

Cannabidiol Effectiveness in Patients with Lennox-Gastaut Syndrome of Diverse Aetiologies Compared with External Placebo Control: A *Post Hoc* Analysis from the US Expanded Access Program

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Objective

- A *post hoc* analysis of the US Expanded Access Program to evaluate the effectiveness of cannabidiol (CBD; Epidiolex® [US], 100 mg/mL oral solution) in patients with Lennox-Gastaut syndrome (LGS) arising from diverse aetiologies



Conclusions

- Treatment with cannabidiol in patients with LGS across different aetiologies was associated with reductions in frequency of convulsive and total seizures
- A comparative effectiveness analysis versus an external control arm indicated that CBD reduced seizure frequency versus placebo
- The safety profile of CBD was comparable with the overall CBD LGS randomised controlled trial populations
- Overall, these findings support CBD use in patients with LGS, irrespective of LGS aetiology

References: 1. Specchio N, et al. *Epilepsia*. 2022;63(6):1398–1442. 2. Na J-H, Lee Y-M. *Int J Mol Sci*. 2026;27(3):1382. 3. Amrulkar CV, Lui F. Lennox-Gastaut Syndrome. In StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025. 4. Auvin S, et al. *Epilepsia Open*. 2024;10(1):185–195. 5. US Food and Drug Administration. Epidiolex® Prescribing Information. 2025. Available from: https://www.accessdata.fda.gov/drugsatfda_docs/label/2026/210365s024s029s030b1.pdf (Accessed 19 August 2026). 6. Devinsky O, et al. *N Engl J Med*. 2018;378(20):1888–1897. 7. Thiele EA, et al. *Lancet*. 2018;391(10125):1085–1095. 8. Nabbutt R, et al. *Seizure*. 2023;110:78–85. 9. Gowda V, et al. *Indian Pediatr*. 2025;62(7):501–505. 10. Strzelczyk A, et al. *Neurol Ther*. 2025;14(5):1935–1959. 11. Berg AT, et al. *Epilepsy Res*. 2024;200:107280. 12. Perry MS, et al. *Seizure*. 2025;130:32–40. 13. Elabdai M, et al. *Epilepsy Behav*. 2024;157:109904. 14. Perulli M, et al. *Epilepsia Open*. 2025;10(6):1806–1813.

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Epidiolex® is approved in the EU for the adjunctive treatment of seizures associated with Lennox-Gastaut syndrome or Dravet syndrome, in conjunction with clobazam, in patients ≥2 years of age; it is additionally approved in the EU for the adjunctive treatment of seizures associated with tuberous sclerosis complex in patients ≥2 years of age. Epidiolex® is approved in the US for the treatment of seizures associated with Lennox-Gastaut syndrome, Dravet syndrome, or tuberous sclerosis complex in patients ≥1 year of age.



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Introduction

- Lennox-Gastaut syndrome (LGS) is a severe developmental and epileptic encephalopathy. The underlying aetiology of LGS is diverse and may encompass genetic causes, structural abnormalities such as cortical malformations and metabolic disorders, or it may be unknown or not specified in some patient records^{1–3}
 - Figure S1** provides a conceptual framework for the diversity of epilepsies and LGS-related aetiologies, including structural causes, tuberous sclerosis complex, and genetic/monogenic epilepsies that may contribute to, or overlap with, LGS. **Supplementary material** can be accessed by scanning the poster QR code
 - Identification of the underlying aetiologies is important in LGS, as it may provide opportunities for targeted treatment approaches⁴
- The efficacy of plant-derived, highly purified cannabidiol (CBD; Epidyolex® [EU]/ Epidiolex® [US], 100 mg/mL oral solution)⁵ for reducing seizures associated with LGS has been demonstrated in randomised controlled trials, and its effectiveness in clinical practice is supported by real-world evidence.^{6–14} However, data on effectiveness of CBD by underlying LGS aetiology remain limited
- This *post hoc* analysis evaluated the effectiveness and safety of CBD in patients with LGS arising from diverse aetiologies, using data from the US Expanded Access Program (EAP). Comparative effectiveness was assessed versus an external control arm (ECA) derived from the placebo groups of prior LGS CBD clinical trials^{5,7}

Methods

Study design and patients

- This *post hoc* analysis used data from a multicentre, prospective interventional trial that enrolled children and young adults with childhood-onset treatment-resistant epilepsies who received CBD as part of the US EAP (maximum dose range: 25–50 mg/kg/day, at investigator/institutional review board discretion)
 - Note, since regulatory approval of CBD, the maximum approved dose for use in patients with LGS is 20 mg/kg/day⁵
- Eligible patients were receiving stable doses of antiseizure medications (ASMs) for ≥4 weeks before enrolment

Descriptive analyses: CBD EAP cohort

- Using Case Report Forms, patients diagnosed with LGS were further subclassified based on aetiology, including LGS associated with structural defects (LGS-structural), genetic epilepsies (LGS-genetic), or not otherwise specified (NOS)
- Effectiveness: percent change from baseline in median 28-day convulsive and total seizure frequencies, and responder proportions (≥50% reduction) at Weeks 12, 24, 36, and 48
- Safety: incidence of treatment-emergent adverse events (TEAEs) was recorded through Week 48
- Descriptive analyses were conducted in the overall LGS population and aetiology subgroups. Baseline characteristics and safety were summarised in the CBD EAP **Safety Analysis Set**, and seizure-frequency and responder endpoints were evaluated in the CBD EAP **Effectiveness Analysis Set**

