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RESEARCH ARTICLE

Sex differences in treatment effects of lecanemab and donanemab: A Bayesian reanalysis of CLARITY-AD and TRAILBLAZER-ALZ2

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Abstract

INTRODUCTION: This study investigated evidence for or against a difference in treatment effect between women and men for lecanemab and donanemab.

METHODS: Data were derived from supplementary analyses of the regulatory studies CLARITY-AD (lecanemab) and TRAILBLAZER-ALZ2 (donanemab). Bayes factor functions were used to analyze treatment effects on Clinical Dementia Rating Sum of Boxes (CDR-SB) scores.

RESULTS: We found moderate evidence of a lower treatment effect in women than in men for lecanemab (maximum Bayes factor = 5.97), suggesting that the presence of an effect was almost six times more likely than the absence of an effect. For donanemab, there was evidence against a treatment effect difference between women and men. There was evidence of a treatment effect difference between lecanemab and donanemab (maximum Bayes factor = 8.47) in women, but not in men.

DISCUSSION: A better understanding of sex differences in treatment efficacy and their causes is urgently needed.

KEYWORDS
anti-amyloid antibodies, personalized treatment, prespecified secondary analysis, subgroups, treatment efficacy

Highlights

- Lecanemab was six times more likely to be ineffective than effective in women.
- There was no evidence of a difference between the sexes in the effect of donanemab.
- Lecanemab and donenamb differed in treatment efficacy in women but not in men.
- Future trials should include sufficient power for sex related interaction effects.

1 | INTRODUCTION

The approval of anti-amyloid antibodies for the treatment of prodromal and mild stages of Alzheimer's disease (AD) has led to a paradigm shift in the treatment of AD. A key challenge for the field is to identify which subgroups of patients will or will not benefit from these new treatments. Secondary analyses of phase 3 anti-amyloid antibody trials of the new-generation antibodies gantenerumab,[1] aducanumab,[2] lecanemab,[3] and donanemab[4] have suggested possible sex differences in treatment efficacy. Surprisingly, these findings have so far received little attention in the scientific and public debate on the risk–benefit ratio of the new treatments.

A simulation study based on data from the Alzheimer's Disease Neuroimaging Initiative (ADNI) cohort suggested that women with prodromal to mild AD dementia should have significantly less pronounced rates of cognitive decline compared to men if the observed differences in treatment effects between female and male participants in the lecanemab Clarity study were due to different trajectories of cognitive decline and not to actual differences in treatment efficacy.[5] In contrast, however, the ADNI data indicated more, rather than less pronounced rates of cognitive decline for women versus men.[5] These and similar approaches are important to overcome the current limitations of a discussion that often stops at the notion that the trials were not sufficiently powered to detect treatment effects only in the subgroups of female or male participants. This limitation has taken on new urgency in light of the controversy surrounding the United States Food and Drug Administration's (FDA's) accelerated approval of aducanumab, which highlighted gaps between biomarker outcomes and clinically meaningful benefit—particularly among diverse patient subgroups. Interestingly, in contrast to not significant treatment effects in female participants for gantenerumab,[1] aducanumab,[2] and lecanemab,[3] donanemab showed significant treatment effects on the primary endpoint AD Composite Score (ADCOMS) and the prespecified secondary endpoint Clinical Dementia Rating Sum of Boxes (CDR-SB) in female participants.[4] Many meta-analyses have been conducted on treatment effects of anti-amyloid antibodies but evidence on sex differences in treatment efficacy is still scarce.

We re-analyzed the evidence for or against a treatment effect difference between women and men and between lecanemab and donanemab by sex using Bayes factor functions,[6] a recent extension of the Bayes factor.[7] In a Bayesian framework, Bayes factors directly quantify evidence for or against an effect, specifically "how much the prior odds [for or against an effect] change, given the data" ([8], page 268). Here, we used the Bayes factor functions for three reasons (see also Methodological notes on Bayes factor functions in the Supplement):

First, we aimed for a Bayesian framework which allowed us to determine both evidence for and against a difference. In the Bayesian framework, the effect size is treated as an unknown random variable with an underlying probability distribution. This is much closer to common understanding of an estimate of the effect size in a clinical trial or clinical treatment scenario than the assumption underlying the frequentist analysis that the effect size has an unknown but fixed value.

