Erenumab (AMG 334) in episodic migraine: Interim analysis of an ongoing open-label study.

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Interim analysis of an ongoing open-label study

ABSTRACT
Objective: To assess long-term safety and efficacy of anti–calcitonin gene-related peptide receptor erenumab in patients with episodic migraine (EM).

Methods: Patients enrolled in a 12-week, double-blind, placebo-controlled clinical trial (NCT01952574) who continued in an open-label extension (OLE) study will receive erenumab 70 mg every 4 weeks for up to 5 years. This preplanned interim analysis, conducted after all participants had completed the 1-year open-label follow-up, evaluated changes in monthly migraine days (MMD), achievement of ≥50%, ≥75%, and 100% reductions, Headache Impact Test (HIT-6) score, Migraine-Specific Quality of Life (MSQ), Migraine Disability Assessment (MIDAS), and safety. Data reported as observed without imputation for missing data.

Results: Of 472 patients enrolled in the parent study, 383 continued in the OLE with a median exposure to erenumab of 575 days (range 28–822 days). Mean (SD) MMD were 8.8 (2.6) at parent study baseline, 6.3 (4.2) at week 12 (beginning of OLE), and 3.7 (4.0) at week 64 (mean change from baseline [reduction] of 5.0 days). At week 64, 65%, 42%, and 26% achieved ≥50%, ≥75%, and 100% reduction in MMD, respectively. Mean HIT-6 scores were 60.2 (6.3) at baseline and 51.7 (9.2) at week 64. MSQ and MIDAS improvements from baseline were maintained through week 64. Safety profiles during the OLE were similar to those in the double-blind phase, which overall were similar to placebo.

Conclusions: One-year efficacy, supported by functional improvements and favorable safety and tolerability profiles, supports further investigation of erenumab as a preventive treatment in patients with EM.

Clinicaltrials.gov identifier: NCT01952574.

Classification of evidence: This study provides Class IV evidence that for patients with episodic migraine, erenumab reduces long-term MMD and improves headache-related disability and migraine-specific quality of life. Neurology® 2017;89:1237-1243

GLOSSARY
AE = adverse event; CGRP = calcitonin gene-related peptide; EF = emotional function; EM = episodic migraine; HIT-6 = Headache Impact Test; MIDAS = Migraine Disability Assessment; MMD = monthly migraine days; MSQ = Migraine-Specific Quality of Life; OLE = open-label extension; RFP = role function—preventive; RFR = role function—restrictive; SC = subcutaneously.

Migraine is a disabling neurologic disorder, often accompanied by nausea, vomiting, photophobia, and phonophobia, affecting approximately 10% of the US population. Although at least one-third of individuals with episodic migraine (EM) and almost all patients with chronic migraine should receive preventive drug treatment, the majority do not receive preventive drug treatment. Over 80% of patients do not adhere to or persist with treatment 1 year after starting preventive medication. The suboptimal efficacy and tolerability of available preventive treatments, none of which was designed to treat migraine, are well-known and contribute to poor compliance and adherence. Migraine-specific preventive treatment is a major unmet need.
Erenumab (AMG 334) is a human monoclonal antibody that blocks the calcitonin gene-related peptide (CGRP) receptor. CGRP is a neuropeptide that plays a key role in migraine pathogenesis. Although the exact mechanism by which CGRP contributes to migraine attacks has not been fully elucidated, there is ample clinical evidence showing that CGRP and the CGRP receptor are suitable targets to prevent migraine. Although multiple studies have demonstrated clinical responses to antibodies targeting CGRP signaling, there have been no studies of the long-term safety and efficacy of these treatments. Here we report the preplanned interim results from an ongoing (up to 5 years) open-label extension (OLE) study of patients completing a 12-week, placebo-controlled, phase 2 study of erenumab for EM. At the time of analysis, all patients have either received open-label erenumab 70 mg every 4 weeks for at least 1 year or have discontinued treatment.