Comparative analyses: CBD EAP versus placebo-LGS ECA

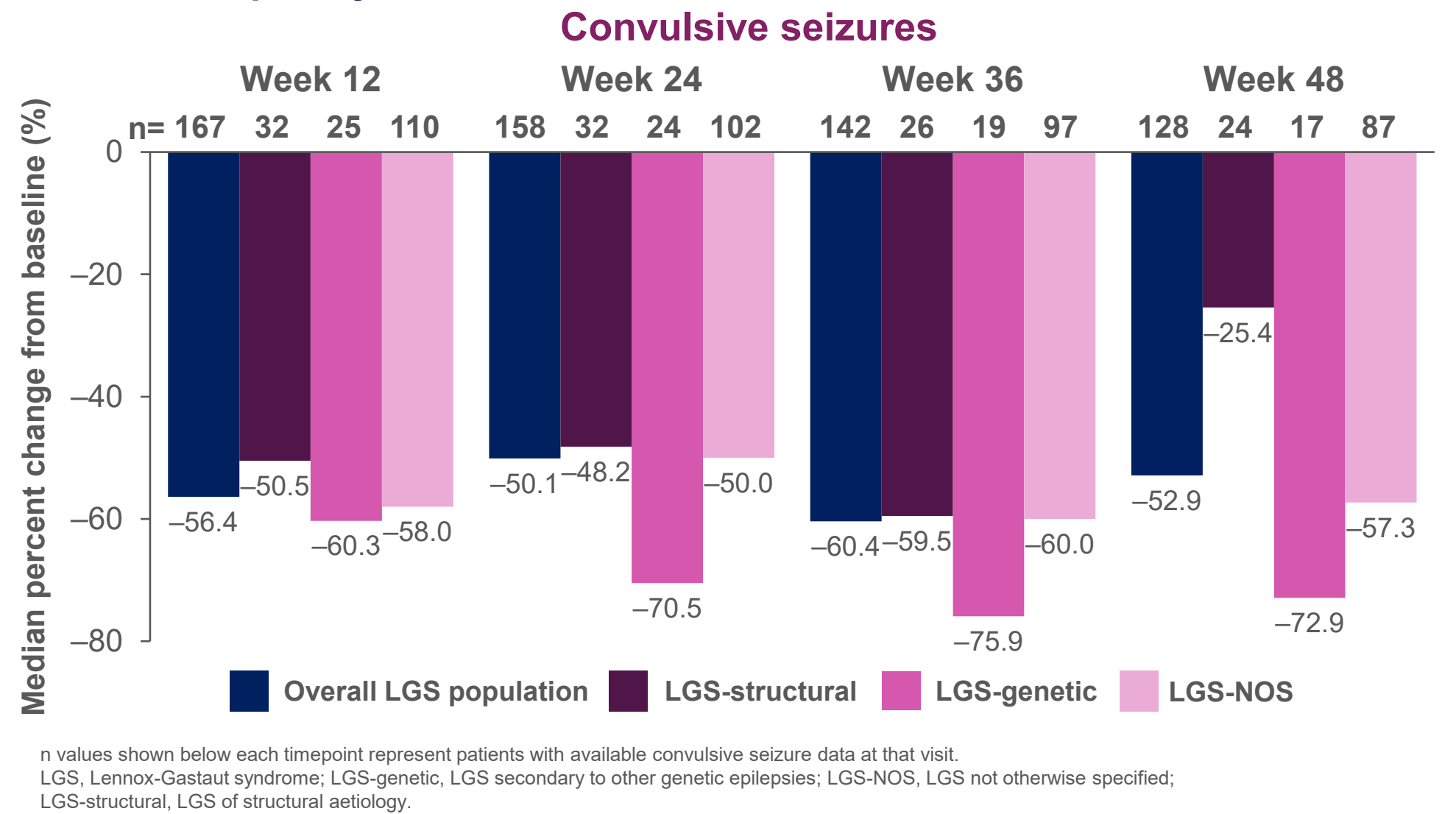
- Comparative effectiveness analysis in the weighted CBD EAP versus placebo-LGS ECA population estimated average treatment effects for 28-day average convulsive and total seizure frequencies during the treatment period
- CBD EAP outcomes were compared with a placebo-LGS ECA derived from prior LGS CBD trials, GWPCARE3 (NCT02224560), and GWPCARE4 (NCT02224690)
- Inverse probability weighting based on propensity scores was applied to balance key baseline covariates, including age, sex, baseline convulsive seizure frequency, number of concomitant ASMs, and current clobazam use. Covariate balancing was assessed using standardised weighted mean differences
- Treatment effects were estimated using weighted log-transformed analysis of covariance, with log-transformed baseline seizure frequency and treatment group as model covariates
- Sensitivity to hidden bias was assessed using a hidden-bias parameter analysis
- This study was conducted with Epidiolex®, and results of this *post hoc* analysis do not apply to other CBD-containing products

Results

Descriptive analyses

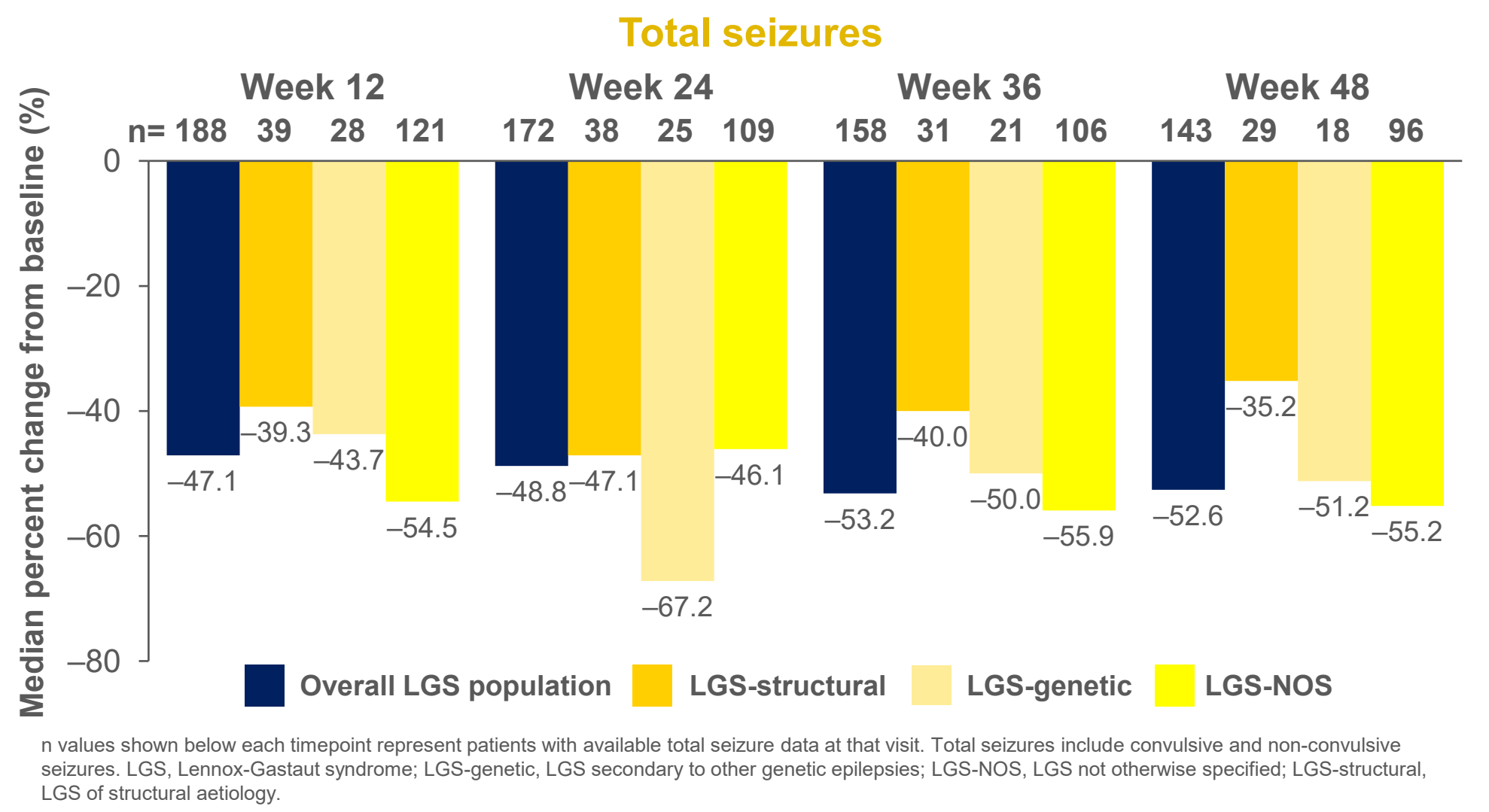
Effectiveness: CBD EAP Effectiveness Analysis Set

