Effects of Finerenone on Natriuretic Peptide Levels in Heart Failure with Mildly Reduced or Preserved Ejection Fraction: The FINEARTS-HF Trial

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Running Title: Finerenone and Natriuretic Peptide Levels

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Structured Abstract

Background: N-terminal pro–B-type natriuretic peptide (NT-proBNP) concentrations serve as markers of prognosis and therapeutic response in patients with heart failure (HF). The effect of the non-steroidal MRA finerenone on NT-proBNP in patients with HF with mildly reduced or preserved ejection fraction (HFmrEF/HFpEF) is currently unknown.

Methods: The FINEARTS-HF trial randomized patients with HFmrEF/HFpEF and NT-proBNP ≥300 pg/ml (≥900pg/ml if atrial fibrillation) or BNP ≥100 pg/ml (≥300 pg/ml if atrial fibrillation) to finerenone vs placebo. Core laboratory NT-proBNP was measured at baseline, 3, and 12 months after randomization. We evaluated the association between log-transformed NT-proBNP and the primary outcome (cardiovascular death and total worsening HF events), whether baseline NT-proBNP modified the effect of finerenone on this outcome, and the effect of finerenone on NT-proBNP concentration.

Results: Baseline NT-proBNP was available in 5843 of 6001 patients analyzed (median 1041 [IQR 449,1946] pg/ml) and was strongly associated with risk of the primary outcome: adjusted rate ratio 1.44 per doubling in biomarker concentration (95% CI 1.37-1.51], P<0.001). Baseline NT-proBNP did not modify the benefit of finerenone on the primary outcome ($P_{interaction}$=0.92). Finerenone reduced NT-proBNP by 12.1% (95% CI 8.5-15.4%) at 3 months and 12.5% (95% CI 8.1-16.7%) at 12 months, compared to placebo.

Conclusions: In patients with HFmrEF/HFpEF, finerenone reduced NT-proBNP within months of initiation, and improved clinical outcomes regardless of baseline NT-proBNP concentration.

Key Words: finerenone; heart failure; mineralocorticoid receptor antagonists; natriuretic peptides

Clinical Trial Registration: ClinicalTrials.gov NCT04435626, EudraCT 2020-000306-29

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Background

N-terminal pro–B-type natriuretic peptide (NT-proBNP) concentrations in plasma are widely used for diagnosis and risk stratification in patients with heart failure (HF).[1] Changes in NT-proBNP in response to therapy have correlated closely with treatment effects on HF hospitalizations in previous clinical trials.[2] The non-steroidal mineralocorticoid receptor antagonist (MRA) finerenone reduced NT-proBNP concentrations by 18% at 4 months in patients with type 2 diabetes and chronic kidney disease.[3] Among patients with HF with mildly reduced or preserved ejection fraction (HFmrEF/HFpEF) in the FINEARTS-HF (FINerenone trial to investigate Efficacy and sAfety superioR to placebo in paTientS with Heart Failure) trial, finerenone reduced the primary outcome of total worsening HF events and cardiovascular death by 16%, but its effect on NT-proBNP in this population is not known.

Aims

We investigated the treatment effect of finerenone across baseline concentrations of NT-proBNP and on NT-proBNP concentrations over time in patients with HFmrEF/HFpEF in the FINEARTS-HF trial.

Methods

The FINEARTS-HF trial compared finerenone and placebo in 6001 patients with symptomatic HFmrEF/HFpEF (≥40%), structural heart disease, and recent diuretic use. Elevated natriuretic peptides at baseline were required for inclusion: for patients in sinus rhythm, NT-proBNP ≥300 pg/ml (BNP ≥100 pg/ml), measured within 30 days (in those without recent worsening HF event) or within 90 days (in those with recent worsening HF event). These thresholds were tripled for patients in atrial fibrillation on the screening electrocardiogram. Finerenone (or matching placebo) were planned to be titrated to

either 20mg or 40mg (based on baseline estimated glomerular filtration rate[eGFR]) at 1 month post-randomization. The design and results of the trial have been previously published. [4]

