FOUNDATION REINVENTED

WITH **DUAL ACTION** in primary IgA nephropathy[1–3]

FILSPARI®▼ (sparsentan) is indicated for the treatment of adults with primary immunoglobulin A nephropathy (IgAN) with a urine protein excretion (UPE) ≥1.0 g/day (or urine protein-to-creatinine ratio (UP/C) ≥0.75 g/g)[1]



Prescribing Information and Adverse Event reporting information can be found at the end of this document.

▼This medicine is subject to additional monitoring.
Adverse events should be reported. Reporting forms and information can be found at http://www.hpra.ie/homepage/about-us/report-an-issue. Adverse events should be reported to Vifor Pharma UK Ltd. Tel: +44 1276 853633.
E-mail: MedicalInfo_UK@viforpharma.com

For Irish Healthcare Professionals only.

CSL

IE-SPT-2600053 | October 2026

FILSPARI®▼ (sparsentan) tablets 200 mg/400 mg

Patients with IgAN (adult diagnosis) may reach kidney failure at a mean of 49 years[4]

Retrospective cohort study: real-world evidence from the IgAN cohort of the UK National Registry of Rare Kidney Diseases (RaDaR) confirmed that patients with IgAN have a high risk of kidney failure[4]

RaDaR is a UK-based rare kidney disease registry with an IgAN cohort (N=2,439 patients: 2,299 adults and 140 children). This cohort was analysed in a study looking at long-term patient outcomes, with a focus on time from diagnosis to kidney failure or death. Patients eligible for enrolment had biopsy-proven IgAN, proteinuria >0.5 g/day or eGFR <60 mL/min/1.73 m^2 at any time in the history of their disease[4]

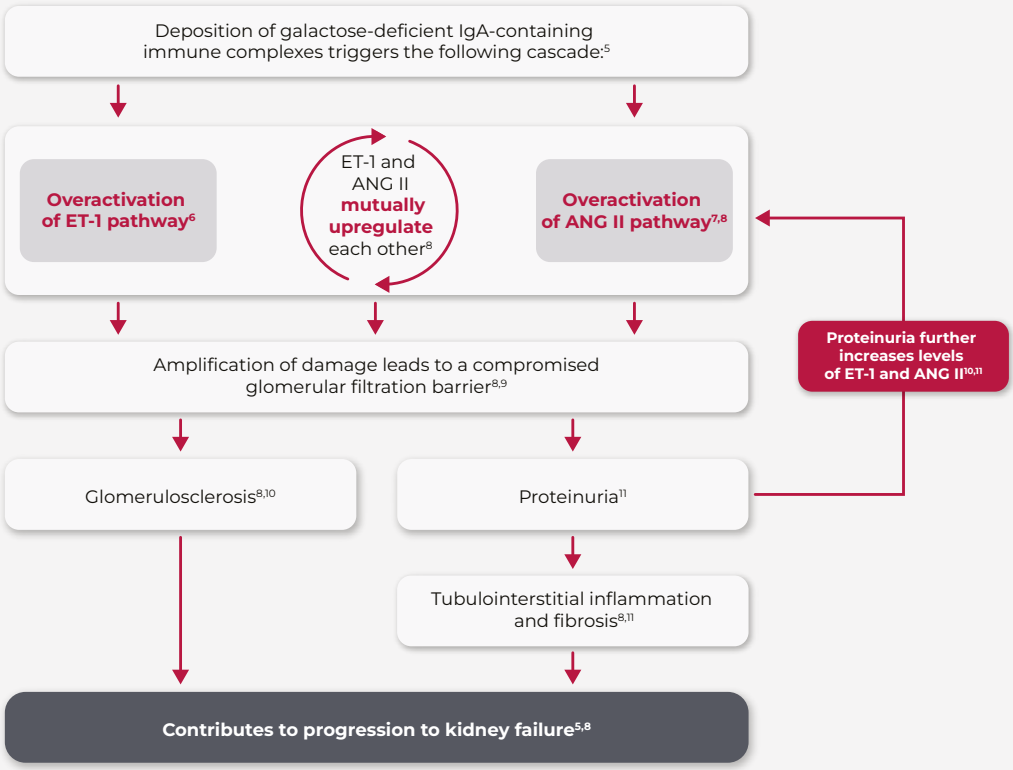
In the RaDaR cohort of adult patients with IgAN (n=2,299):[4*]

42 years	**5.8 years**	**50%**	**10–15 years**
Mean age at diagnosis	Median follow-up time	of patients reached kidney failure or died during the study period	after diagnosis, most patients progressed to kidney failure within this time frame

*UK-based study of biopsy-verified patients with IgAN (N=2,439 patients: 2,299 adults and 140 children).

