Seladelpar for Alkaline Phosphatase Normalization in Primary Biliary Cholangitis: Results from the IDEAL Phase III Randomized-Controlled Trial

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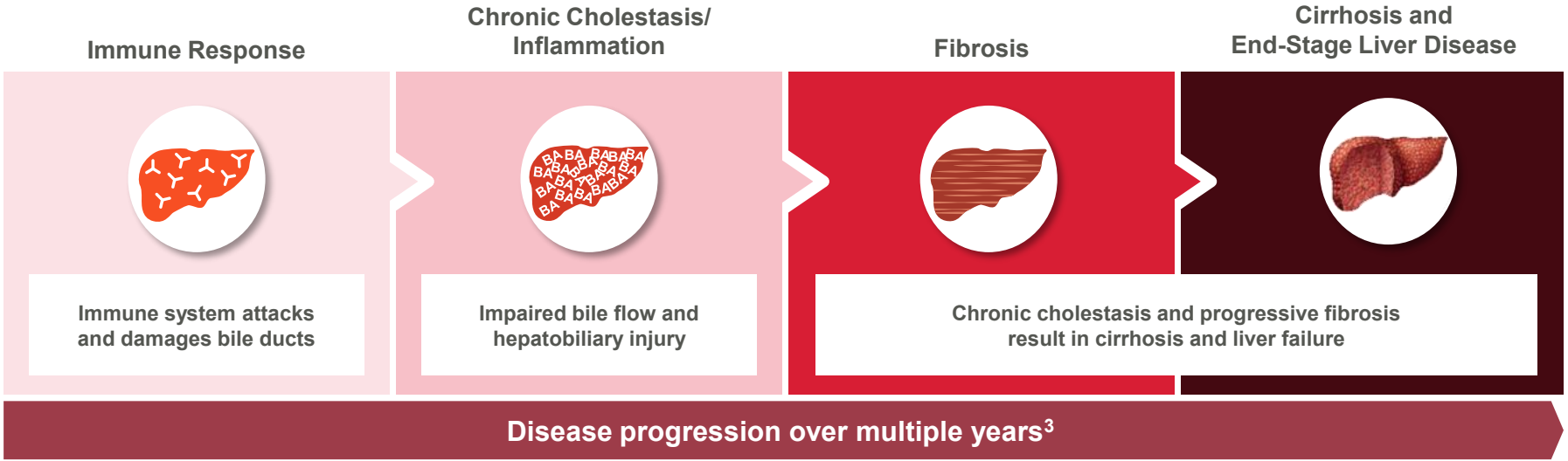
The Liver Meeting, American Association for the Study of Liver Diseases; November 5–9, 2026; Denver, CO, USA

Author Disclosures

Cynthia Levy reports receiving research grants paid to her institution from Calliditas Therapeutics, CymaBay Therapeutics, Escient Pharmaceuticals, Gilead Sciences, Inc., GSK, Intercept Pharmaceuticals, Ipsen, Kowa, Mirum Pharma, Pliant Therapeutics, Target RWE, and Zydus Pharmaceuticals.

Primary Biliary Cholangitis

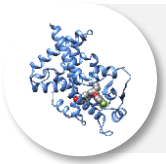
- PBC is a chronic, progressive, autoimmune, cholestatic liver disease that affects approximately 1 in 1000 women over 40 years of age[1]
- Key markers of PBC include ALP, which reflects cholestasis, and total bilirubin as an indicator of disease severity[1]
- First-line therapy is UDCA 13-15 mg/kg; many patients have an inadequate response[1,2]



ALP, alkaline phosphatase; **PBC**, primary biliary cholangitis; **UDCA**, ursodeoxycholic acid.

1. European Association for the Study of the Liver. *J Hepatol*. 2017;67:145-72. **2.** Levy C, et al. *Hepatology*. 2026. Epub ahead of print. **3.** Selmi C, et al. *Lancet*. 2011;377:1600-9.

Seladelpar: PPARδ Agonist



Seladelpar is an approved first-in-class **delpar (selective PPARδ agonist)** indicated for treatment of PBC in combination with UDCA in adults who have an inadequate response to UDCA, or as a monotherapy in patients who are unable to tolerate UDCA[1–4]

Hepatocytes & Cholangiocytes	Macrophages & Kupffer Cells	Hepatocytes	Hepatocytes
Improves cholestasis	**Reduces inflammation**	**Reduces pruritus**	**Increases lipid metabolism**
↓ Bile acid synthesis[5–8]	↓ Inflammatory cytokines[8]	↓ Bile acids[6]	↓ Total cholesterol, LDL, triglycerides[5,8,11]
↓ ALP[5]	↓ Inflammatory lipid mediators[9]	↓ IL-31[10,a]	↑ Fatty acid oxidation[8,9]
↓ GGT[5]	↓ ALT[5]		

Seladelpar is a selective PPARδ agonist with anticholestatic, anti-inflammatory, and antipruritic effects[1-12]

Seladelpar X-ray crystal structure adapted from Choi Y, et al. 2021.[8] [a]Although the mechanism of pruritus in PBC is yet to be fully elucidated, reductions in IL-31 may be related to pruritus improvement, which was observed in the ENHANCE study.[10]

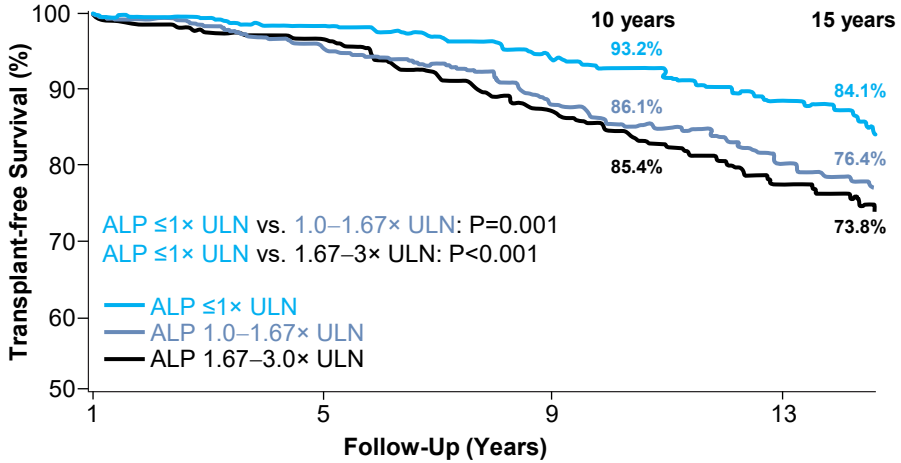
ALP, alkaline phosphatase; **ALT**, alanine aminotransferase; **GGT**, gamma-glutamyl transferase; **IL-31**, interleukin-31; **LDL**, low-density lipoprotein; **PBC**, primary biliary cholangitis; **PPARδ**, peroxisome proliferator–activated receptor delta; **UDCA**, ursodeoxycholic acid.