Secondly, we used Bayes factor functions to compensate for the sensitivity of the Bayes factor to the choice of the parameter priors.[9] Bayes factor functions address this limitation of prior choice dependence,[10] and introduce a continuous function approach for Bayes factors.[6]

Thirdly, we wanted to determine evidence for or against an effect across the prior distributions of a whole range of effect sizes. This enhances the robustness of the Bayesian approach by incorporating prior sensitivity directly into the model comparison process, mitigating problems associated with arbitrary prior choices.

Specifically, for the data of the lecanemab and donanemab treatment trials, Bayes factor functions allowed us to directly quantify the evidence for or against an effect across all possible effect sizes. This differs from the frequentist *p*-value[11] that provides a binary decision to reject or not reject the null hypothesis of no effect.

The results of our analysis address an important knowledge gap regarding sex-specific differences in the efficacy of newly approved anti-amyloid antibodies.

RESEARCH IN CONTEXT

1. **Systematic review:** Few studies have examined the differential efficacy of anti-amyloid antibodies in Alzheimer's disease (AD) between women and men, leaving an important gap in our knowledge.
2. **Interpretation:** The results suggest moderate evidence against a clinical benefit of lecenamab in women and evidence for a difference in treatment effects between lecanemab and donanemab in women but not in men.
3. **Future directions:** The results underscore the urgent need for further research into sex differences in treatment responses to amyloid-targeting therapies. This has direct implications for patient counseling and individualized treatment decisions. Future studies should prioritize the pre-specification of sex-based subgroup analyses and ensure sufficient statistical power to detect interaction effects. Moreover, mechanistic studies are needed to explore potential biological or pharmacokinetic reasons for differential treatment effects between women and men.

2 | METHODS

2.1 | Data source

We analyzed data from the supporting material of the Clarity[3] and the TRAILBLAZER-ALZ2[4] trials. Specifically we determined the mean

differences, their 95% confidence intervals and the number of cases in the placebo and treatment groups for the effect of donanemab and lecanemab, respectively, on the CDR-SB and for the difference in effect between the two antibodies in female and male participants. The data were obtained from the supporting sections of the TRAILBLAZER-ALZ2[4] and Clarity[3] publications, specifically from page 35, "eFigure 9D" of "eMethods and eResults" of TRAILBLAZER-ALZ2,[4] and page 18, "Figure S1b", of the "Appendix" of Clarity.[3]

For TRAILBLAZER-ALZ2, the 95% confidence interval for the percentage mean differences was reported, from which we could directly determine the 95% confidence interval for the CDR-SB score points. For Clarity-AD, the corresponding figure was transferred to the graphics program Paint (version 1809, Microsoft Corporation) and measured using its rulers and gridlines function. The length of the error bars was measured independently by two raters who agreed on the results.

We calculated the standard error (SE) of the effect from the 95% confidence intervals (CI) as:

SE = |upper threshold 95% CI—lower threshold 95%CI|/2* t(df, $p < 0.025$),[12] with t representing the Student's t distribution with df (degrees of freedom) equal to the total number of cases. With df > 120, as was the case in our analysis with > 260 cases per comparison, t(df, $p < 0.025$) approximates z ($p < 0.025$), the standard normal distribution (see Section 6.5.2.2 of the Cochrane Training Manual, https://training.cochrane.org/handbook/current/chapter-06#section-6-5-2-2).

The standard error of the mean difference of the effects between placebo and treatment for each antibody in females versus male participants and between donanemab and lecanemab in the female and male participants, respectively, was calculated as:

$$SE_{diff} = \sqrt{SE_1^2 + SE_2^2},$$

that is, the square root of the sum of the squared standard errors of the treatment effects for lecanemab versus placebo in females and males, donanemab versus placebo in females and males, donanemab versus lecanmeab in females, and donanemab versus lecanemab in males.

The mean effect estimates T were calculated as the mean differences in effects between sex groups and between antibodies within sex groups, respectively, divided by the corresponding standard error, $T = MD/SE_{diff}$.