Figure 1. Median percent change from baseline in convulsive seizure frequency



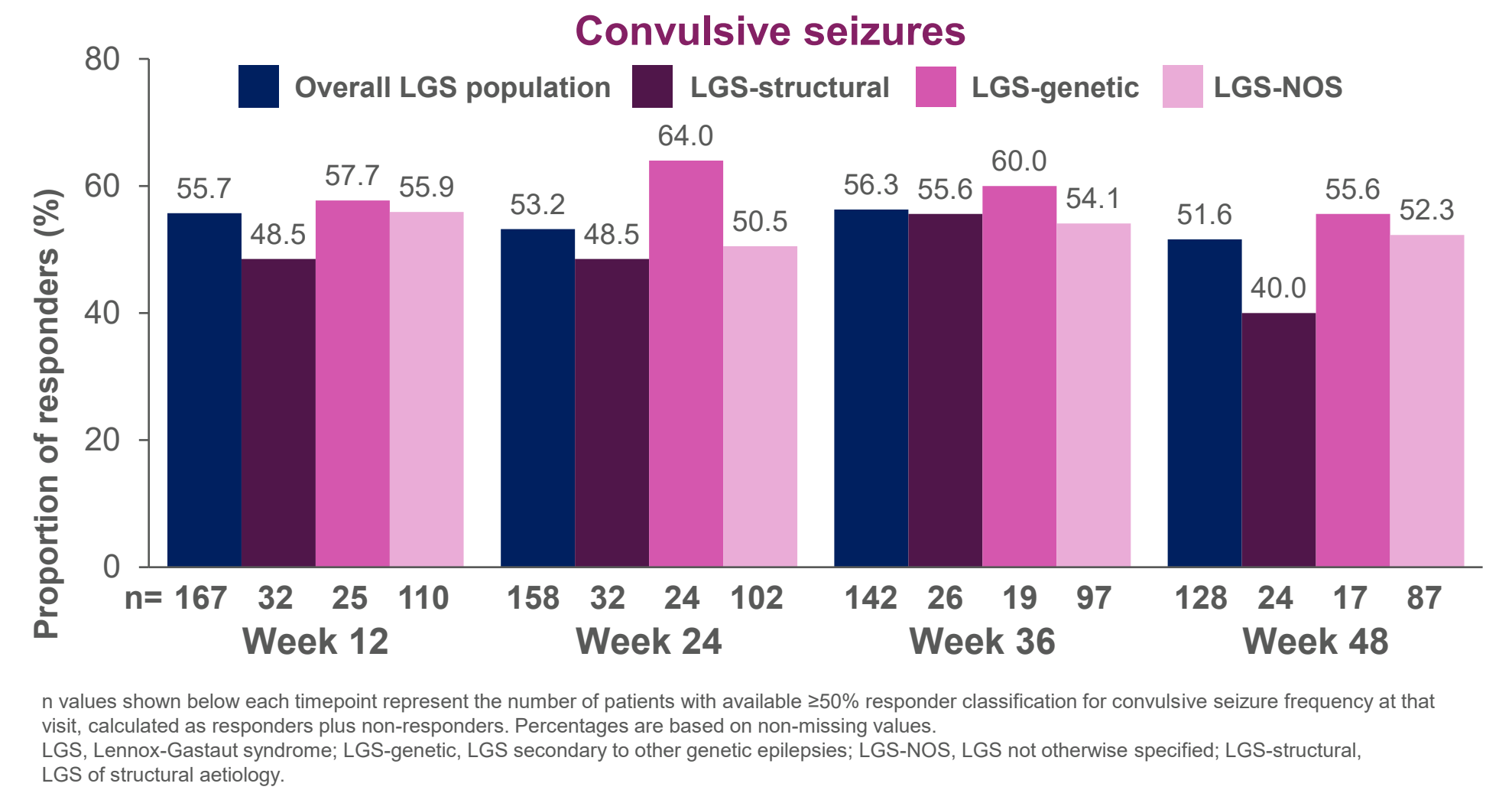
- At Week 12, median percent change from baseline in convulsive seizure frequency was –56.4% in the overall population and ranged from –50.5% to –60.3% across aetiology subgroups (**Figure 1**)
- Median percent change in convulsive seizure frequency was broadly maintained through Week 48 in the overall population (–52.9%), LGS-genetic subgroup (–72.9%), and LGS-NOS subgroup (–57.3%); in the LGS-structural subgroup, reductions were >48% through Weeks 12–36 and –25.4% at Week 48

Figure 2. Median percent change from baseline in total seizure frequency



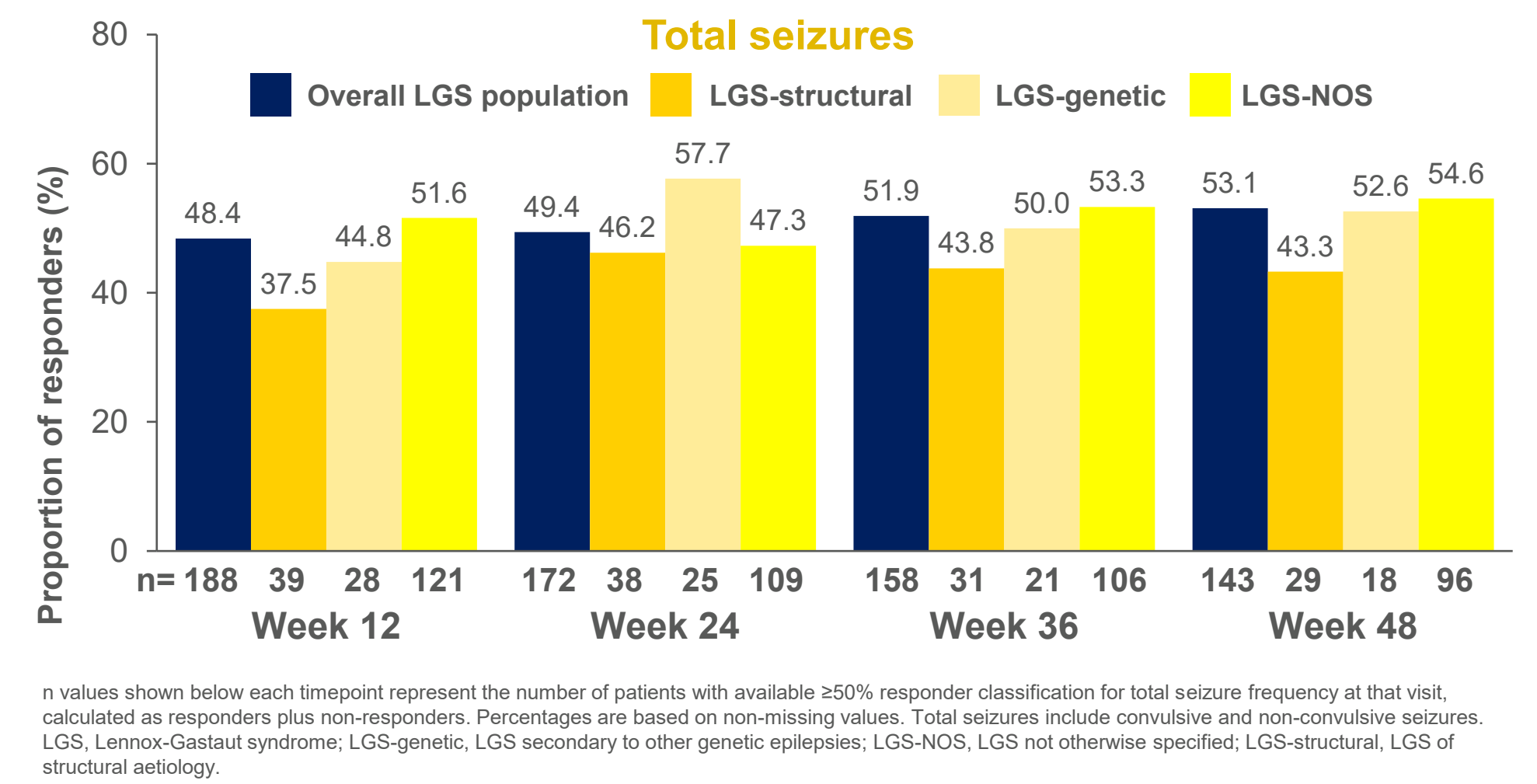
- At Week 12, median percent change from baseline in total seizure frequency was –47.1% in the overall population and ranged from –39.3% to –54.5% across aetiology subgroups (**Figure 2**)
- Median percent change in total seizure frequency was broadly maintained through Week 48 in the overall population (–52.6%) and LGS-NOS subgroup (–55.2%); reductions were >35% through Weeks 12–48 in the LGS-structural subgroup and ≥50% through Weeks 24–48 in the LGS-genetic subgroup