1. Hirschfield GM, et al. *N Engl J Med.* 2024;390:783-94. **2.** Livdelzi. US Prescribing Information. Gilead Sciences, Inc.; 2024. **3.** Livdelzi. UK Summary of Product Characteristics. Gilead Sciences, Inc.; 2026. **4.** Lyvdelzi. EU Summary of Product Characteristics. Gilead Sciences, Inc. 2026. **5.** Hirschfield GM, et al. *Hepatology.* 2023;78:397-415. **6.** Kremer AE, et al. *Liver Int.* 2022;42:112-23. **7.** Kouno T, et al. *J Biol Chem.* 2022;298:102056. **8.** Choi Y, et al. Discovery on Target 2021. Oral presentation. **9.** Choi Y, et al. AASLD 2022. Poster 4731. **10.** Kremer AE, et al. *Hepatology.* 2024;80:27-37. **11.** Bowlus C, et al. AASLD 2022. Poster 4759. **12.** Hirschfield GM, et al. AASLD 2023. Oral presentation 5002.

IDEAL Study Background

- Registrational trials for second-line PBC therapies have consistently evaluated patients with ALP ≥1.67× ULN[1-3]
- Patients with ALP elevations <1.67× ULN are at risk of serious outcomes, including liver transplant and death, and are an understudied population[4-7]
- Recent professional society consensus includes ALP ≤ULN as part of the ideal treatment response in PBC, with an emphasis on patients with liver stiffness ≥10 kPa or those aged ≤60 years[8-10]

Survival Estimate by ALP Level[4]

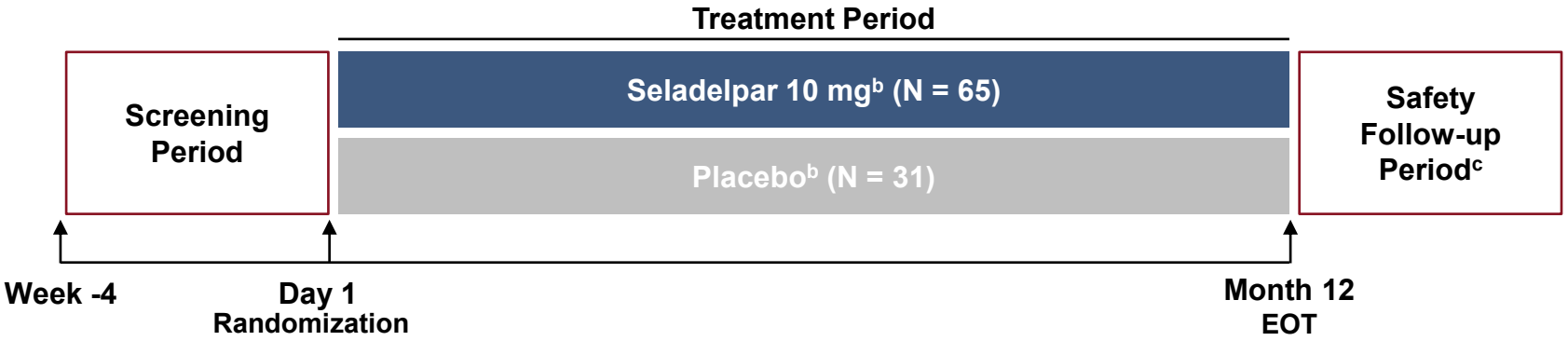


ALP, alkaline phosphatase; **PBC**, primary biliary cholangitis; **UDCA,** ursodeoxycholic acid; **ULN**, upper limit of normal.

1. Nevens F, et al. *N Engl J Med.* 2016;375:631-43. **2.** Kowdley KV, et al. *N Engl J Med.* 2024;390:795-805. **3.** Hirschfield GM, et al. *N Engl J Med.* 2024;390:783-94. **4.** Murillo Perez CF, et al. *Am J Gastroenterol.* 2020;115:1066-74. **5.** Reddy A, et al. *Am J Gastroenterol.* 2026; (Online Ahead of Print). **6.** Jones DEJ, et al. *EBioMedicine*. 2022;80:104068. **7.** Corpechot C, et al. *Hepatology*. 2024;79:39-48. **8.** EASL and AASLD. *J Hepatol,* 2026;85:583-602. **9.** AISF. *Dig Liver Dis.* 2026; (Online Ahead of Print). **10.** Levy C, et al. *Hepatology.* 2026. Epub ahead of print.

IDEAL: Phase 3 Study in Patients With PBC

PATIENT POPULATION[a]: PBC with ALP between 1–1.67× ULN, despite UDCA treatment or intolerance



PRIMARY ENDPOINTS[d]: ALP normalization defined as the composite of ALP ≤ULN and ≥15% decrease from baseline at Month 12; safety

SECONDARY ENDPOINTS[d]: ALP normalization composite, ALP ≤ULN, and change in ALP over 12 months

NCT06060665. [a]The patient population included patients with ALT and AST ≤5× ULN and total bilirubin ≤2× ULN. [b]Study drug was administered orally once daily as an add-on to UDCA or as monotherapy in patients intolerant to UDCA. Patients were stratified by ALP <1.3× ULN vs. ≥1.3× ULN and pruritus NRS <4 vs. NRS ≥4. [c]Safety follow-up visit occurred 2 weeks (+3 days) after the last dose of trial drug, including those who discontinued treatment early. [d]Only the primary endpoint was controlled for type I error; all P values except for the primary endpoint are considered nominal.
ALP, alkaline phosphatase; **ALT**, alanine aminotransferase; **AST**, aspartate aminotransferase; **EOT**, end of treatment; **NRS**, numerical rating scale; **PBC**, primary biliary cholangitis; **UDCA**, ursodeoxycholic acid; **ULN**, upper limit of normal.