**METHODS Study design.** The parent study for this OLE was a 12-week, double-blind, placebo-controlled clinical trial. In the parent study, patients received erenumab monthly at 7, 21, or 70 mg administered subcutaneously (SC) or placebo. Patients who enrolled in the OLE study received erenumab at 70 mg SC monthly. This was a preplanned analysis of data once all patients completed at least 52 weeks of follow-up of the OLE study (week 64 overall) or discontinued open-label erenumab. Efficacy data were collected only up to 1 year of open-label erenumab, while safety data continued to be collected.

**Patients.** Eligibility criteria for enrollment in the parent study have been reported previously. Briefly, key inclusion criteria included age ≥18 and ≤60 years with history of migraine based on International Classification of Headache Disorders, second edition, for ≥12 months prior to screening, with at least 4 and ≤14 migraine days per month and <15 headache (migraine and nonmigraine) days per month. Patients could have failed up to 2 previous preventive therapies due to efficacy. To be eligible to continue in the OLE phase, patients had to complete the double-blind treatment phase and not discontinue investigational product early, and continue to provide informed consent; continued treatment had to be considered appropriate by the investigator. In addition, the investigators had to consider appropriateness of continued treatment for patients who developed any unstable or clinically significant medical condition, laboratory or ECG abnormality, or experienced a serious adverse event (AE) that could reasonably be related to study drug following randomization into the double-blind treatment phase.

**Study outcomes.** Efficacy endpoints for the OLE phase included change in monthly migraine days, achievement of ≥50%, ≥75%, and 100% reduction in monthly migraine days, and change in migraine-specific medication use for the last respective month. Patient-reported outcomes included the change in Headache Impact Test (HIT-6) score, Migraine-Specific Quality of Life (MSQ), and Migraine Disability Assessment (MIDAS). HIT-6 and MSQ data were collected every 4 weeks, and MIDAS was collected at weeks 12, 24, 36, 52, and 64. The HIT-6 is a short-form self-administered questionnaire that was developed as a global measure of adverse headache effect to assess headache severity in the previous month and change in a patient’s clinical status over a short period of time. Six questions cover severe pain, limitation of daily activity (household, work, school, and social), wanting to lie down when headache is experienced, feeling too tired to work or do daily activities because of headache, feeling fed up or irritated because of headache, and headache limiting ability to concentrate or work on daily activities. HIT-6 scores are categorized into 4 grades, representing little or no effect (≤50%), some effect (50–55%), substantial effect (56–59), and severe effect (60–78) due to headache. The within-person minimally important change for the HIT-6 score is ≥5 points and the between-group minimally important difference is estimated to be 2.3 points. The MSQ (version 2.1) is a self-administered 14-item instrument measuring 3 dimensions: role function-restrictive (RFR; 7 items assessing how migraines limit daily

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Demographics, clinical characteristics, and patient-reported outcome scores at baseline of the parent study for patients who entered the open-label extension*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>41.3 (10.9)</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>303 (79)</td>
</tr>
<tr>
<td>White race, n (%)</td>
<td>354 (92)</td>
</tr>
<tr>
<td>Age at migraine onset, y, mean (SD)</td>
<td>20.9 (11.3)</td>
</tr>
<tr>
<td>Duration of disease, y, mean (SD)</td>
<td>20.9 (11.9)</td>
</tr>
<tr>
<td>History of migraine with aura, n (%)</td>
<td>137 (36)</td>
</tr>
<tr>
<td>Monthly migraine days, mean (SD)</td>
<td>8.7 (2.7)</td>
</tr>
<tr>
<td>Monthly headache days, mean (SD)</td>
<td>9.8 (2.7)</td>
</tr>
<tr>
<td>Monthly migraine-specific medication days, mean (SD)</td>
<td>4.3 (3.7)</td>
</tr>
<tr>
<td>Prior prophylactic history, n (%)</td>
<td>214 (56)</td>
</tr>
<tr>
<td>Prior use</td>
<td>169 (44)</td>
</tr>
<tr>
<td>Treatment failure</td>
<td>138 (36)</td>
</tr>
<tr>
<td>Other</td>
<td>31 (9)</td>
</tr>
<tr>
<td>HIT-6, median score (Q1, Q3)</td>
<td>61.0 (56.0, 64.0)</td>
</tr>
<tr>
<td>MSQ, median score (Q1, Q3)</td>
<td>60.0 (48.6, 71.4)</td>
</tr>
<tr>
<td>MSQ-RFP</td>
<td>75.0 (65.0, 90.0)</td>
</tr>
<tr>
<td>MSQ-EF</td>
<td>73.3 (60.0, 86.7)</td>
</tr>
<tr>
<td>MIDAS, median score (Q1, Q3)</td>
<td>22.0 (11.0, 38.0)</td>
</tr>
<tr>
<td>Total score</td>
<td>22.0 (11.0, 38.0)</td>
</tr>
<tr>
<td>Absenteeism</td>
<td>10.0 (5.0, 19.0)</td>
</tr>
<tr>
<td>Presenteeism</td>
<td>10.0 (5.0, 19.0)</td>
</tr>
</tbody>
</table>