Figure 3. Proportion of responders (≥50% reduction from baseline) for convulsive seizures (Effectiveness Analysis Set)



- At Week 12, the proportion of responders for convulsive seizure frequency was 55.7% in the overall population and ranged from 48.5% to 57.7% across aetiology subgroups (**Figure 3**)
- Across Weeks 12–48, responder proportions were broadly maintained in the overall population (51.6–56.3%), LGS-genetic subgroup (55.6–64.0%), and LGS-NOS subgroup (50.5–55.9%); in the LGS-structural subgroup, responder proportions were 48.5–55.6% through Weeks 12–36 and 40.0% at Week 48

Figure 4. Proportion of responders (≥50% reduction from baseline) for total seizures (Effectiveness Analysis Set)



- At Week 12, the proportion of responders for total seizure frequency was 48.4% in the overall population and ranged from 37.5% to 51.6% across aetiology subgroups (**Figure 4**)
- Across Weeks 12–48, responder proportions were broadly maintained in the overall population (48.4–53.1%) and LGS-NOS subgroup (47.3–54.6%); in the LGS-genetic subgroup, responder proportions were ≥50% from Weeks 24–48, and in the LGS-structural subgroup ranged from 37.5% to 46.2%

Baseline characteristics: CBD EAP Safety Analysis Set

Table 1. Baseline characteristics in the overall population and aetiology subgroups (CBD EAP Safety Analysis Set)

	Overall (N=226)	LGS-structural (n=49)	LGS-genetic (n=32)	LGS-NOS (n=145)
Age, median (min–max)	13.1 (0–58)	13.6 (3–58)	13.0 (0–21)	13.1 (1–51)
Sex (female), n (%)	81 (35.8)	23 (46.9)	14 (43.8)	44 (30.3)
ASMs, mean (SD)	3.1 (1.3)	3.3 (1.2)	2.8 (1.2)	3.1 (1.3)
Concomitant ASM use, n (%)				
Clobazam	125 (55.3)	26 (53.1)	14 (43.8)	85 (58.6)
Levetiracetam	86 (38.1)	20 (40.8)	16 (50.0)	50 (34.5)
Valproate	64 (28.3)	16 (32.7)	5 (15.6)	43 (29.7)
Lamotrigine	60 (26.5)	17 (34.7)	7 (21.9)	36 (24.8)
Rufinamide	50 (22.1)	7 (14.3)	10 (31.3)	33 (22.8)
Topiramate	33 (14.6)	7 (14.3)	3 (9.4)	23 (15.9)
Felbamate	27 (11.9)	6 (12.2)	4 (12.5)	17 (11.7)
Stiripentol	1 (0.4)	0	1 (3.1)	0

- In the overall population (N=226), the mean (standard deviation) age was 13.8 (8.5) years and approximately two thirds of patients were male
- Clobazam (55% [125/226]), levetiracetam (38% [86/226]), and valproate (28% [64/226]) were the most common concomitant ASMs (**Table 1**)
- Baseline characteristics were generally balanced between the overall population and the different EAP subgroups (LGS-structural: n=49; LGS-genetic: n=32; LGS-NOS: n=145), with some numerical differences (**Table 1**):
 - Patients with LGS-structural had a higher mean age (15.8) compared with the overall population, and a higher proportion (47% [23/49]) were female
 - Structural and genetic aetiology categories are detailed in **Supplementary Tables S1 and S2**, respectively
 - Compared with the overall population, more patients with LGS-genetic were female (44% [14/32]); fewer received clobazam (44% [14/32]) or valproate (16% [5/32]); and more received levetiracetam (50% [16/32]) or rufinamide (31% [10/32])

Safety: CBD EAP Safety Analysis Set

Table 2. Summary of adverse events

	Overall (N=226)	LGS-structural (n=49)	LGS-genetic (n=32)	LGS-NOS (n=145)
Patients reporting any TEAE, n (%)	196 (86.7)	44 (89.8)	26 (81.3)	126 (86.9)
TEAEs reported in ≥10% of patients in any group, n (%)				
Somnolence	58 (25.7)	8 (16.3)	5 (15.6)	45 (31.0)
Diarrhoea	55 (24.3)	17 (34.7)	10 (31.3)	28 (19.3)
Convulsion	51 (22.6)	11 (22.4)	11 (34.4)	29 (20.0)
Upper respiratory tract infection	38 (16.8)	5 (10.2)	5 (15.6)	28 (19.3)
Pyrexia	35 (15.5)	10 (20.4)	7 (21.9)	18 (12.4)
Decreased appetite	34 (15.0)	7 (14.3)	5 (15.6)	22 (15.2)
Vomiting	30 (13.3)	5 (10.2)	7 (21.9)	18 (12.4)
Fatigue	29 (12.8)	6 (12.2)	7 (21.9)	16 (11.0)
Pneumonia	27 (11.9)	7 (14.3)	7 (21.9)	13 (9.0)
Nasopharyngitis	23 (10.2)	8 (16.3)	2 (6.3)	13 (9.0)
Decreased weight	23 (10.2)	6 (12.2)	3 (9.4)	14 (9.7)
Status epilepticus	21 (9.3)	6 (12.2)	4 (12.5)	11 (7.6)
Constipation	20 (8.8)	4 (8.2)	4 (12.5)	12 (8.3)
Ear infection	17 (7.5)	6 (12.2)	0	11 (7.6)
Aggression	17 (7.5)	3 (6.1)	4 (12.5)	10 (6.9)
Urinary tract infection	16 (7.1)	3 (6.1)	4 (12.5)	9 (6.2)
Influenza	15 (6.6)	1 (2.0)	5 (15.6)	9 (6.2)
Sedation	14 (6.2)	6 (12.2)	1 (3.1)	7 (4.8)
Insomnia	14 (6.2)	5 (10.2)	2 (6.3)	7 (4.8)
Viral gastroenteritis	10 (4.4)	1 (2.0)	4 (12.5)	5 (3.4)

LGS, Lennox-Gastaut syndrome; LGS-genetic, LGS secondary to other genetic epilepsies; LGS-NOS, LGS not otherwise specified; LGS-structural, LGS of structural aetiology; TEAE, treatment-emergent adverse event.