NT-proBNP was measured at baseline, 3 and 12 months after randomization using standard central lab chemiluminescence-based analyzers. We first evaluated the association between log-transformed baseline NT-proBNP and the primary endpoint (total worsening HF events and cardiovascular death) using semiparametric proportional rates models. The components of the primary endpoint, total worsening HF events alone and cardiovascular death, were evaluated using a semiparametric proportional rates model and a Cox proportional hazards model, respectively. Models were adjusted for 12 clinically relevant covariates: age, sex, region, systolic blood pressure, body mass index (BMI), eGFR, ejection fraction, history of myocardial infarction, stroke, or hospitalization for HF, and baseline diabetes mellitus or atrial fibrillation. We then tested whether finerenone's effect on the primary endpoint was modified by baseline NT-proBNP concentrations by interaction testing. Additionally, we evaluated the placebo-corrected effect of finerenone on NT-proBNP concentrations at 3 and 12 months using linear regression, adjusted for the baseline value. Statistical analyses were performed using Stata, version 16 (StataCorp, LLC; College Station, TX, USA); P-values of <0.05 were considered statistically significant.

Results

Among 5843 patients (97%) with available data, median baseline NT-proBNP was 1041 [interquartile range (IQR) 449, 1946] pg/ml. NT-proBNP was higher in participants with older age, lower BMI, lower eGFR, and (by design) atrial fibrillation (**Table 1**). Median NT-proBNP was 1714 (IQR 1152-2807) pg/ml for participants with atrial fibrillation on baseline electrocardiogram and 588 (IQR 313-1255) pg/ml for participants without atrial fibrillation. NT-proBNP was strongly and independently associated with risk of the primary outcome (adjusted rate ratio 1.44 per doubling [95% CI 1.37-1.51], $P<0.001$)

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and with each component alone: total worsening HF events (adjusted rate ratio 1.41 [95% CI 1.34-1.49], p<0.001) and cardiovascular death (adjusted hazard ratio 1.52 [95% CI 1.43-1.63], p<0.001]). Baseline NT-proBNP was furthermore associated with risk of all-cause death (adjusted hazard ratio 1.44 [95% CI 1.38-1.51, p<0.001]). Adding NT-proBNP concentration at 3 months to a regression model including baseline NT-proBNP and clinical covariates improved prediction of subsequent first worsening HF or CV death events (c-statistic 0.73 vs 0.71, p<0.001).

Baseline NT-proBNP did not significantly modify the beneficial effects of finerenone on relative risk of the primary outcome ($P_{interaction}$=0.92); finerenone reduced the risk of primary outcome events across the spectrum of baseline NT-proBNP concentrations (**Figure 1**). No significant treatment interaction was observed for key secondary outcomes including total worsening HF events ($p_{interaction}$=0.91), cardiovascular death ($p_{interaction}$=0.98), and all-cause death ($p_{interaction}$=0.47) (**Supplemental Figure 1**). For the primary outcome, NT-proBNP did not significantly modify the effect of finerenone in patients with atrial fibrillation ($p_{interaction}$=0.49) or without ($p_{interaction}$=0.62) atrial fibrillation (**Supplemental Figure 2**).

Finerenone reduced NT-proBNP by 12.1% (95% CI 8.5-15.4%) at 3 months in 5218 patients with available data and 12.5% (95% CI 8.1-16.7%) at 12 months in 4655 patients with available data, compared to placebo (**Figure 2**). The effect of finerenone on NT-proBNP concentrations at 3 months was not significantly modified by age, sex, race, atrial fibrillation, BMI, eGFR, or left ventricular ejection fraction.

Discussion

In this prespecified analysis of the FINEARTS-HF trial, finerenone reduced worsening HF events and cardiovascular death in patients with HFmrEF/HFpEF with any degree of natriuretic peptide elevation. This absence of treatment interaction differs from the TOPCAT trial with the steroidal MRA

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spironolactone in HFmrEF/HFpEF, which found less benefit in patients with higher natriuretic peptide concentrations.[5] Aligned with these clinical benefits, we found early and sustained reductions in NT-proBNP with finerenone. Among patients with HFmrEF/HFpEF, the 12% reduction in NT-proBNP with finerenone is greater than with empagliflozin (4%) and lower than sacubitril/valsartan (19%), acknowledging differences in trial inclusion criteria. The treatment effect on NT-proBNP in FINEARTS-HF was somewhat lower than in patients with chronic kidney disease and type 2 diabetes in the FIDELITY trials (18% [95% CI: 10-24%]),[3] though confidence intervals overlap. Spironolactone compared to placebo reduced NT-proBNP by 20% (95% CI 0-40%) in the TOPCAT trial, 14% (95% CI 1%-25%), and approximately 20-25% in HOMAGE,[6–8] though these studies enrolled different patient populations from FINEARTS-HF and had smaller sample size. In the TOPCAT trial, collection of follow-up natriuretic peptide data was not protocolized for all participants; data were available only in a limited biomarker substudy. Concordant effects on NT-proBNP and primary outcome events in FINEARTS-HF support NT-proBNP as a reliable surrogate outcome in HF clinical trials, consistent with a prior analysis of many HF randomized trials reporting treatment effects on both NT-proBNP and clinical outcomes.[2]