2.2 | Statistical analysis

We conducted a Bayes factor functions analysis following Johnson et a.[6] Bayes factor functions depend on a single non-centrally parameter that distinguishes a non-central distribution, which has a mean value other than zero, from its central counterpart, which has a mean value equal to zero. It can be expressed as a function of standardized effects, and plots of Bayes factor functions versus effect size provide summaries of hypotheses. Here we used the T-statistic as the non-centrally parameter, and we calculated the corresponding Bayes factors as functions of effect sizes of treatment effect differences, expressed as Cohen's D,[13] ranging between an effect size of 0 (no effect) to 1 (strong effect). The calculation was conducted using an adaptation of the code provided in the Bayes factor function package BFF in the R statistic software.

3 | RESULTS

Effect size estimates for the comparisons are shown in the Table S1 in the Supplement. Bayes factor function analysis showed moderate evidence of a treatment effect difference between women and men for lecanemab with a peak at a small effect size (maximum Bayes factor = 5.97), see Figure 1), with a smaller treatment effect in women, suggesting that the presence of a difference was almost six times more likely than the absence of a difference. For donanemab, there was evidence against a treatment effect difference between women and men, see Figure 2. The evidence was inconclusive for very small to small effect sizes and at least moderately in favor of no difference with higher effect sizes. We found moderate evidence for a treatment effect difference in women between lecanemab and donanemab with a peak at a small to medium effect size (maximum Bayes factor = 8.47, Figure 3), with smaller effects for lecanemab, and evidence for no difference in the treatment effect between lecanemab and donanemab in men, see Figure 4. The evidence was inconclusive for very small to small effect sizes and at least moderately in favor of no difference with higher effect sizes.

4 | DISCUSSION

The Bayes factor functions suggested a true treatment effect difference on the CDR sum of boxes for lecanemab between women and men, in favor of male participants, and between lecanemab and donanemab in women. In contrast, we found evidence against a treatment effect difference between women and men for donanemab and against a treatment effect difference between lecanemab and donanemab in men. Of note, evidence was in favor of a small to moderate effect size of the treatment effect difference of lecanemab in women compared with men and a small to medium effect size of the treatment effect difference between donanemab and lecanemab in women. These findings emerge against a backdrop of heightened scrutiny regarding amyloid-directed treatments and their clinical impact. The observed sex differences underscore the necessity of stratified analyses to guide individual-level treatment decisions, as called for in ongoing evaluations of immunotherapies.

These data confirm and extend the results of a previous simulation analysis[5] that suggested that the difference in treatment effect between women and men for lecanemab were not due to chance but may reflect a real difference between sexes with a small to moderate effect size. Our results are important as they suggest that despite the insufficient power in a frequentist framework evidence was substantially in favor of a true difference.

The reasons for these differences remain unclear. Obviously, our analysis cannot resolve the cause of effect, but can only indicate its