Abbreviations: EF = emotional function; HIT = Headache Impact Test; MIDAS = Migraine Disability Assessment; MSQ = Migraine-Specific Quality of Life; Q1 = first quartile; Q3 = third quartile; RFP = role function-preventive; RFR = role function-restrictive.

* Baseline was prior to the parent study double-blind phase.

† migraine-specific medications were triptans and ergot amine-derivative. A total of 259 (68%) patients received triptans and 4 (1%) patients received ergotamine derivatives during the baseline period.

‡ Treatment failure included discontinuation due to lack of efficacy or side effects.
social and work-related activities), role function—preventive (RFP; 4 items assessing how migraines prevent these activities), and emotional function (EF; 3 items assessing the emotions associated with migraines). Raw dimension scores are computed as a sum of item responses and rescaled from a 0 to 100 scale; higher scores indicate better quality of life.

The MIDAS is a 5-item self-administered questionnaire that sums the number of productive days lost over the last 3 months in the workplace and the home. The MIDAS also assesses disability in family, social, and leisure activities. The MIDAS score is the sum of missed days due to a headache from paid work, housework, and nonwork (family, social, leisure) activities and days at paid work or housework where productivity was reduced by at least one-half.

Safety endpoints included all AEs, clinical laboratory values and vital signs, and development of anti-erenumab antibodies. AEs were coded according to the Medical Dictionary for Regulatory Activities version 18.1, and severity was graded using the Common Terminology Criteria for Adverse Events version 4.03. Patient-year adjusted incidence rate was defined as the total number of patients who reported that event in a given time period of follow-up divided by total patient-years of exposure in that period. Total patient-years of exposure was defined as the sum of the number of patients times the duration of exposure from first erenumab dose to last erenumab dose, end of study, or first report of event.

Statistical considerations. All patients who received at least 1 dose of erenumab in the OLE study were included in the analysis. Descriptive summaries are provided for efficacy and patient-reported outcomes. AEs are summarized as the exposure-adjusted patient incidence rate per 100 patient-years. Data are reported as observed, without imputation for missing data.

Primary research question. Does erenumab treatment reduce monthly migraine days and improve patient-reported outcomes? This study provides Class IV evidence that for patients with EM, erenumab reduces long-term monthly migraine days (MMD) and improves headache-related disability and migraine-specific quality of life.

Standard protocol approvals, registrations, and patient consents. This trial is registered with ClinicalTrials.gov (NCT 01952574). All procedures were approved by institutional review boards at all participating sites. Patients provided written informed consent.