Baseline Demographic and Disease Characteristics

		Seladelpar 10 mg N = 65	Placebo N = 31
Age, years, mean (SD)		61 (8.1)	60 (9.3)
Female, n (%)		61 (93.8)	29 (93.5)
Race or ethnic group, n (%)[a]			
White		58 (89.2)	25 (80.6)
Black or African American		3 (4.6)	3 (9.7)
Asian		3 (4.6)	0
Hispanic/Latinx[b]		9 (13.8)	6 (19.4)
On ursodeoxycholic acid, n (%)		62 (95.4)	30 (96.8)
ALP, U/L, mean (SD)	ULN: 116 U/L	154 (22.7)	150 (22.9)
Total bilirubin, mg/dL, mean (SD)	ULN: 1.10 mg/dL	0.61 (0.19)	0.62 (0.22)
>0.6× ULN, n (%)		19 (29.2)	11 (35.5)
ALT, U/L, mean (SD)	ULN: 41 U/L	28 (14.8)	27 (13.8)
GGT, U/L, mean (SD)	ULN: 52 U/L men; 38 U/L women	76 (75.5)	75 (50.0)
Liver stiffness, kPa, mean (SD)		6.3 (2.3)	7.8 (3.5)
Pruritus NRS score, mean (SD)		2.2 (2.3)	1.8 (2.4)
Pruritus NRS ≥4 (moderate to severe), n (%)		14 (21.5)	5 (16.1)
≥1 risk factor for disease progression, n (%)[c]		38 (58.5)	19 (61.3)

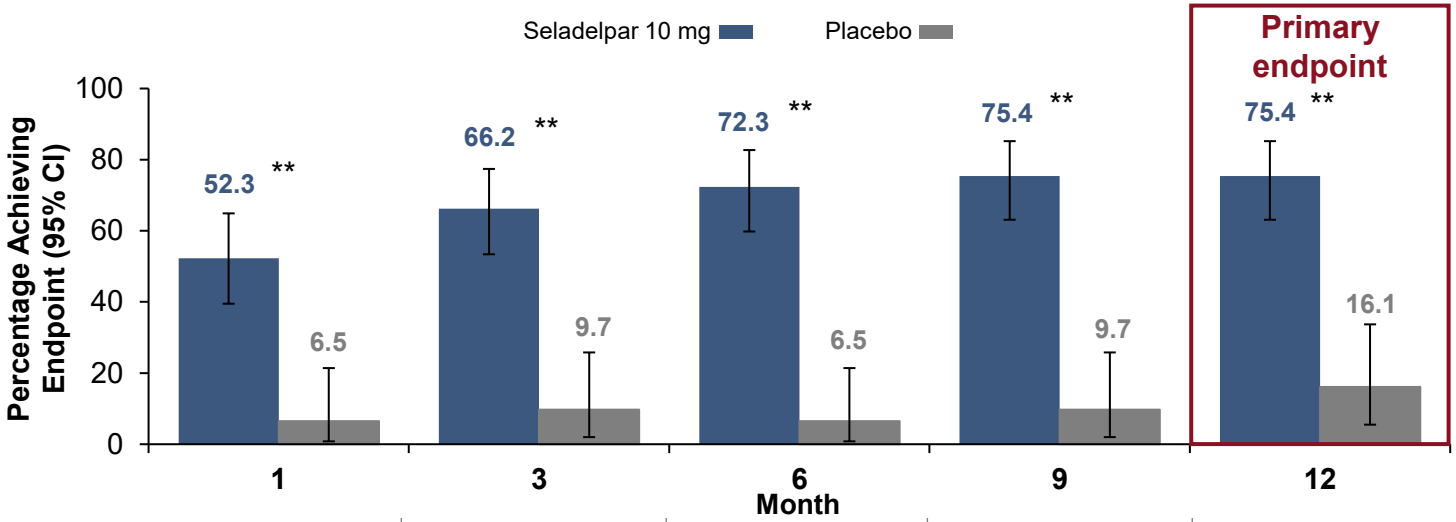
[a]Patients could report multiple race or ethnicity categories; therefore, percentages do not sum to 100. Race was reported as "Other" for 1 patient in the placebo group; race was not reported in 1 (1.5%) and 2 (6.5%) patients in the seladelpar and placebo groups, respectively. [b]Ethnicity was not reported in 3 (4.6%) and 1 (3.2%) patients in the seladelpar and placebo groups, respectively. [c]Identified post-hoc and defined as meeting at least one of the following categories at baseline: male sex, age <50 years at PBC diagnosis, liver stiffness ≥10 kPa, and/or total bilirubin >0.6× ULN.

ALP, alkaline phosphatase; **ALT,** alanine aminotransferase; **GGT,** gamma-glutamyl transferase; **NRS**, numeric rating scale; **PBC,** primary biliary cholangitis; **SD,** standard deviation; **ULN**, upper limit of normal.

Primary Endpoint of ALP Normalization Achieved With Seladelpar Treatment

ALP Normalization

ALP ≤ULN with ALP decrease ≥15% from baseline



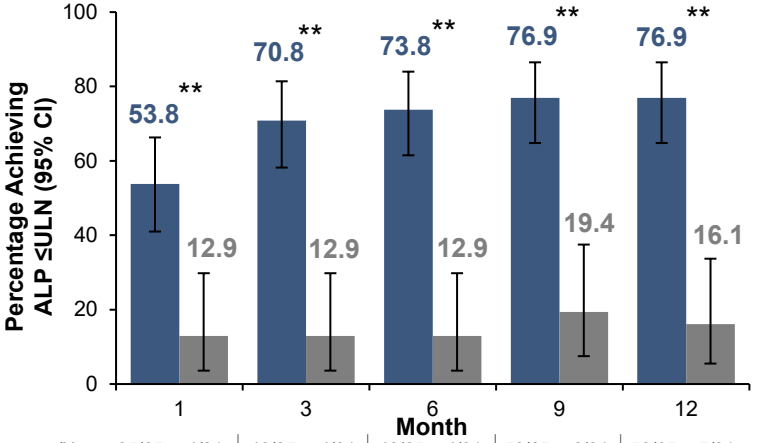
	1		3		6		9		12	
n/N =	34/65	2/31	43/65	3/31	47/65	2/31	49/65	3/31	49/65	5/31
Treatment difference, % (95% CI)	47.9 (32.2 to 63.6)		58.5 (42.7 to 74.4)		67.4 (52.6 to 82.1)		67.0 (51.6 to 82.3)		61.3 (44.9 to 77.7)	

**P<0.0001 vs. placebo. A two-sided P value was provided for the primary endpoint based on the Cochran-Mantel-Haenszel test stratified by the baseline ALP level (ALP <1.3 x ULN versus ALP ≥1.3 x ULN), and all other P values are nominal. Treatment differences are stratum-adjusted.

