## Supplemental Table 1. Six-month Effect (95% CI) of 4 g/d Lovaza Treatment versus Placebo in Post MI Patients (Excluding 36 Patients with History of Prior MI at Baseline)

	LVESVI	Non-Infarct	Infarct Size	LVEF
		Myocardial Fibrosis		
ITT Analysis (GLMM <sup>†</sup> )	-7.2% (-11.9%, -2.2%) P = 0.0056, N = 322	-5.9% (-10.9%, -0.9%) P = 0.020, N = 322	-4.6% (-20.0%, 13.7%) P = 0.60, N = 322	2.8% (-0.2%, 5.7%) P = 0.073, N = 322
Per Protocol Analysis (t-test <sup>‡</sup> )	-7.7% (-12.8%, -2.7%) P = 0.0028, N = 221	-5.4% (-10.6%, -0.3%) P = 0.036, $N = 152$	-8.2% (-21.4%, 5.0%) P = 0.22, N = 228	2.8% (-0.4%, 6.0%) P = 0.083, N = 221

ITT was defined as intention-to-treat, LVEF left ventricular ejection fraction, and LVESVI left ventricular endsystolic volume index.

<sup>&</sup>lt;sup>†</sup> The general linear mixed model (GLMM) produces unbiased estimates for responses with missing data (see statistical analysis). LVESVI and Infarct Size were natural logarithm transformed to reduce skewness and/or heteroscedasticity of residuals. Estimates are relative changes.

<sup>‡</sup> The per protocol analysis only included patients that attended both visits. No transformations were required, instead Satterthwaite approximation was used for heteroscedasticity. Estimates are relative changes.

## Supplemental Table 2. Adjusted Analysis for Six-month Effect (95% CI) of 4 g/d Lovaza Treatment versus Placebo in Post MI Patients by Intention-to-treat Analysis

	LVESVI†	Non-Infarct	Infarct Size†¶	LVEF
		Myocardial Fibrosis		
Model 0*	-5.8% (-10.3%, -1.1%)	-5.6% (-10.4%, -0.9%)	-3.4% (-17.8%, 13.6%)	2.4% (-0.4%, 5.2%)
	P=0.017	P=0.022	P=0.68	P=0.094
Model 1 <sup>©</sup>	-5.4% (-10.1%, -0.6%)	-5.0% (-10.1%, 0.0%)	1.9% (-13.2%, 19.7%)	2.0% (-0.9%, 5.0%)
	P=0.030	P=0.046	P=0.81	P=0.16
Model 2§	-5.7% (-10.4%, -0.9%)	-4.7% (-9.7%, 0.3%)	2.2% (-13.3%, 20.3%)	2.2% (-0.7%, 5.2%)
	P=0.021	P=0.067	P=0.80	P=0.14

LVEF left ventricular ejection fraction, and LVESVI left ventricular end-systolic volume index.

<sup>\*</sup>Model 0 (N=358) demonstrates unadjusted analysis of O-3FA treatment versus placebo for the primary and secondary study endpoints

<sup>&</sup>lt;sup>Φ</sup>Model 1 (N=354) demonstrates Model 0 adjusted for fixed covariates, including age, gender, race, enrolling site, pre-treatment omega-3 index, and pre-treatment log transformed infarct mass.

<sup>§</sup>Model 2 (N=326) demonstrates Model 1 additionally adjusted for medical therapy (renin-angiotensin system blockade, β-adrenergic–receptor antagonists, dual antiplatelet therapy, hydroxymethylglutaryl–coenzyme A reductase inhibitors), and baseline coronary heart disease risk factors (diabetes mellitus, hypertension, hypercholesterolemia, body mass index, active smoking, and heart rate.)

<sup>¶</sup>Infarct mass (pre-treatment) was not included in these models.

<sup>&</sup>lt;sup>†</sup>Natural logarithm transformation was used to improve normality and/or homoscedasticity of residuals.