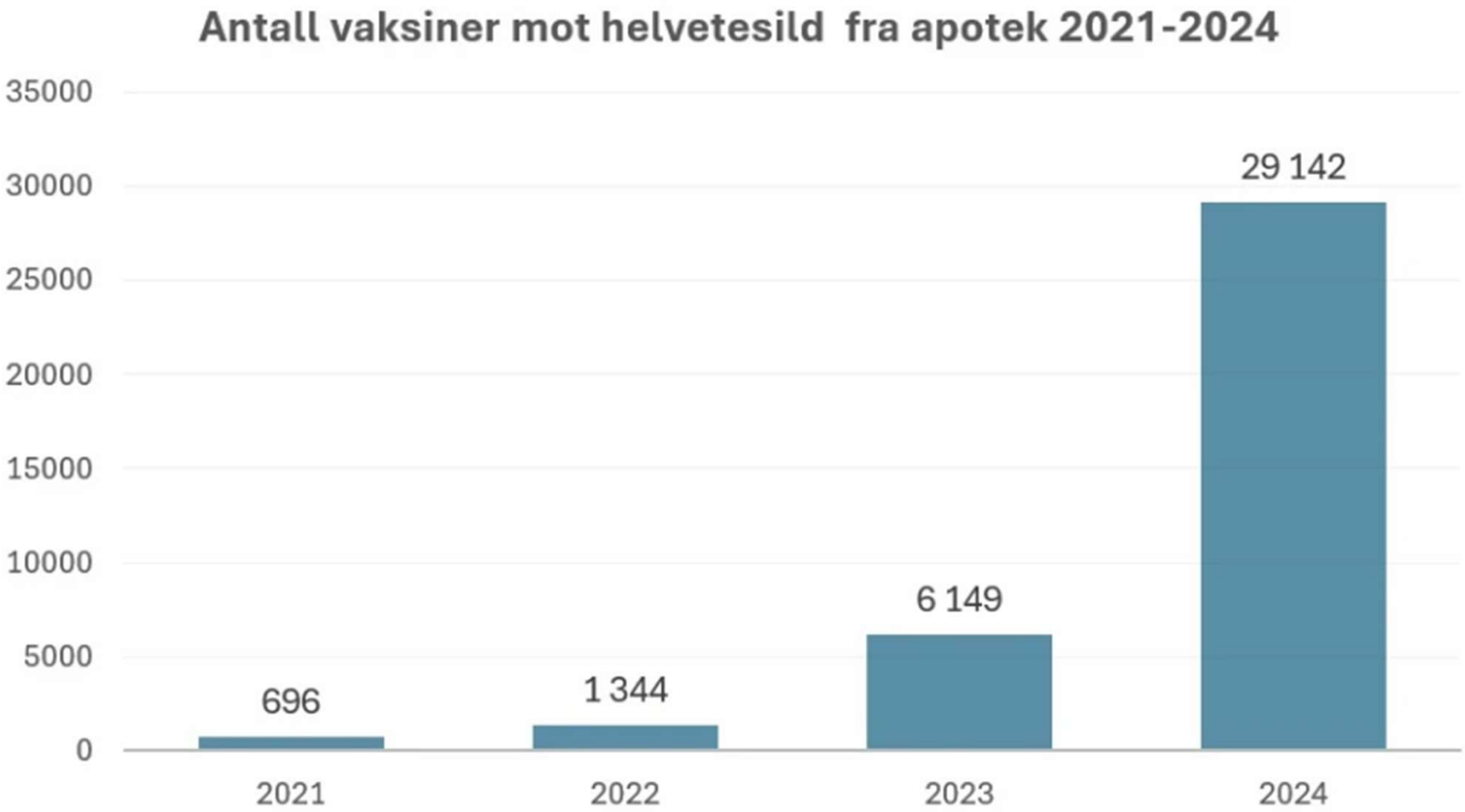
Sykehuset Innlandet HF

Forskjell mellom menn og kvinner



AFS FORSKNINGSSENTER

Sykehuset Innlandet HF



www.apotek.no

AFS FORSKNINGSSENTER

Sykehuset Innlandet HF

www.AFS-SI.no Epost: sverre.bergh@sykehuset-innlandet.no

AFS FORSKNINGSSENTER Sykehuset Innlandet HF