These results should be interpreted in the context of several limitations. The FINEARTS-HF trial required elevated natriuretic peptides for inclusion; thus findings may not generalize to patients with normal natriuretic peptide concentrations. NT-proBNP concentrations were higher in patients with atrial fibrillation, both because atrial fibrillation induces natriuretic peptide release and because the minimum NT-proBNP required for inclusion was higher in atrial fibrillation. Associations between NT-proBNP and clinical outcomes may be confounded by atrial fibrillation. Missing data were likely not random, but rather driven by death, worsening clinical status, or study drug discontinuation. Study design strengths include large sample size, randomized treatment exposure, and protocol-driven measurements of NT-proBNP at a central laboratory.

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In conclusion, in patients with HFmrEF/HFpEF, finerenone led to a fast (within 3 months of initiation) and sustained reduction of NT-proBNP, and reduced the risk of worsening HF and cardiovascular death regardless of baseline NT-proBNP concentration.

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Table 1: Baseline Characteristics by NT-proBNP Tertile in Patients With and Without Atrial Fibrillation

Panel A: Patients Without Atrial Fibrillation

	NT-proBNP Tertile			
	Tertile 1	Tertile 2	Tertile 3	P-Value
	n=1202	n=1202	n=1202	
Baseline NT-proBNP, pg/ml	233 [137,313]	588 [479,764]	1760 [1255,2940]	<0.001
Age, years	69 [62,74]	72 [65,77]	74 [66,80]	<0.001
Women	556 (46.3%)	533 (44.3%)	526 (43.8%)	0.22
Race				<0.001
Asian	133 (11.1%)	207 (17.2%)	244 (20.3%)	
Black	24 (2.0%)	22 (1.8%)	21 (1.7%)	
Other	48 (4.0%)	27 (2.2%)	34 (2.8%)	
White	997 (82.9%)	946 (78.7%)	903 (75.1%)	
Previous hospitalization for HF	635 (52.8%)	636 (52.9%)	806 (67.1%)	<0.001
Diabetes mellitus	530 (44.1%)	491 (40.8%)	558 (46.4%)	0.25
Systolic blood pressure, mm Hg	131 [123,139]	131 [120,142]	131 [120,141]	0.59
Body mass index, kg/m^2	31 [27,35]	29 [26,34]	28 [24,32]	<0.001
Estimated glomerular filtration rate, ml/min/1.73m^2	71 [56,86]	63 [49,79]	56 [41,72]	<0.001
Left ventricular ejection fraction, %	55 [48,59]	53 [46,58]	50 [45,56]	<0.001

Panel B: Patients With Atrial Fibrillation

	NT-proBNP Tertile			
	Tertile 1	Tertile 2	Tertile 3	P-Value
	n=747	n=745	n=745	
Baseline NT-proBNP, pg/ml	979 [733,1152]	1718 [1516,1963]	3415 [2807,4740]	<0.001
Age, years	73 [66,78]	76 [70,81]	77 [71,82]	<0.001
Women	328 (43.9%)	357 (47.9%)	355 (47.7%)	0.15
Race				0.31
Asian	130 (17.4%)	152 (20.4%)	124 (16.6%)	
Black	5 (0.7%)	5 (0.7%)	6 (0.8%)	
Other	32 (4.3%)	17 (2.3%)	18 (2.4%)	
White	580 (77.6%)	571 (76.6%)	597 (80.1%)	
Previous hospitalization for HF	457 (61.2%)	433 (58.1%)	549 (73.7%)	<0.001
Diabetes mellitus	255 (34.1%)	271 (36.4%)	270 (36.2%)	0.40