ALP, alkaline phosphatase; **CI**, confidence interval; **n/N,** number of responders/patients in full analysis set; **ULN**, upper limit of normal.

Reduction in ALP Levels With Seladelpar Treatment

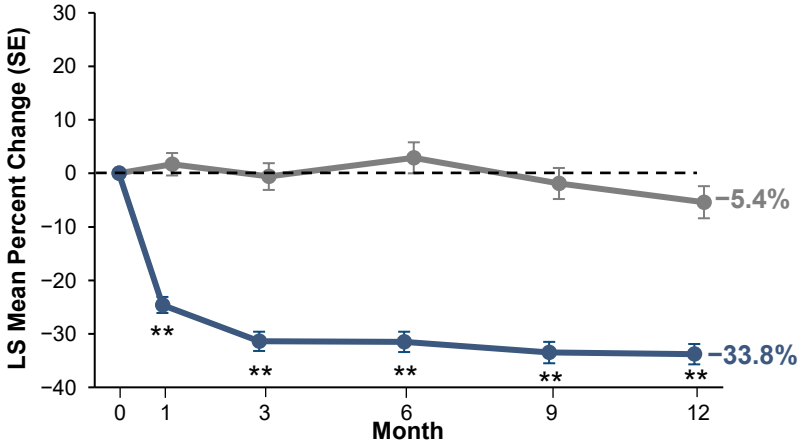
ALP ≤ULN[a]



n/N =	35/65	4/31	46/65	4/31	48/65	4/31	50/65	6/31	50/65	5/31
Treatment difference, % (95% CI)	43.6 (26.6 to 60.5)		60.8 (45.0 to 76.6)		63.0 (47.1 to 79.0)		59.6 (42.9 to 76.3)		63.1 (46.9 to 79.2)	

Seladelpar 10 mg

Percent Change From Baseline in ALP[b]



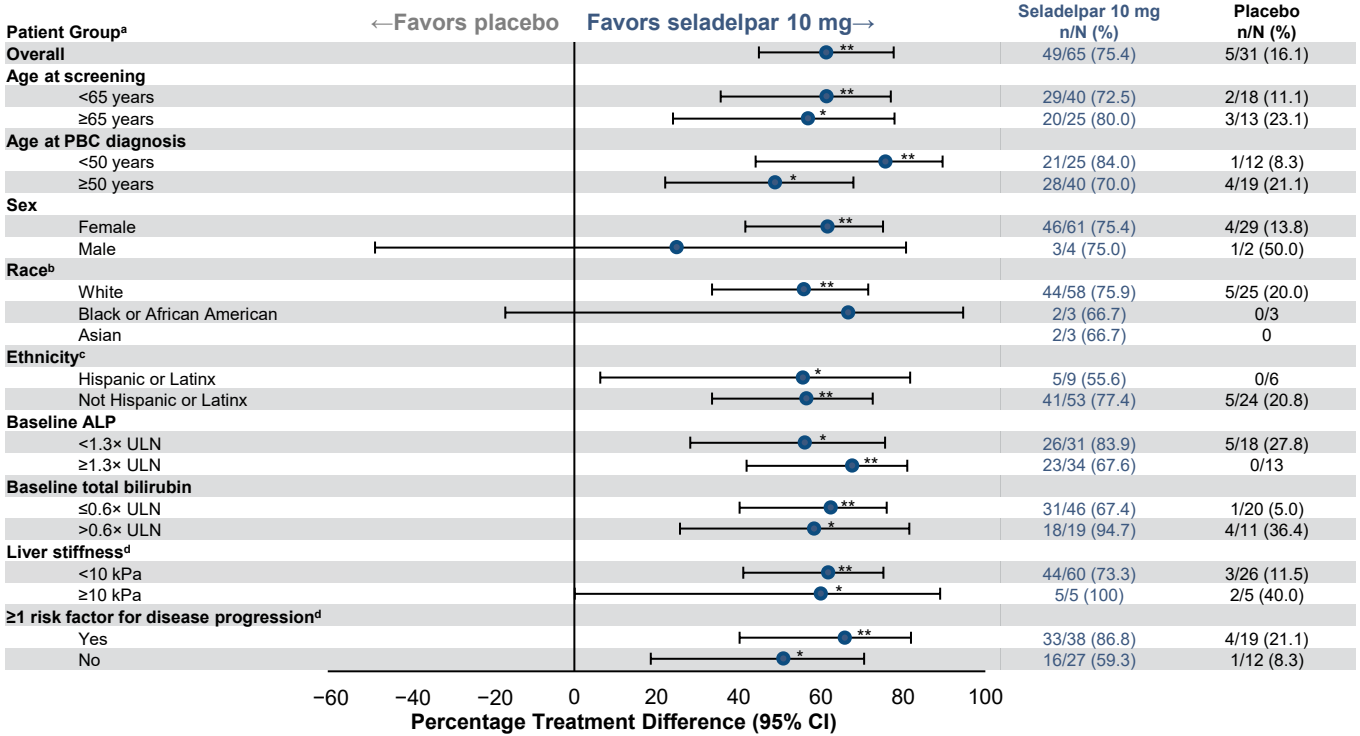
Seladelpar n =	65	62	63	63	61	62
Placebo n =	31	30	30	28	27	26

Placebo

**P<0.0001 vs. placebo. Two-sided P values were based on the Cochran-Mantel-Haenszel test and are nominal.
[a]ALP ULN defined as 116 U/L for women and men. [b]Post-baseline percent changes were assessed using the mixed-effects model for repeated measures with adjustment for baseline ALP, treatment group, visit, and treatment-by-visit interaction as fixed effects.
ALP, alkaline phosphatase; **CI**, confidence interval; **LS**, least-squares; **n,** number of evaluable patients at each time point; **n/N,** number of responders/patients in full analysis set; **SE,** standard error; **ULN**, upper limit of normal.