RESULTS Patients. Of 472 patients who received erenumab or placebo in the parent study, 383 patients entered into the OLE study. A total of 307 patients (80%) completed 1 year of open-label treatment. At the time of this analysis, 273 (71%) were continuing investigational product, 3 (1%) received their last scheduled dose of OLE investigational product at week 48 (planned final administration based on the original protocol), and 107 (28%) discontinued the investigational product because of patient request (n = 54), AE (n = 14), lost to follow-up (n = 11), lack of efficacy (n = 11), noncompliance (n = 4), ineligibility determined (n = 1), protocol deviation (n = 1), or other reasons (n = 11) (figure e-1 at Neurology.org). The mean age was 41 years and most patients (79%) were female (table 1). Clinical characteristics specific to migraine at baseline were consistent with the patient population with EM (table 1).

Efficacy results. For patients enrolled in the OLE, mean (SD) monthly migraine days were 8.8 (2.6) days at baseline (prior to double-blind treatment in the parent study), 6.3 (4.2) at week 12 (end of double-blind treatment), and 3.7 (4.0) at week 64 (figure 1A), representing a reduction of 5.0 (4.2) monthly migraine days. After switching from placebo or lower erenumab dosages (7, 21 mg) to 70 mg at week 12 of the parent study, reductions in monthly migraine days were observed at week 16, the first efficacy assessment time point of the OLE (figure e-2). At week 64, 184 (65%) patients had achieved...
50% reduction, 119 (42%) had achieved ≥75% reduction, and 73 (26%) had achieved 100% reduction in monthly migraine days. Mean (SD) monthly migraine-specific medication days were 4.3 (3.7) at baseline and 2.1 (3.3) at week 64 (figure 1B), representing a mean reduction of 2.4 monthly migraine-specific medication days in the OLE phase. The mean change (SD) from baseline to week 64 in monthly headache days with moderate/severe pain was −4.7 (4.2) days; in monthly migraine attacks was −2.9 (2.5) attacks; in cumulative hours of migraine pain was −47.4 (57.4) hours; and in cumulative hours of headache was −48.9 (60.1) hours.

**Patient-reported outcomes.** The mean (SD) HIT-6 total score was 60.2 (6.3) at baseline and 51.7 (9.2) at week 64. Mean change from baseline is shown in figure 2. Improvements in HIT-6 total score were maintained through week 64 with 187 (64%) patients achieving ≥5-point reduction (the within-person minimally important change) in HIT-6 score. MSQ-RFR, MSQ-RFP, and MSQ-EF scores improved from baseline and were maintained through week 64 (figure 2). Similarly, MIDAS total score, presenteeism, and absenteeism improved from baseline and were maintained through week 64 (figure 2). These improvements on traditional patient-reported outcomes paralleled the observed reductions in MMD.

**Safety.** Median erenumab exposure during the open-label phase was 575 days (range 28–822 days) with a total exposure of 555.4 patient-years. Overall, 300 patients reported AEs throughout the OLE study for an exposure-adjusted patient rate of 140.6 per 100 patient-years, which is less than the placebo and erenumab 70 mg rates observed during the 12-week double-blind treatment period (table 2). The types and natures of AEs and the incidence rates were comparable with previous observations and did not reveal any new safety concerns. There were no clinically significant changes in vital signs, laboratory values, or ECG findings during the OLE. A single event each of arteriosclerosis and myocardial ischemia were reported in the OLE study. The arteriosclerosis event was a fatal event in a 52-year-old man with history of migraine with aura, and was confounded by preexisting cardiovascular risk factors. The patient had a 3-year history of diagnosed hypertension with prior treatment with lisinopril and hydrochlorothiazide, obesity (body mass index 37 kg/m²), a screening low-density

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**Figure 2** Change in Headache Impact Test (HIT-6) total score, Migraine-Specific Quality of Life (MSQ) scores, and Migraine Disability Assessment (MIDAS) scores