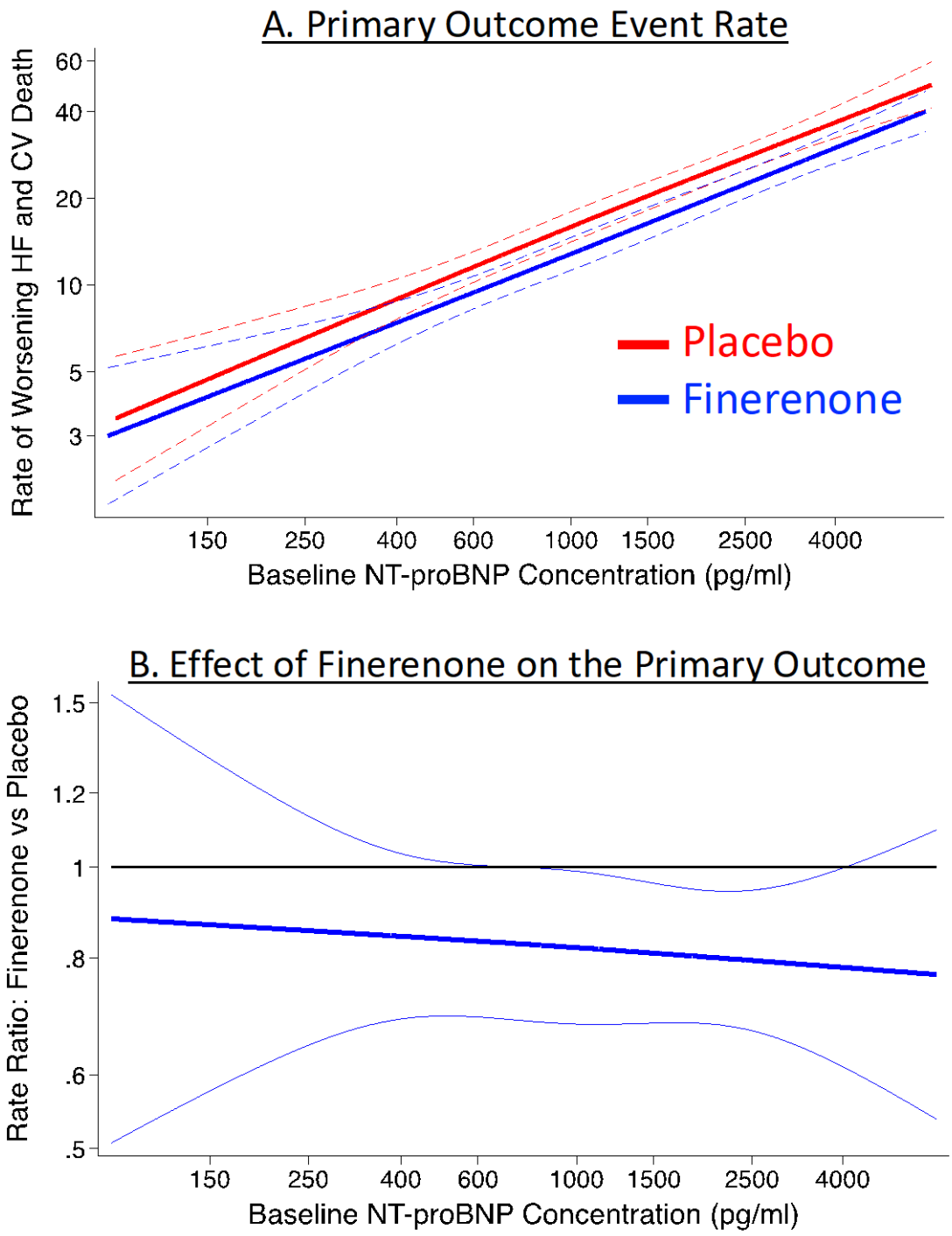
Systolic blood pressure, mm Hg	129 [118,139]	129 [117,139]	126 [116,136]	0.02
Body mass index, kg/m^2	31 [27,35]	29 [25,33]	28 [24,32]	<0.001
Estimated glomerular filtration rate, ml/min/1.73m^2	65 [52,79]	57 [46,70]	50 [38,63]	<0.001
Left ventricular ejection fraction, %	53 [47,57]	55 [48,58]	50 [45,57]	0.002

Atrial fibrillation was determined on the baseline electrocardiogram. NT-proBNP (N-terminal pro-B-type natriuretic peptide) tertiles were defined separately in patients with and without atrial fibrillation. Categorical variables are presented as number (%) and continuous variables as median [25th-75th percentile].