ALP Normalization With Seladelpar Across All Subgroups at Month 12

ALP ≤ULN with ALP decrease ≥15% from baseline

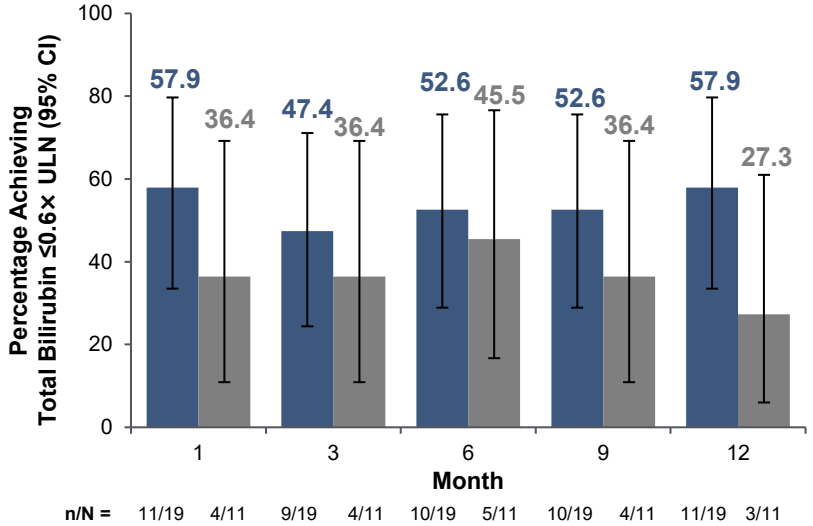


Patient Group[a]	Seladelpar 10 mg n/N (%)	Placebo n/N (%)
Overall	49/65 (75.4)	5/31 (16.1)
Age at screening		
<65 years	29/40 (72.5)	2/18 (11.1)
≥65 years	20/25 (80.0)	3/13 (23.1)
Age at PBC diagnosis		
<50 years	21/25 (84.0)	1/12 (8.3)
≥50 years	28/40 (70.0)	4/19 (21.1)
Sex		
Female	46/61 (75.4)	4/29 (13.8)
Male	3/4 (75.0)	1/2 (50.0)
Race[b]		
White	44/58 (75.9)	5/25 (20.0)
Black or African American	2/3 (66.7)	0/3
Asian	2/3 (66.7)	0
Ethnicity[c]		
Hispanic or Latinx	5/9 (55.6)	0/6
Not Hispanic or Latinx	41/53 (77.4)	5/24 (20.8)
Baseline ALP		
<1.3× ULN	26/31 (83.9)	5/18 (27.8)
≥1.3× ULN	23/34 (67.6)	0/13
Baseline total bilirubin		
≤0.6× ULN	31/46 (67.4)	1/20 (5.0)
>0.6× ULN	18/19 (94.7)	4/11 (36.4)
Liver stiffness[d]		
<10 kPa	44/60 (73.3)	3/26 (11.5)
≥10 kPa	5/5 (100)	2/5 (40.0)
≥1 risk factor for disease progression[d]		
Yes	33/38 (86.8)	4/19 (21.1)
No	16/27 (59.3)	1/12 (8.3)

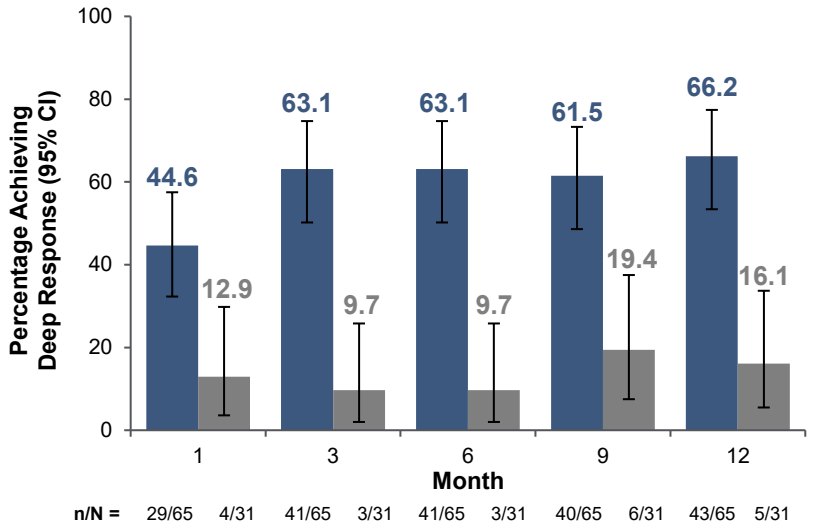
*P≤0.05 vs. placebo. **P<0.0001 vs. placebo. [a]P values for subgroups were based on the Miettinen-Nurminen method and are nominal. [b]One patient in the seladelpar group with race not reported achieved the primary endpoint. In the placebo group, two patients did not report their race, and one reported their race as Other. Risk differences for the Asian race subgroup were not estimable. [c]Three patients in the seladelpar group and one in the placebo group did not report their ethnicity. [d]Analyses of patients by liver stiffness (≥10 kPa, <10 kPa) and risk of disease progression (high risk [defined as age <50 years at PBC diagnosis, total bilirubin >0.6× ULN, liver stiffness ≥10 kPa, and/or male sex], not high risk) were performed post-hoc.
ALP, alkaline phosphatase; **CI**, confidence interval; **n/N,** number of responders/patients in full analysis set; **PBC**, primary biliary cholangitis; **ULN**, upper limit of normal.