- Overall, 196/226 patients (86.7%) reported any TEAE
 - The most common TEAEs in the overall population were somnolence (58/226 [25.7%]), diarrhoea (55/226 [24.3%]), and convulsion (51/226 [22.6%]; **Table 2**)
- Numerical differences were observed across aetiology subgroups (eg, diarrhoea 34.7% and pyrexia 20.4% in the LGS-structural subgroup, convulsion 34.4% and diarrhoea 31.3% in the LGS-genetic subgroup); however, safety profiles were generally consistent with the overall population

Comparative analyses

Effectiveness: percent change from baseline with CBD EAP versus placebo ECA

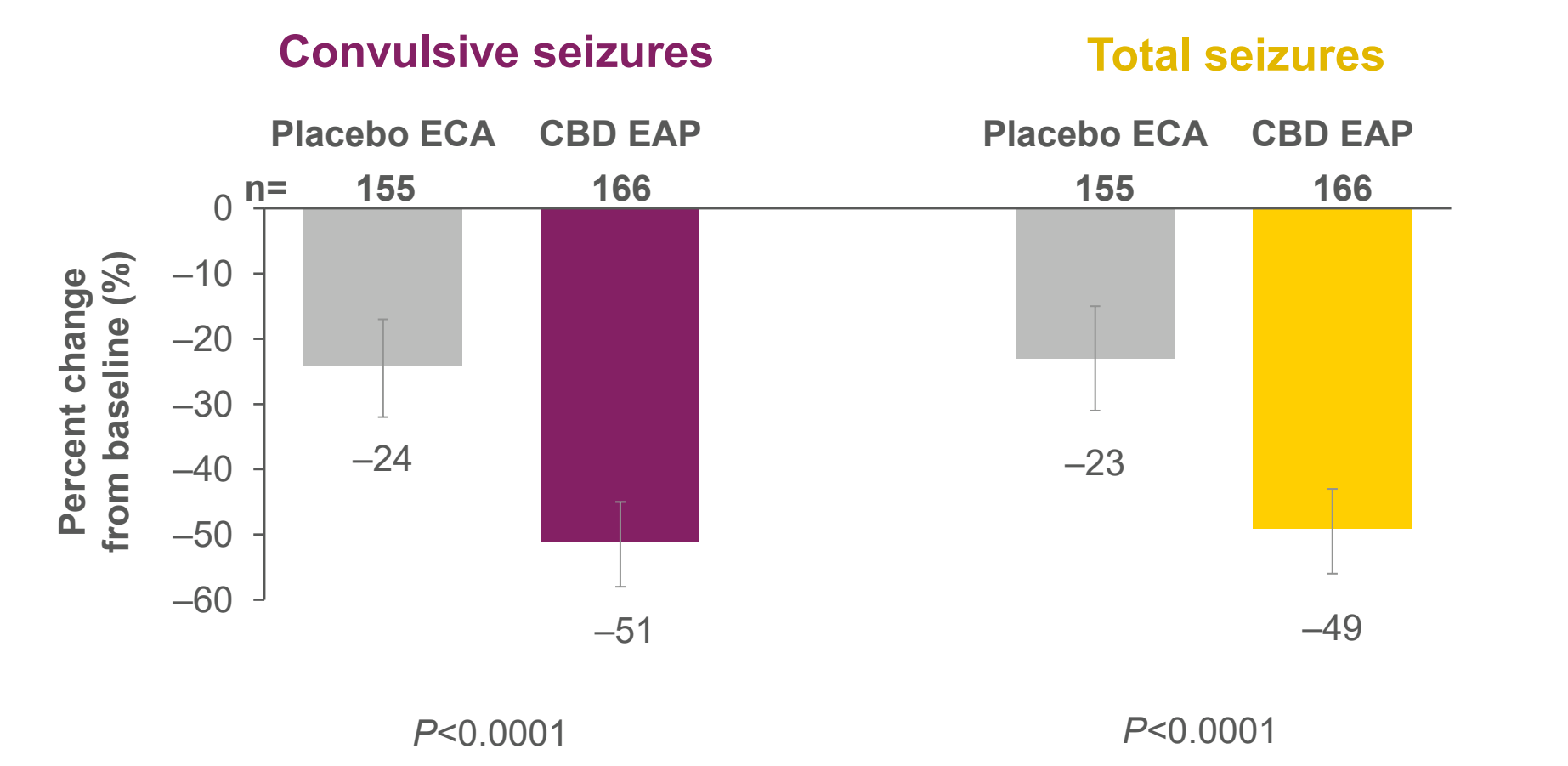
- During the 14-week treatment period, model-based least squares (LS) mean percent changes from baseline were –51% with CBD EAP versus –24% with placebo ECA for convulsive seizure frequency, and –49% versus –23%, respectively, for total seizure frequency (**Figure 5**)
- Compared with placebo ECA, CBD EAP was associated with 36% lower convulsive seizure frequency (95% confidence interval [CI]: 24, 46) and 34% lower total seizure frequency (95% CI: 21, 44) (both *P*<0.0001; **Table 3**)

Table 3. Comparative analysis of percentage reductions from baseline and treatment ratios

	Reduction from baseline, % (95% CI)	Treatment ratio, % (95% CI)	<i>P</i> -value
Convulsive seizures			
Placebo ECA	24 (16, 31)	36 (24, 46)	<0.0001
CBD EAP	51 (44, 57)		
Total seizures			
Placebo ECA	23 (15, 31)	34 (21, 44)	<0.0001
CBD EAP	49 (42, 55)		

Treatment ratios estimated from weighted log-transformed ANCOVA and calculated as the difference in the percentage change in geometric mean ratio to baseline between CBD EAP and placebo ECA arms. The placebo ECA included patients with LGS randomised to placebo in prior CBD trials. Percentage reductions from baseline and treatment ratios were calculated as (1–estimate) ×100. ANCOVA, analysis of covariance; CBD, cannabidiol; CI, confidence interval; EAP, Expanded Access Program; ECA, external control arm; LGS, Lennox-Gastaut syndrome; LS, least squares.

Figure 5. Model-based percent change from baseline for convulsive and total seizure frequencies with CBD EAP versus placebo ECA



P<0.0001 (convulsive), *P*<0.0001 (total)

Baseline characteristics: CBD EAP versus placebo ECA

Table 4. Baseline characteristics before and after inverse probability weighting: CBD EAP versus placebo ECA

	Unweighted		Weighted	
	CBD EAP (n=175)	Placebo ECA (n=161)	CBD EAP (n=175)	Placebo ECA (n=161)
Age, median (min–max)	12.8 (0–51)	12.8 (3–45)	12.8 (0–51)	12.8 (3–45)
Sex (female), n (%)	64 (36.6)	74 (46.0)	64 (41.9)	74 (42.2)
Number of ASMs taken at baseline, mean (SD)	3.1 (1.2)	2.9 (0.9)	3.0 (1.6)	3.0 (1.3)
ASM use, n (%)				
Clobazam	105 (60.0)	79 (49.1)	105 (55.2)	79 (55.0)
Levetiracetam	68 (38.9)	58 (36.0)	68 (38.1)	58 (38.4)
Valproate	47 (26.9)	63 (39.1)	47 (27.6)	63 (38.8)
Lamotrigine	45 (25.7)	56 (34.8)	45 (25.2)	56 (33.5)
Rufinamide	45 (25.7)	41 (25.5)	45 (24.1)	41 (25.4)
Topiramate	23 (13.1)	0	23 (12.8)	0
Felbamate	22 (12.6)	0	22 (12.0)	0
Stiripentol	1 (0.6)	0	1 (0.4)	0

Baseline convulsive seizure frequency				
Mean (SD)	282.9 (810.0)	219.7 (409.4)	249.2 (990.0)	243.1 (670.3)
Median	84.0	102.7	84.0	102.7
(min–max)	(8.0–8596.0)	(11.0–4058.0)	(8.0–8596.0)	(11.0–4058.0)

Baseline total seizure frequency				
Mean (SD)	455.9 (1465.5)	437.4 (713.5)	434.2 (2114.5)	465.8 (1097.8)
Median	108.0	177.7	108.0	177.7
(min–max)	(8.0–14,982.0)	(11.0–4357.0)	(8.0–14,982.0)	(11.0–4357.0)

Propensity scores were estimated using age, sex, baseline convulsive seizure frequency, number of concomitant ASMs, and current clobazam use. Total seizures include convulsive and non-convulsive seizures. ASMs, antiseizure medications; CBD, cannabidiol; EAP, Expanded Access Program; ECA, external control arm; SD, standard deviation.