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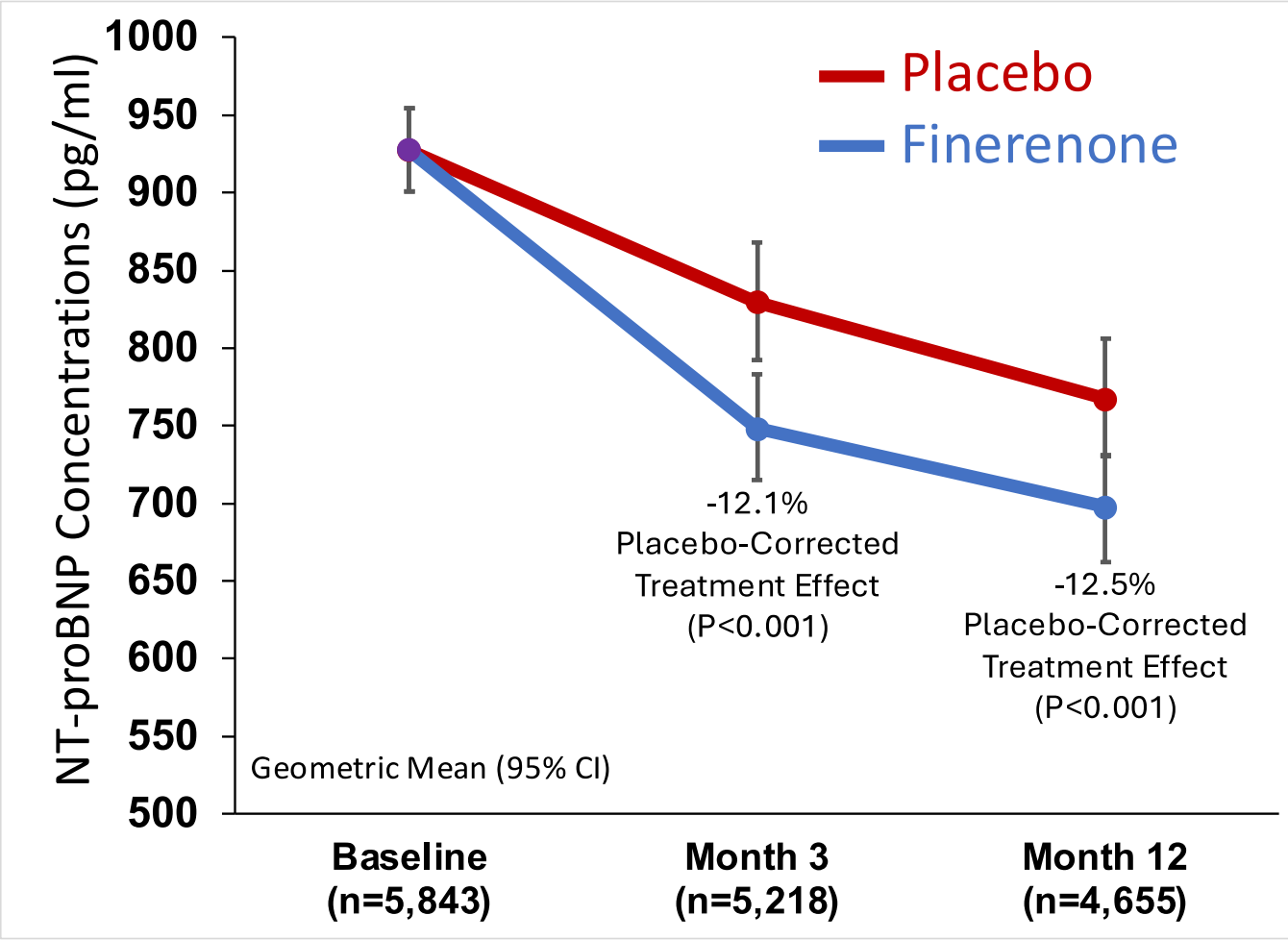
Figure 1: Clinical Benefit of Finereone Across the Spectrum of NT-proBNP



Panel A: The restricted cubic spline displays the predicted rate of the primary outcome, total worsening heart failure events and cardiovascular death, by baseline NT-proBNP concentration in each treatment group. Dashed lines indicate 95% confidence intervals.