Normalization of Liver Markers with Seladelpar Treatment

Total Bilirubin ≤0.6× ULN[a]



Deep Response[b]

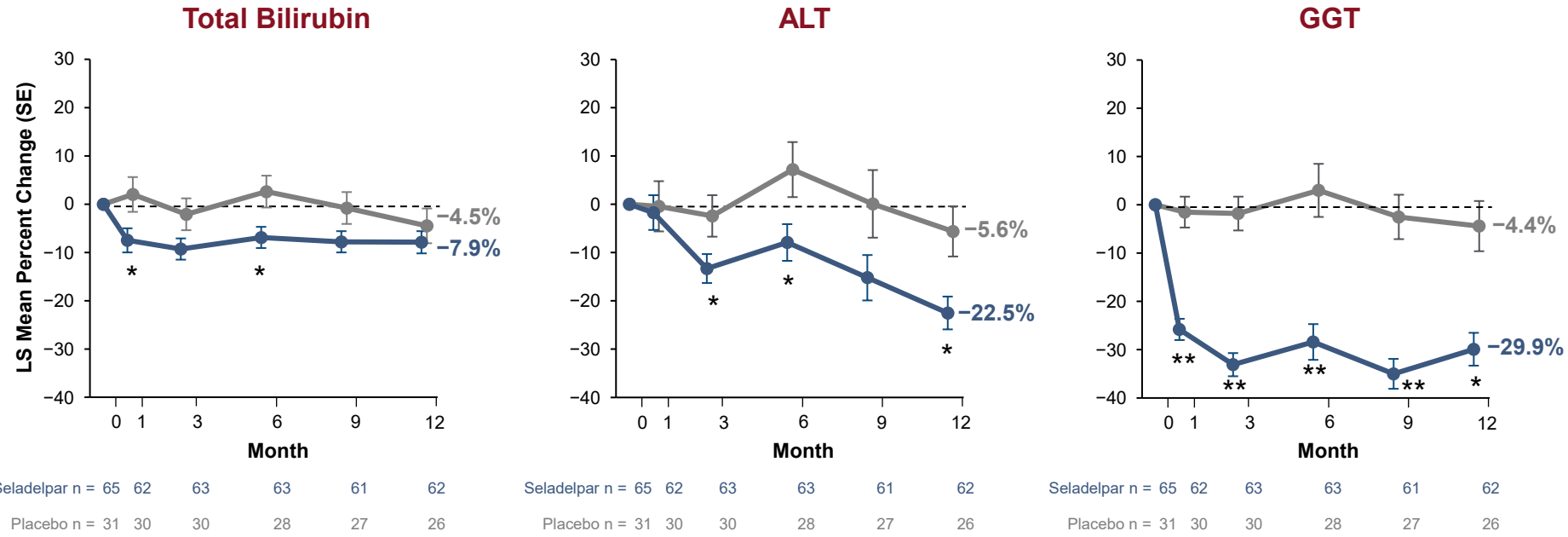


Seladelpar 10 mg ■ Placebo ■

[a]Percentages of patients with baseline total bilirubin >0.6× ULN achieving total bilirubin ≤0.6× ULN through Month 12. The reference range for total bilirubin is 0.10–1.10 mg/dL. n/N is defined as the number of responders/patients in the full analysis set with total bilirubin >0.6× ULN at baseline. [b]Deep response was defined as normal (≤ULN) ALP, total bilirubin, ALT, and AST. Reference ranges for ALP, total bilirubin, ALT, and AST are 37–116 U/L, 0.10–1.10 mg/dL, 6–41 U/L, and 9–34 U/L, respectively. n/N is defined as the number of responders/patients in the full analysis set.

ALP, alkaline phosphatase; **ALT,** alanine aminotransferase; **AST,** aspartate aminotransferase; **CI**, confidence interval; **GGT**, gamma-glutamyl transferase; **ULN**, upper limit of normal.

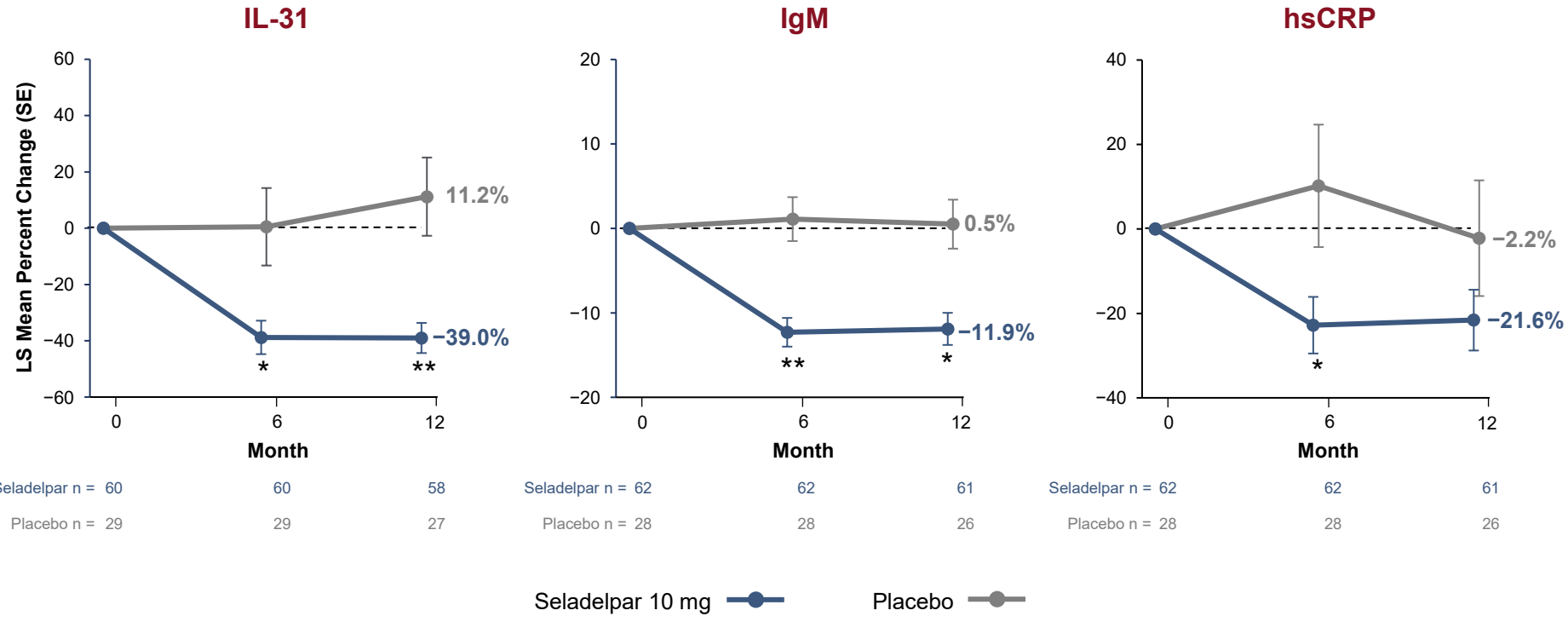
Reductions in Total Bilirubin, ALT, and GGT With Seladelpar Treatment



*P≤0.05 vs. placebo. **P<0.0001 vs. placebo. Post-baseline percent changes were assessed using the MMRM. Nominal P values ≤0.05 are designated at all applicable timepoints. Reference range for total bilirubin is 0.1–1.1 mg/dL; for ALT is 6–41 U/L; and for GGT is 7–38 U/L in women and 11–52 U/L in men.