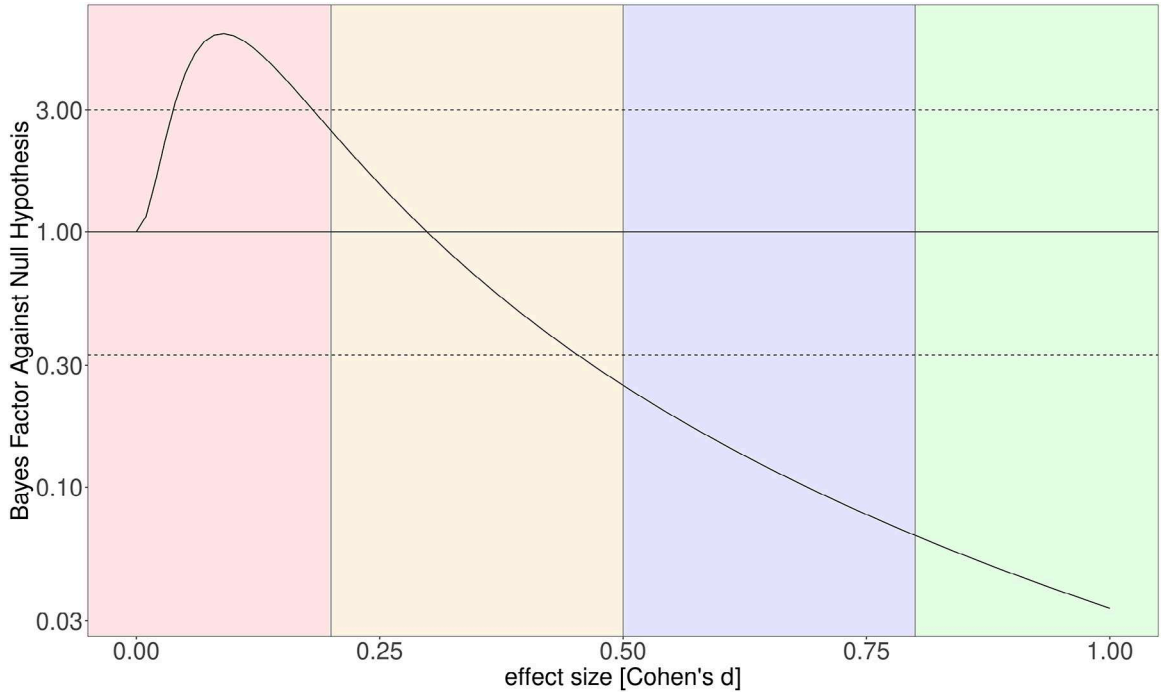

FIGURE 1 Evidence for a difference in lecanemab efficacy between female and male patients. This plot illustrates the strength of statistical evidence in favor of the alternative hypothesis (H_1) that there is a sex effect on treatment efficacy versus the null hypothesis (H_0) that there is no sex effect on treatment efficacy using a Bayes factor function. Unlike a single *p*-value or point estimate, the Bayes factor function provides a continuous measure of evidence across a range of hypothetical effect sizes.
The x-axis represents a continuum of plausible population effect sizes — not observed values, but possible differences between groups that could be true in the population. For each effect size shown on the x-axis, the corresponding Bayes factor (on the y-axis) quantifies how consistent the observed data are with that hypothetical effect size being true, compared to the null hypothesis that the effect size is 0. The possible treatment effect differences are expressed as Cohen's d effect size metric. The vertical lines and colors indicate ranges of the effect size (Cohen's d) between 0 to 0.2 (very small to small), 0.2 to 0.5 (moderate to medium), 0.5 to 0.8 (medium to large), and 0.8 to 1.0 (large to very large). The x-axis reflects "what if the true effect size were X?" and the plot shows how well the data support that scenario relative to H_0.
The y-axis shows the Bayes factor for each effect size, which tells us how much more likely the observed data are under the alternative hypothesis (H_1) than under the null hypothesis (H_0), given that particular effect size. The horizontal upper dashed line indicates a Bayes factor of 3, indicating moderate evidence for an effect, and the lower dashed line indicates a Bayes factor of 1/3, indicating moderate evidence against an effect. The y-axis is log scaled. There was evidence of a difference in the effect of lecanemab treatment on CDR-SB between female and male patients with a small effect size. CDR-SB, Clinical Dementia Rating Sum of Boxes.

existence. It still could be a chance finding; however, our data suggest that a chance finding is unlikely. The difference between lecanemab and donanemab could indicate that stratification according to brain tau status may play a role. The hypothesis would be that women with the same degree of cognitive impairment may harbor more pronounced brain pathology suggesting a higher cognitive resilience in women compared with men. Thus, women had higher verbal memory performance than men at the same level of cerebral glucose consumption measured by fluorodeoxyglucose-positron emission tomography (FDG-PET) in the mild cognitive impairment (MCI), but not the dementia stage of AD,[14] and at the same level of tau pathology measured using tau sensitive PET in vivo as well as Braak stages post mortem.[15] A meta-analysis suggested that cognitively unimpaired women with increased amyloid showed faster tau accumulation in longitudinal PET examinations than amyloid positive cognitively unimpaired men.[16] These data would suggest that women have more brain pathology, including tau pathology, than men despite a similar level of cognitive impairment. At the same time, people with a higher degree of brain pathology would be expected to benefit less from amyloid-targeted treatment. Unlike the Clarity-AD trial, TRAILBLAZER-ALZ2 controlled for tau pathology levels using tau PET. One could speculate that this may at least partially explain why treatment effects of donanemab were more pronounced than lecanemab effects in women. The donanemab groups were stratified according to tau status, which may have eliminated at least part of the difference in brain pathology between sexes. It is important to note, however, that the effects of sex on cognitive reserve and rates of cognitive decline are complex and the data are equivocal.[17] An alternative explanation would be that differences in hormone status, such as absolute oestrogen deprivation in postmenopausal women,[18,19] drive differences in treatment effect. Gendered social roles and opportunities, such as lifestyle and educational and occupational attainment, may also influence the risk of cognitive decline and response to treatment.