Panel B: The restricted cubic spline displays the ratio for the primary outcome, total worsening heart failure events and cardiovascular death, according to baseline NT-proBNP. Baseline NT-proBNP does not significantly modify the relative reduction in the primary outcome with finerenone.

Figure 2: Treatment Effect of Finerenone on NT-proBNP



Geometric mean (95% confidence interval) NT-proBNP concentrations are shown at baseline and 3 and 12 months after randomization. Patients with missing NT-proBNP data at baseline were excluded at all three time points.

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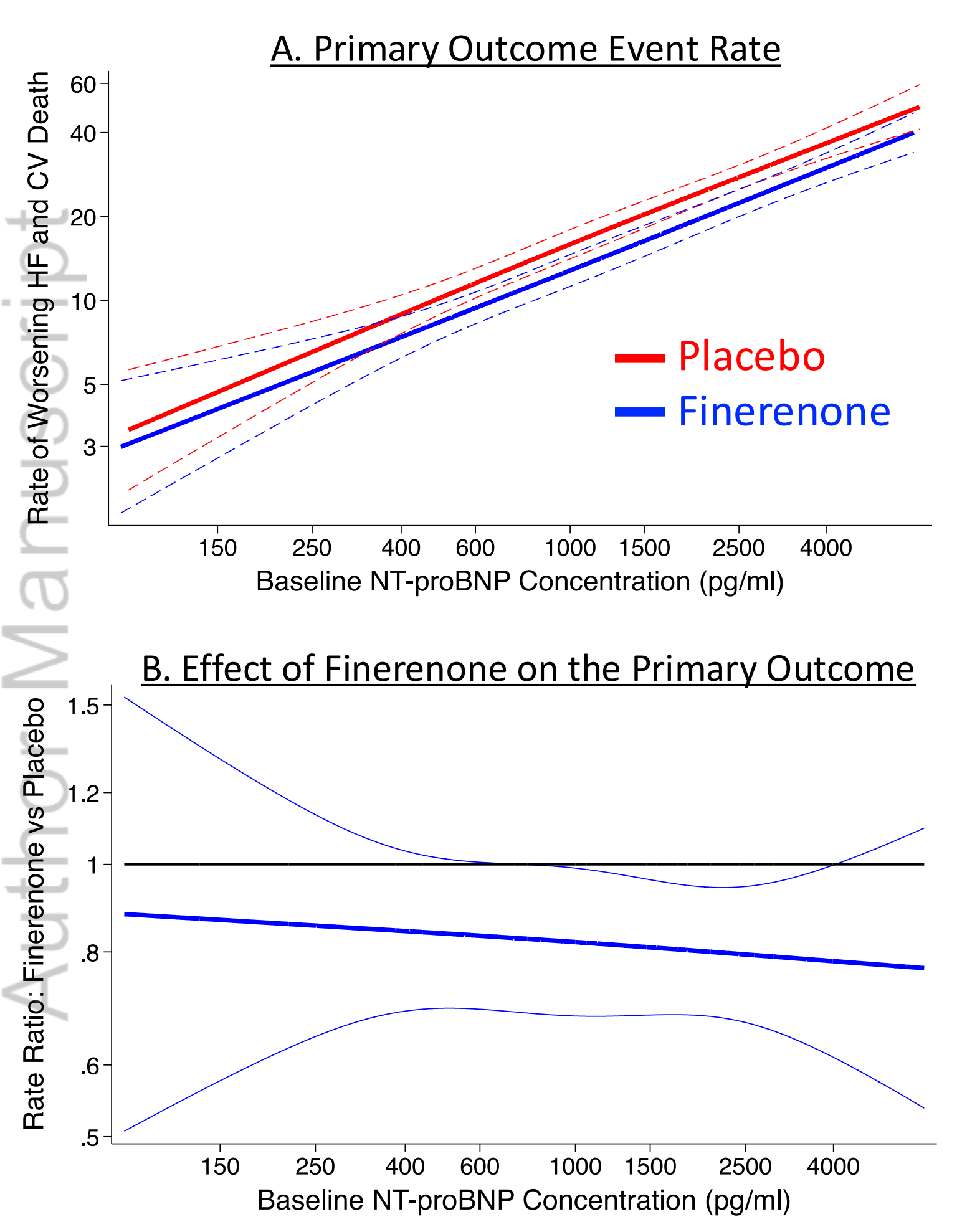
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