ALT, alanine aminotransferase; **GGT**, gamma-glutamyl transferase; **LS**, least-squares; **MMRM,** mixed-effects model for repeated measures; **n,** number of evaluable patients at each time point; **SE,** standard error.

Decreases in Immune and Inflammatory Markers With Seladelpar Treatment



*P≤0.05 vs. placebo. **P<0.0001 vs. placebo. The reference ranges for IgM and hsCRP are 40–230 mg/dL and ≤3.0 mg/L, respectively. Post-baseline percent changes were assessed using the MMRM, applied to either raw (IgM) or log-transformed (IL-31 and hsCRP) values based on normality assumptions.
hsCRP, high-sensitivity C-reactive protein; **Ig,** immunoglobulin; **IL,** interleukin; **LS,** least-squares; **MMRM,** mixed-effects model for repeated measures; **n,** number of evaluable patients at each time point; **SE,** standard error.

Similar Overall Safety Profile Between Seladelpar and Placebo

n (%)	Seladelpar 10 mg N = 65	Placebo N = 31
Any AE	51 (78.5)	26 (83.9)
Serious AEs[a]	2 (3.1)	4 (12.9)
Treatment-related serious AEs	0	0
Grade ≥3 AE	3 (4.6)	4 (12.9)
AE leading to treatment discontinuation	1 (1.5)	2 (6.5)
AE leading to death	0	0
Liver-related AEs	0	1 (3.2)
Muscle-related AEs	3 (4.6)	1 (3.2)
Renal-related AEs	0	0

Severity graded by the National Cancer Institute Common Terminology Criteria for Adverse Events (v5.0). All AEs were treatment-emergent, defined as occurring from the first dose of the trial drug up to 30 days after the last dose of trial drug. [a]Serious AEs for seladelpar included metastatic breast cancer (which led to treatment discontinuation) and neutropenia; and for placebo included unstable angina, hypomagnesemia, squamous cell carcinoma of the lung (which led to treatment discontinuation), and transient global amnesia.
AE, adverse event; **n,** number of patients in the category.

Similar Overall Incidence of Adverse Events Between Seladelpar and Placebo

n (%)	Seladelpar 10 mg N = 65	Placebo N = 31
Any AE	51 (78.5)	26 (83.9)
AEs in ≥5% of patients overall[a]		
Influenza	1 (1.5)	5 (16.1)
Arthralgia	9 (13.8)	2 (6.5)
Constipation	5 (7.7)	4 (12.9)
Pruritus	8 (12.3)	3 (9.7)
Fatigue	7 (10.8)	1 (3.2)
Rash	3 (4.6)	3 (9.7)
COVID-19	6 (9.2)	2 (6.5)
Dry eye	4 (6.2)	2 (6.5)
Sinusitis	4 (6.2)	2 (6.5)
Dry mouth	3 (4.6)	2 (6.5)

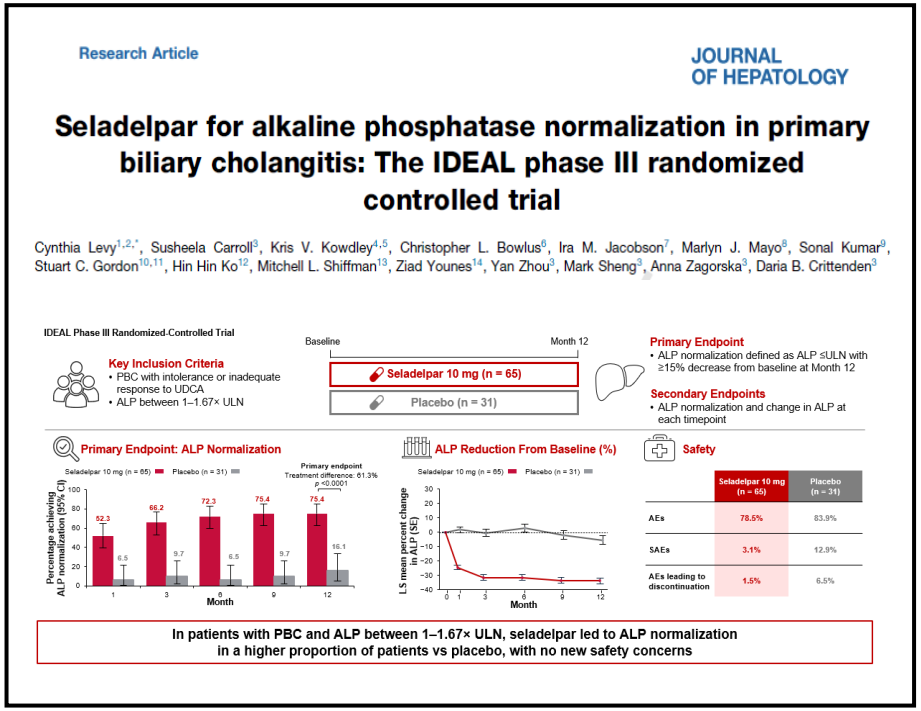
AEs were summarized according to preferred terms in the Medical Dictionary for Regulatory Activities (v28.1), with severity graded by the National Cancer Institute Common Terminology Criteria for Adverse Events (v5.0). All AEs were treatment-emergent, defined as occurring from the first dose of the trial drug up to 30 days after the last dose of trial drug. Multiple AEs were counted only once per patient for each preferred term. [a]Urinary tract infections occurred in 4 (6.2%) patients receiving seladelpar; none occurred in patients receiving placebo.
AE, adverse event; **COVID-19**, coronavirus disease 2019; **n,** number of patients in the category.