- When weighted for propensity scores, baseline characteristics were balanced overall between CBD EAP and external placebo (**Table 4**)

Strengths and Limitations

- This analysis provides real-world evidence on the effectiveness and safety of CBD in patients with LGS across diverse underlying aetiologies, including structural and genetic causes
- The aetiology subgroup analyses were descriptive in nature; therefore, statistical significance cannot be inferred from comparisons between subgroups
- Although the comparative effectiveness analysis incorporated propensity score weighting to balance key baseline characteristics, residual confounding and differences between the CBD EAP and external placebo populations cannot be excluded

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PLS



AUDIO

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Supplementary Material

Figure S1. Understanding the diversity of epilepsies: a focus on various Lennox-Gastaut syndrome aetiologies in the diverse family of epilepsies

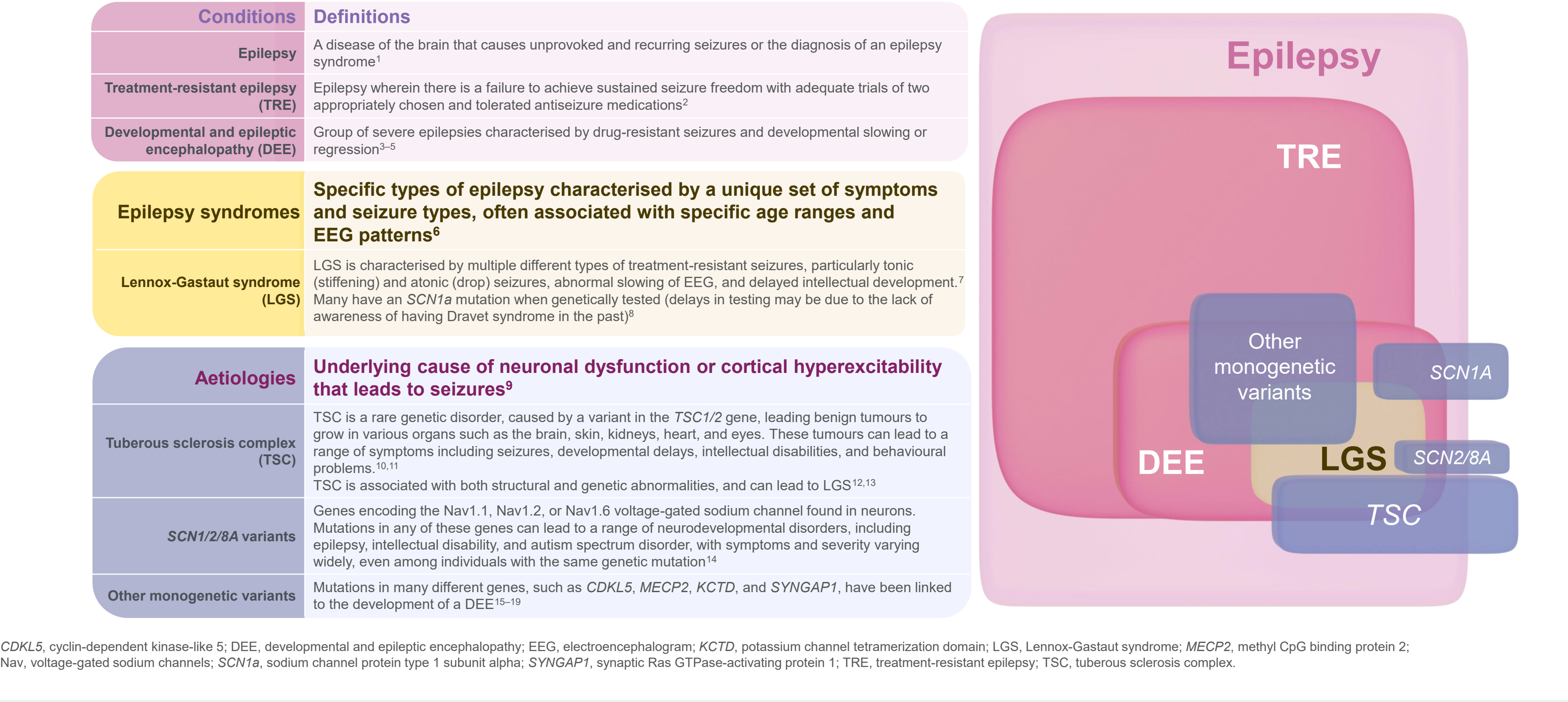


Table S1. Structural aetiology diagnoses reported by physicians among patients categorised as having LGS-structural aetiology (n=49)

	n (%)	Underlying reported aetiology ^a	n
Acquired structural injury	17 (34.7)	Epileptic encephalopathy secondary to HIE	1
		Neonatal HIE with neonatal seizures and hypoglycaemia/sepsis, cortical visual impairment	1
		Hypoxic-ischaemic encephalopathy (also reported as “HIE”)	8
		Cerebral palsy	1
		Temporal lobectomy	1
		Soto’s syndrome and hypoxic-ischaemic encephalopathy	1
		Non-paraneoplastic limbic encephalitis	1
		Perinatal stroke and Lennox-Gastaut syndrome	1
		Probable structural/metabolic hypoxic-ischaemic encephalopathy	1
		Static encephalopathy with intractable epilepsy	1
		Brain heteroplasia	1
		Congenital brain malformation with left perisylvian polymicrogyria	1
		Congenital brain malformation, band heterotopia	1
		Cortical dysplasia	3
		Epilepsy due to chromosomal encephalopathies and structural cortical malformations	1
		Heterotopia	1
Malformations of cortical development (MCD)	17 (34.7)	Hypomelanosis of Ito, periventricular heterotopias	1
		Hypomelanosis of Ito, cortical malformation, right hemisphere	1
		Lennox-Gastaut syndrome with multifocal congenital malformations	1
		Malformations of cortical or other brain development with or without known genetic determinants	1
		Microdysplasia; Lennox-Gastaut syndrome	1
		Multiple heterotopia	1
		Schizencephaly	1
		Polymicrogyria	1
		Tonic seizures with lissencephaly	1
		Congenital hydrocephalus	3
		Congenital malformation of the corpus callosum	1
		CHARGE syndrome; craniosynostosis; static encephalopathy; intractable complex partial seizures; agenesis of corpus callosum and periventricular leukomalacia	1
		Dandy-Walker malformation	1
		Microcephaly	1
		Focal epilepsy with parietal lesionectomy and <i>TSC1</i> mutation of unknown significance	1
		Brain tumour	1
Other congenital (non-MCD) malformations	7 (14.3)	Idiopathic partial epilepsy	1
		Auriculotemporal syndrome	1
Structural lesion	2 (4.1)	Complex partial seizures with secondary generalisation	1
		Partial symptomatic epilepsy	1
Unknown	6 (12.2)	Refractory symptomatic multifocal epilepsy with different seizure types	1
		Refractory multifocal epilepsy	1

^aAetiologies were reported as free-text, therefore some conditions may be the same though reported differently. CHARGE syndrome, coloboma, heart defect, atresia of the choanae, retardation of growth and development, ear abnormalities; HIE, hypoxic-ischaemic encephalopathy; LGS, Lennox-Gastaut syndrome.