Several limitations should be considered. First, our analysis did not explore potential interactions between sex and apolipoprotein E

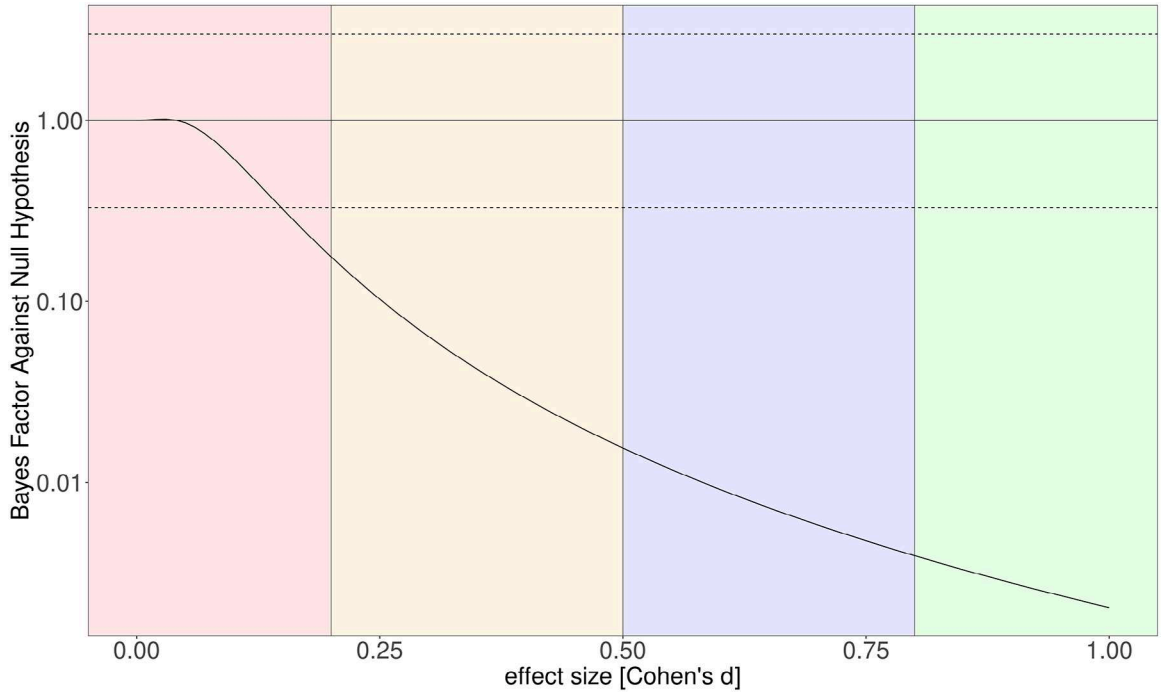

FIGURE 2 Evidence for no difference in donanemab efficacy between female and male patients. For a general description see legend of Figure 1. There was evidence of no difference in the treatment effect of donanemab on CDR-SB between female and male patients. CDR-SB, Clinical Dementia Rating Sum of Boxes.