Conclusions

- Seladelpar led to a high proportion of patients achieving ALP normalization compared to placebo
  - Approximately 3 in 4 patients receiving seladelpar met the primary endpoint. The treatment difference between seladelpar and placebo was 61.3%
  - Effects with seladelpar were consistent across all evaluated subgroups, including those with features known to increase risk for disease progression
- Seladelpar led to rapid and sustained reductions in ALP
- Seladelpar treatment reduced GGT, liver injury marker ALT, pruritogenic cytokine IL-31, and markers of inflammation (hsCRP and IgM)
- Seladelpar demonstrated a safety profile generally similar to placebo, and no new safety concerns were identified

ALP, alkaline phosphatase; **ALT,** alanine aminotransferase; **GGT**, gamma-glutamyl transferase; **hsCRP**, high-sensitivity C-reactive protein; **Ig**, immunoglobulin; **IL,** interleukin.

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Research Article

JOURNAL OF HEPATOLOGY

Seladelpar for alkaline phosphatase normalization in primary biliary cholangitis: The IDEAL phase III randomized controlled trial

Cynthia Levy[1,2,*], Susheela Carroll[3], Kris V. Kowdley[4,5], Christopher L. Bowlus[6], Ira M. Jacobson[7], Marlyn J. Mayo[8], Sonal Kumar[9], Stuart C. Gordon[10,11], Hin Hin Ko[12], Mitchell L. Shiffman[13], Ziad Younes[14], Yan Zhou[3], Mark Sheng[3], Anna Zagorska[3], Daria B. Crittenden[3]

DOI: 10.1016/j.jhep.2026.10.021

Acknowledgements

- We extend our thanks to the patients, their families, and all participating investigators, as well as Peyvand Amini, MD, PhD, for her contributions to trial oversight and data interpretation; Robert Kustra, PharmD, for his insights and analysis of the IDEAL safety data; and Jayamary Divya Ravichandar, PhD, and Yan Liang, PhD, for their insights, analysis, and review of the IDEAL biomarker data.
- This study was funded by Gilead Sciences, Inc.
- All authors contributed to and approved the presentation; medical writing support was provided by Denise Kenski, PhD, CMPP of Gilead Sciences, Inc., and Sophie Dorf-Kamienny, BA, and Yasha Najafi, MSc, from Costello Medical, and was funded by Gilead Sciences, Inc.

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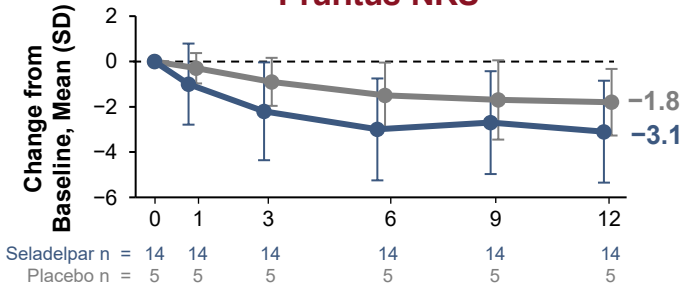
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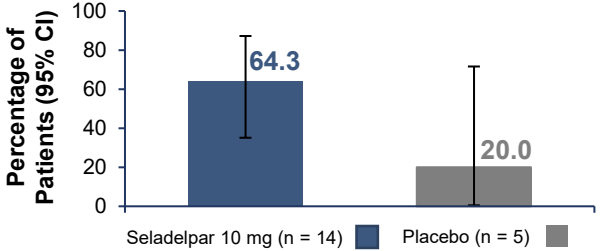
BACKUP

Decreases in Pruritus and Fatigue Scores in Patients With Moderate to Severe Pruritus (NRS ≥4) at Baseline

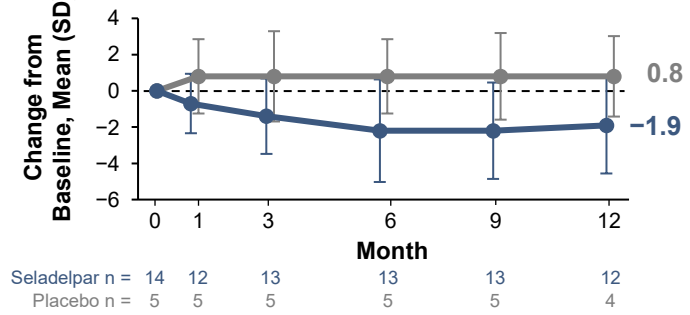
Pruritus NRS[a]



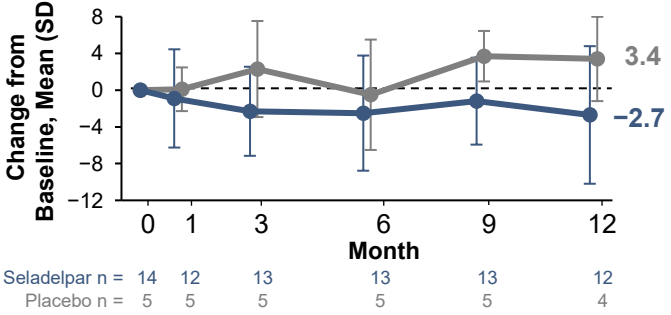
≥3-point decrease in pruritus NRS from baseline



PBC-40 Itch[b]



PBC-40 Fatigue[c]



Seladelpar 10 mg

Placebo

[a]NRS scores range from 0 (no itch) to 10 (worst itch imaginable).[1] Mean (SD) baseline pruritus NRS was 5.9 (1.2) for seladelpar and 6.5 (1.8) for placebo. [b]Higher scores within the ranges of PBC-40 Itch domain (0–15) indicate worse quality of life.[2] Mean (SD) baseline PBC-40 Itch domain was 7.8 (2.4) for seladelpar and 8.8 (2.2) for placebo. [c]Higher scores within the ranges of PBC-40 fatigue domain (11–55) indicate worse quality of life.[2] Mean (SD) baseline PBC-40 fatigue domain was 31.9 (11.4) for seladelpar and 30.7 (8.7) for placebo.

CI, confidence interval; **n,** number of evaluable patients at each time point; **NRS**, numeric rating scale; **PBC,** primary biliary cholangitis; **SD**, standard deviation. **1.** Phan NQ, et al. *Acta Derm Venereol*. 2012;92:502-7. **2.** Jacoby A, et al. *Gut.* 2005;54:1622-29.