Table S2. Genetic aetiology diagnoses reported by physicians among patients categorised as having LGS-genetic aetiology (n=32)

	n (%)
LGS-genetic	4 (12.5)
Aicardi syndrome	2 (6.3)
<i>DCX</i> mutation	2 (6.3)
Dup15q syndrome	2 (6.3)
13q13.3 deletion	1 (3.1)
9p21.1 deletion	1 (3.1)
Angelman’s syndrome	1 (3.1)
Angelman-like	1 (3.1)
<i>CDKL5</i>	1 (3.1)
<i>CHRNA4</i>	1 (3.1)
<i>CLCN4</i> mutation	1 (3.1)
<i>EEF1A2</i> mutation	1 (3.1)
<i>EIMFS</i> - <i>KCNT1</i> mutation	1 (3.1)
ESES	1 (3.1)
<i>GABRB3</i> variant	1 (3.1)
<i>GABRA2</i>	1 (3.1)
GLYCOSYLATION 1P	1 (3.1)
<i>GRIN2B</i> & <i>CACNA1H</i> mutations	1 (3.1)
<i>HNRNPU</i> mutation	1 (3.1)
<i>KCNQ3</i> mutation	1 (3.1)
<i>KCNT1</i> mutation	1 (3.1)
<i>PURA</i> mutation	1 (3.1)
<i>SCN2A</i> mutation	1 (3.1)
<i>SEPSECS</i> mutation	1 (3.1)
<i>SPATA5</i> mutation	1 (3.1)
Trisomy 2p25.3	1 (3.1)

Percentages are based on the CBD EAP Safety Analysis Set for patients with LGS secondary to other genetic epilepsies (n=32). CBD, cannabidiol; EAP, Expanded Access Program; LGS, Lennox-Gastaut syndrome.

Cannabidiol Effectiveness in Patients with Lennox-Gastaut Syndrome of Diverse Aetiologies Compared with External Placebo Control: A *Post Hoc* Analysis from the US Expanded Access Program

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What is Lennox-Gastaut syndrome (LGS)?

- LGS is a serious brain disorder that causes seizures and problems with how a child learns and grows
 - Medicines for LGS are available, but many children and adults who are being treated with them still experience frequent seizures¹
- LGS can have different causes, including gene changes, structural brain changes, metabolic disorders, or causes that are unknown^{2,3}



What is cannabidiol (CBD)?

- A plant-derived, highly purified pharmaceutical formulation of CBD (Epidyolex® [EU]/Epidiolex® [US])⁴ is approved for the treatment of seizures associated with certain rare forms of epilepsy, including LGS^{4,5}
- Previous clinical trials have shown that CBD can help reduce seizures in people with LGS^{6,7}
 - However, less is known about whether the effectiveness of CBD changes depending on the underlying cause of LGS

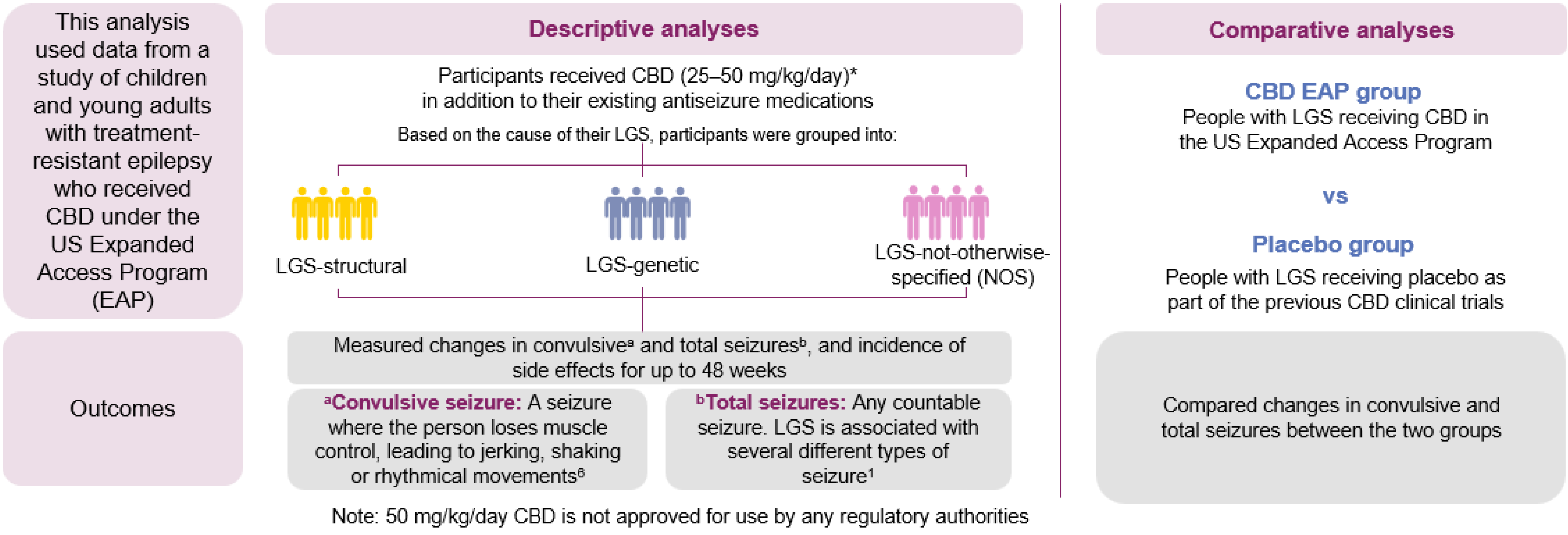


Why was this analysis done?

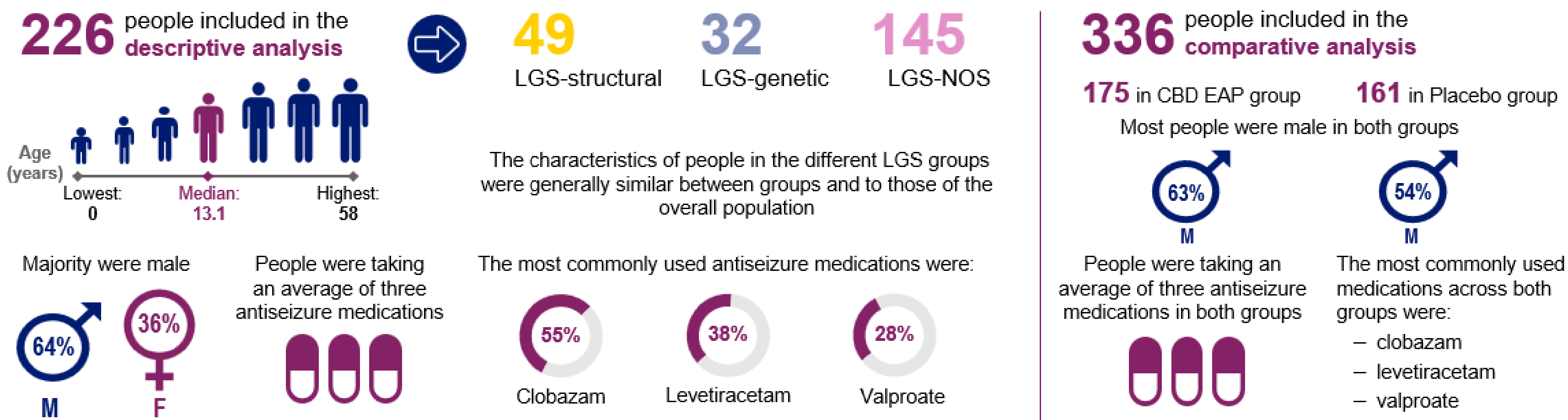
- This analysis was carried out to determine whether the effect of CBD treatment varied in people with different underlying causes of LGS and whether any differences in side effects were seen with CBD. The comparative effectiveness analysis versus an external control arm was used to indicate the extent of reduction in seizures with CBD when compared with an external placebo arm