(A) The change from baseline in mean HIT-6 total score is shown for patients on placebo (blue line), erenumab 7 mg (red line), erenumab 21 mg (green line), and erenumab 70 mg (purple line) during the double-blind parent study and for all patients on erenumab 70 mg (purple line) during the open-label extension (OLE). Error bars represent SE. The changes from baseline in mean (B) MSQ-role function-restrictive (RFR) score, (C) MSQ-role function-preventative (RFP) score, (D) MSQ-emotional function (EF) score, (E) MIDAS total score, (F) MIDAS presenteeism, and (G) MIDAS absenteeism are shown for patients on placebo (blue line), erenumab 7 mg (red line), erenumab 21 mg (green line), and erenumab 70 mg (purple line) during the double-blind parent study and for all patients on erenumab 70 mg (purple line) during the OLE. Error bars represent SE.
lipoxymporin level of 153 mg/dL, left anterior hemiblock on baseline ECG, and a family history of myocardial infarction. Autopsy showed evidence of severe coronary atherosclerosis and presence of cardiac stimulants (phenylpropanolamine and norpseudoephedrine) in the liver; this event was considered not related to treatment per the investigator. The myocardial ischemia event was based on results of an exercise treadmill test (performed to evaluate exercise-induced dyspnea), which showed transient exercise-induced myocardial ischemia (ST segment depression on ECG, angina was not reported). However, this case was confounded by sumatriptan administration 4 hours prior to the event. No cardiac enzymes were reported on the day of the exercise ECG, but they were normal 4 days prior; coronary angiography was subsequently performed and was normal.

Of 382 patients with antibody testing result after the first erenumab dose, 50 (13.1%) patients enrolled in the OLE study developed binding (non-neutralizing) antibodies on at least one occasion, and 29 of the 50 patients had a transient response, with a negative result at the last time point tested. No patients in any group had preexisting antibodies prior to the first erenumab dose. Of 382 patients, 9 (2.4%) patients were positive for neutralizing antibodies against erenumab on at least one occasion; of these, 8 patients had a transient response. Development of anti-erenumab antibodies was not associated with any clinical finding or safety events.

**DISCUSSION** Erenumab treatment resulted in long-term durable, stable improvements in disability, headache effect, and migraine-specific quality of life. Safety and tolerability profiles during the OLE (total exposure 555.4 patient-years) were similar to those observed for erenumab 70 mg in the double-blind parent study (total exposure 23.9 patient-years) and overall were similar to placebo (34.1 patient-years) in the parent study. Discontinuation rates due to AEs were low, which is in contrast to current migraine prophylactics that are associated with high discontinuation rates. In the 8-month OLE of the pivotal topiramate trials, 28.7% of participants withdrew and of those withdrawing 42% withdrew due to an AE. The safety data in this analysis were based on a median exposure of 575 days with 75% of participants having received erenumab for 474 days. The mean reduction in monthly migraine days with erenumab 70 mg persisted through at least 1 year of open-label treatment. Reductions in monthly migraine days were evident 4 weeks after patients switched from placebo or lower erenumab dosages (7, 21 mg) to 70 mg and treatment effect was sustained throughout the OLE, such that at week 64 patients experienced a 5.0-day reduction in monthly migraine days compared to baseline of the parent study. At week 64, response rates of ≥50%, ≥75, and 100% were achieved by 65%, 42%, and 26% of patients, respectively. Notably, at the group level there was no evidence of tachyphylaxis after 1 year of erenumab treatment.

No new safety concerns were identified during the OLE. This confirms previous observations from other studies, where the safety and tolerability profile of