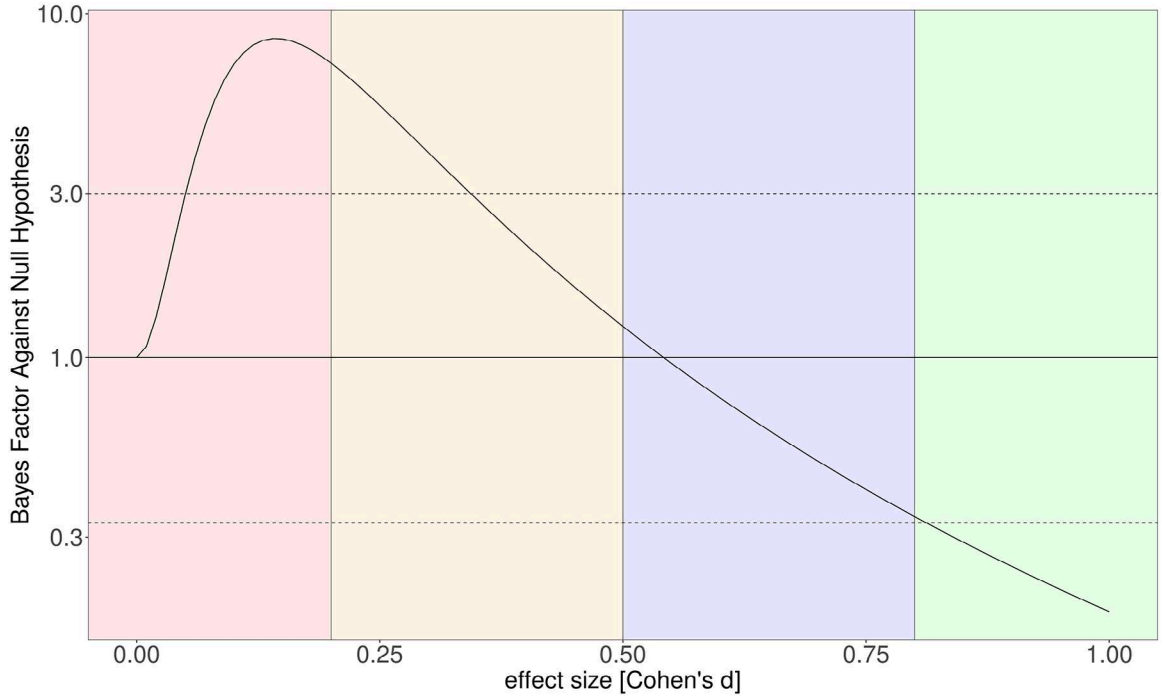

FIGURE 3 Evidence for a difference in lecanemab and donanemab efficacy in female patients. For a general description see legend of Figure 1. There was evidence of a difference between the effect of lecanemab and donanemab treatment on CDR-SB in female patients with a small to medium effect size. CDR-SB, Clinical Dementia Rating Sum of Boxes.