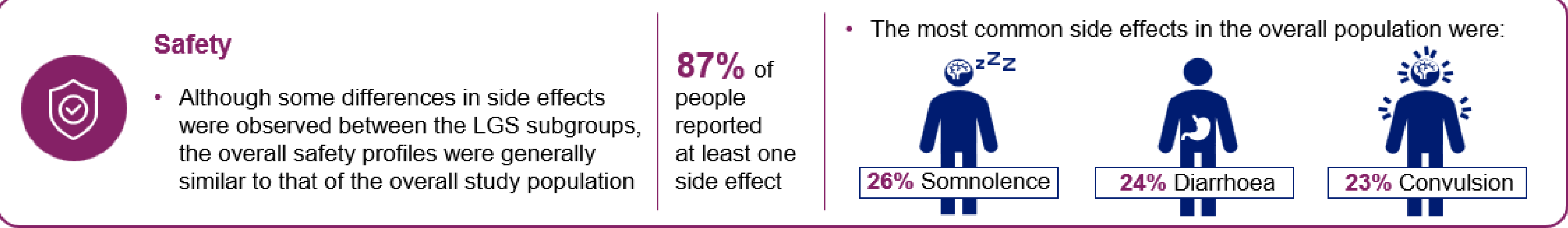
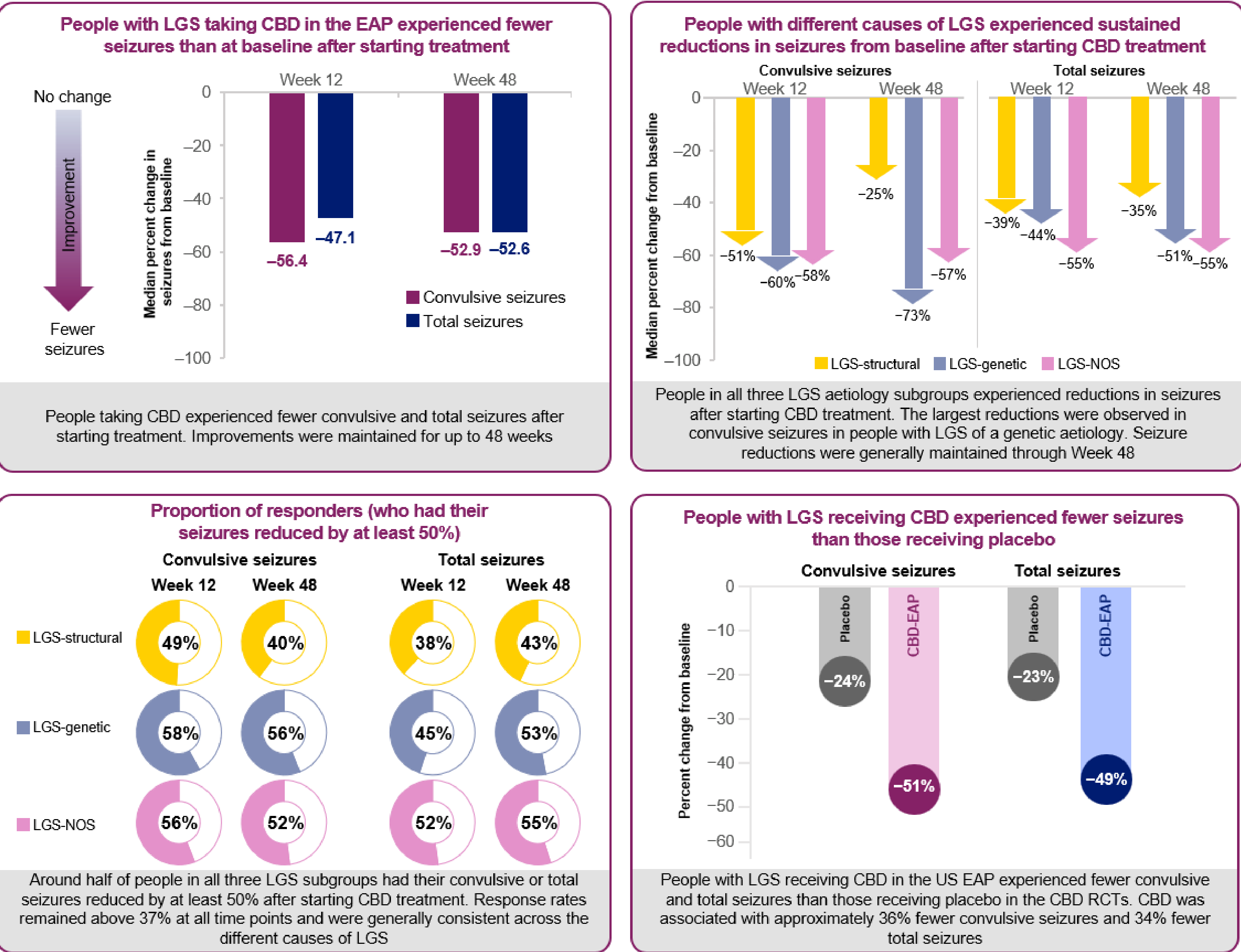
How was this study designed?



What were the key characteristics of the people included in this analysis?



What were the key results from this analysis?



What do these results mean?

- CBD treatment was associated with fewer convulsive and total seizures compared with baseline, in people with different causes of LGS
- A comparison of those with LGS receiving CBD in the US EAP with people with LGS receiving placebo in the previous LGS CBD clinical trials suggested that CBD reduced the frequency of seizures
- The safety profile of CBD was similar to that reported in previous CBD clinical trials and was generally consistent across people with different causes of LGS
- Overall, these findings agree with previous clinical trial data that indicate response to CBD treatment in patients with LGS is not influenced by differing underlying causes

*Epidyolex® is approved in the EU for the adjunctive treatment of seizures associated with Lennox-Gastaut syndrome or Dravet syndrome, in conjunction with clobazam, in patients ≥2 years of age; it is additionally approved in the EU for the adjunctive treatment of seizures associated with tuberous sclerosis complex in patients ≥2 years of age.

¹Specchio N, et al. *Epilepsia*. 2022;63(6):1398–1442. 2. Na J-H, Lee Y-M. *Int J Mol Sci*. 2026;27(3):1382. 3. Amrutkar CV, Lui F. Lennox-Gastaut Syndrome. In StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. 4. European Medicines Agency. Epidyolex® 100 mg/ml oral solution: summary of product characteristics. 2026. Available from: https://www.ema.europa.eu/en/documents/product-information/epidyolex-epar-product-information_en.pdf (Accessed 19 August 2026). 5. US Food and Drug Administration. Epidiolex® Prescribing Information; 2026. Available from: https://www.accessdata.fda.gov/drugsatfda_docs/label/2026/210365s024s029s030lbl.pdf (Accessed 19 August 2026). 6. Devinsky O, et al. *N Engl J Med*. 2018;378(20):1888–1897. 7. Thiele EA, et al. *Lancet*. 2018;391(10125):1085–1096.



POSTER



AUDIO

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