### Table 2  
**Follow-up exposure-adjusted patient rates of adverse events (AEs)**

<table>
<thead>
<tr>
<th>Event, n° [r]</th>
<th>Double-blind treatment phase, placebo (n = 153)</th>
<th>Double-blind treatment phase, erenumab 70 mg (n = 106)</th>
<th>OLE phase, erenumab 70 mg (n = 383)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All AEs</td>
<td>82 (350.1)</td>
<td>57 (326.2)</td>
<td>300 (140.6)</td>
</tr>
<tr>
<td>Common AEs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nasopharyngitis</td>
<td>12 (33.9)</td>
<td>6 (23.3)</td>
<td>66 (13.4)</td>
</tr>
<tr>
<td>Upper respiratory tract infection</td>
<td>3 (8.0)</td>
<td>3 (11.4)</td>
<td>42 (8.0)</td>
</tr>
<tr>
<td>Back pain</td>
<td>4 (10.8)</td>
<td>1 (3.8)</td>
<td>27 (5.0)</td>
</tr>
<tr>
<td>Influenza</td>
<td>5 (13.5)</td>
<td>1 (3.5)</td>
<td>27 (5.0)</td>
</tr>
<tr>
<td>Grade ≥2</td>
<td>37 (117.1)</td>
<td>23 (98.0)</td>
<td>216 (85.1)</td>
</tr>
<tr>
<td>Grade ≥3</td>
<td>2 (5.3)</td>
<td>3 (11.5)</td>
<td>47 (9.0)</td>
</tr>
<tr>
<td>Serious AEs</td>
<td>0</td>
<td>1 (3.8)</td>
<td>21 (3.8)</td>
</tr>
<tr>
<td>AEs leading to discontinuation of IP</td>
<td>2 (5.3)</td>
<td>3 (11.5)</td>
<td>14 (2.5)</td>
</tr>
<tr>
<td>Ischemic heart disease/cerebrovascular AEs of interest</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arteriosclerosis</td>
<td>0</td>
<td>0</td>
<td>1 (0.2)</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
<td>0</td>
<td>0</td>
<td>1 (0.2)</td>
</tr>
<tr>
<td>ECG T-wave inversion</td>
<td>1 (2.7)</td>
<td>0</td>
<td>1 (0.2)</td>
</tr>
</tbody>
</table>

Abbreviations: IP = investigational product; OLE = open-label extension; r = exposure-adjusted rate per 100 patient-years (n/e*10^2).  

A Number of patients reporting at least 1 occurrence of event.
Erenumab were largely comparable to placebo. The events of arteriosclerosis and myocardial ischemia, which occurred during the uncontrolled OLE period, were clinically confounded and their relevance uncertain without a placebo comparator, particularly given that cardiovascular events occur with higher frequency in migraine patients compared to individuals without migraine. There is a theoretical cardiovascular risk with inhibition of the CGRP pathway, as CGRP is among a number of mediators (including substance P, neurokinins, and nitric oxide) released during ischemia that have vasodilatory properties. Accordingly, preclinical and clinical safety studies with erenumab are being conducted to better characterize the putative cardiovascular effect of antagonizing the CGRP pathway. Importantly, in the double-blind placebo-controlled portion of the study, no increased incidence of cardiovascular events compared to placebo and in phase 1 studies of erenumab there was no effect of erenumab on blood pressure. In addition to this OLE, longer-term safety will be assessed in a controlled manner in larger phase 3 studies, one with a 6-month placebo-controlled phase.

In short-term phase 1 and phase 2 clinical trials of erenumab (including the double-blind treatment period of the study reported here), there was a low incidence of binding and neutralizing anti–erenumab antibodies. The incidence of anti–erenumab antibodies remained low throughout the OLE study. The development of anti–erenumab antibodies has not been associated with any clinical finding or safety events; however, this tolerability profile of erenumab in patients with EM requires long-term safety outcomes for confirmation.

A limitation of the study is the lack of a placebo group for efficacy and safety comparisons. It is therefore difficult to interpret the possible relatedness of an AE without a placebo arm, and it is difficult to distinguish spontaneously occurring AEs from AEs due to erenumab. However, the OLE study is ongoing and will continue to provide a long-term safety experience for erenumab.

Retention rates, efficacy, patient-reported outcomes, and safety results after 1 year for erenumab in patients with EM are promising. These data support further investigation of erenumab as a potential preventive treatment option for patients with EM.

AUTHOR CONTRIBUTIONS
M.A., D.D., U.R., S.S., F.Z., S.C., D.D.M., and R.A.L. interpreted the data and revised the manuscript for content. P.J.G. and R.A.L. were involved in design of the study, interpreted the data, and revised the manuscript for content. J.R.G. wrote the first draft of the manuscript based on an outline developed with all the coauthors.

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