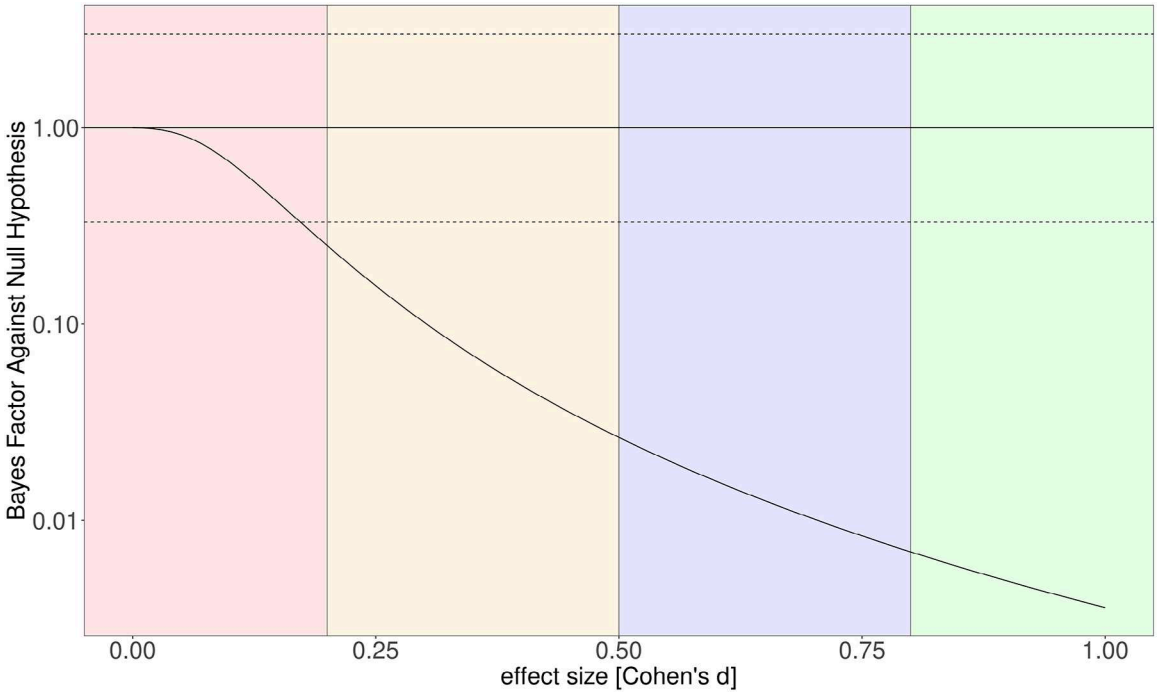

FIGURE 4 Evidence for no difference in lecanemab and donanemab efficacy in male patients. For a general description see legend of Figure 1. There was evidence of no difference between the effect of lecanemab and donanemab treatment on CDR-SB in male patients. CDR-SB, Clinical Dementia Rating Sum of Boxes.

(APOE) ε4 status, cognitive reserve, or other moderating variables that may shape treatment efficacy. Second, we lacked neuroimaging or fluid biomarker data to investigate neurobiological correlates of sex-related treatment response. Third, the generalizability of these findings is constrained by trial populations that may not represent the full diversity of real-world patients, particularly with respect to race, socioeconomic status, and healthcare access. These considerations align with recent calls for more inclusive and mechanistically grounded research to validate clinical utility across subgroups.

Further clarification of these issues is one of the most urgent tasks of clinical research in the field of AD to ensure that future treatment decisions take into account the differences between the sexes in terms of risks and benefits, as proposed previously.[20,21] The current situation, where it is not clear whether and why lecanemab and donanemab have different effects in women and men, makes it difficult for doctors and patients to decide for or against treatment on an individual basis.

To improve this situation, we propose a multi-pronged strategy. First, in a hypothesis driven approach in the original data of TRAILBLAZER-ALZ2 and Clarity one could study if differences in sex effects are partly explained by a moderating effect of tau to test the hypothesis if different resilience to tau may account for some of the sex effects. Obviously, such an analysis cannot be done based on secondary data. Future individual participant data meta-analysis or collaboration with trial sponsors is needed to formally test this hypothesis. This is also important as the current FDA labels and appropriate use recommendations (AUR)[22] for lecanemab and donanemab do not include tau measurement for reaching a treatment decision. If analyses, however, suggest that tau levels moderate treatment efficacy differentially in women and men it would be an argument to consider measuring tau for treatment decision. Second, future studies should incorporate sex-stratified randomization and endpoint powering to ensure subgroup effects are not underpowered or obscured by pooled analysis. Second, companion diagnostic tools[23] need to be developed that can integrate genetic, vascular, and inflammatory biomarkers-such as APOE status,[24] YKL-40, vascular endothelial growth factor (VEGF), and triggering receptor expressed on myeloid cells 2 (TREM2).[25] The potential role of these biomarkers by sex needs to be further elucidated to allow individualized prediction of safety and efficacy prior to initiation of therapy.

Third, a clearer understanding of molecular heterogeneity—at the level of gene expression and protein signaling—will be critical. Integrating proteomics and transcriptomics into early-phase trials can illuminate how women and men differentially activate neuronal stress pathways, microglial responses, and synaptic resilience cascades. Multi-omics can also reveal therapeutic targets that are either sex-specific or disproportionately modulated in one sex due to hormonal or epigenetic context.[26,27] Even preceding clinical trials, considering sex effects in preclinical studies, albeit challenging and costly, would inform whether or not a difference by biological sex should be expected in human studies and may open avenues to study potentially underlying mechanisms.

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CONFLICT OF INTEREST STATEMENT

S.T. was member of Advisory Boards of Eisai, Lilly, GE Healthcare, and Biogen. He was member of the Data Safety and Monitoring Board of the ENVISION study (Biogen). Y.T. and A.K. report no conflicts of interest. Author disclosures are available in the supporting information.

CONSENT STATEMENT

The study presents a reanalysis of published data, therefore, consent of human subjects was not necessary.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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