Two critical pathways are involved in disease progression[5–8]

The mutual upregulation of endothelin-1 (ET-1) and angiotensin II (ANG II) is involved in the pathogenesis of IgAN[5–8]



Despite renin-angiotensin system inhibitor (RASi) treatment, just under half of patients with IgAN do not achieve adequate proteinuria reduction at 6 months[12†]

†In a prospective trial of first-line angiotensin-converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB) in patients with UP/C ≥1 g/day, 43 out of 96 patients did not achieve UP/C <1 g/day after 6 months.[12]

FILSPARI targets two critical pathways involved in IgAN disease progression in one molecule[1,13,14]

The first and only DEARA – dual endothelin angiotensin receptor antagonist[13,14]



FILSPARI
A dual endothelin angiotensin receptor antagonist **(DEARA)**



ET_A receptor
FILSPARI blocks ET-1 from binding to the ET_A receptor



AT_1 receptor
FILSPARI blocks ANG II from binding to the AT_1 receptor

ET-1, via ET_AR, and ANG II, via AT_1R, mediate processes that lead to IgAN progression through:[1]



Haemodynamic actions



Mesangial cell proliferation



Podocyte injury



Oxidative stress

Increased expression and activity of:



Pro-inflammatory mediators

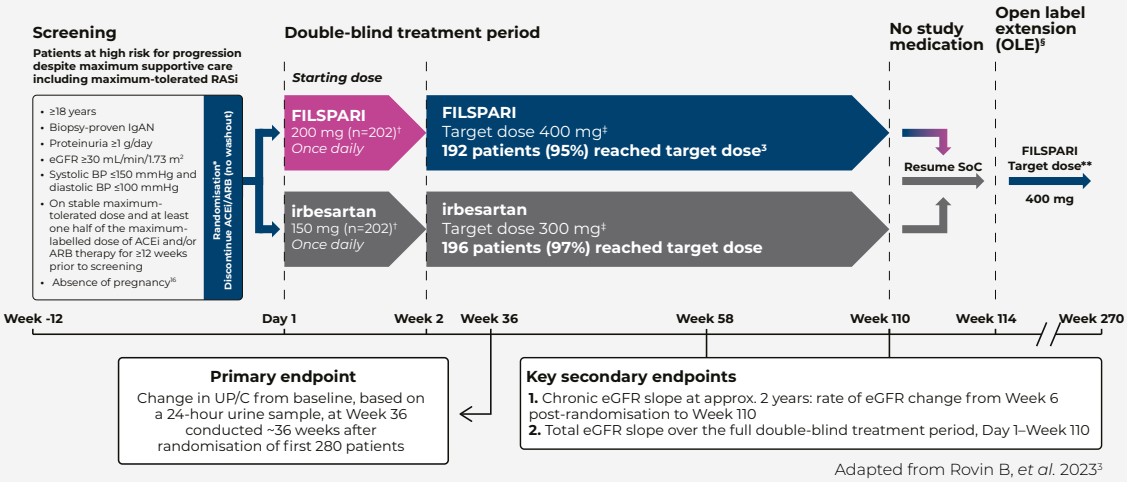


Pro-fibrotic mediators

FILSPARI, a DEARA, is the first and only non-immunosuppressive IgAN therapy that targets both pathways[13,14]

FILSPARI is the only IgAN treatment evaluated head-to-head against irbesartan[3,14,15]

PROTECT is a randomised, double-blind (110 weeks), active-controlled, multicentre, global Phase 3 trial in patients with IgAN[1,15]



Adapted from Rovin B, *et al.* 2023[3]

*On Day 1, patients were randomised 1:1 to FILSPARI or irbesartan.[3]

†One patient in each arm did not receive the study drug and was excluded.[3]

‡Before the randomisation visit, participants discontinued prohibited concomitant medications, including RASi. The initial study drug dose for the first 2 weeks was half of the target dose (200 mg oral FILSPARI once daily; 150 mg oral irbesartan once daily). At the Week 2 visit, doses were titrated to the target dose (400 mg FILSPARI once daily; 300 mg irbesartan once daily) following evaluation of dose tolerability. Dose tolerance was defined as systolic blood pressure (BP) higher than 100 mmHg, diastolic BP higher than 60 mmHg and no treatment-emergent adverse events (TEAEs). Participants with asymptomatic BP of 100/60 mmHg or less continued the initial dose. Dose titrations (down or back up) were permitted at any time at the investigator's discretion.[3]

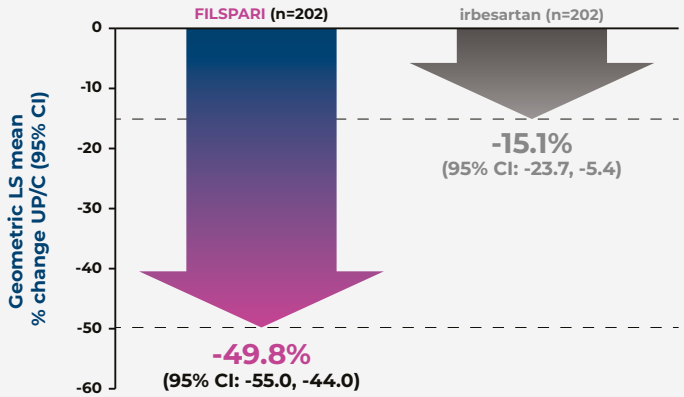
§Patients participating in the OLE period may be evaluated for eligibility to participate in the OLE sub-study. The OLE of PROTECT is currently ongoing.[17]

**Starting dose of FILSPARI for the OLE was 200 mg. Titration to 400 mg was based on tolerability after 2 weeks of treatment in the OLE.[16]

FILSPARI delivered significant and superior proteinuria reduction at Week 36 vs irbesartan[3,15]

Primary endpoint

% Change from baseline in UP/C at Week 36[15]



Baseline UP/C: FILSPARI: 1.3 g/g (0.8, 1.8) irbesartan: 1.2 g/g (0.9, 1.7)
Baseline UPE: FILSPARI: 1.8 g/day (1.2, 2.8) irbesartan: 1.8 g/day (1.3, 2.6)

At Week 36:[15]
41%
relative reduction* of proteinuria with FILSPARI vs irbesartan **p<0.0001**

At Week 36:[15]
34.7%
absolute risk reduction

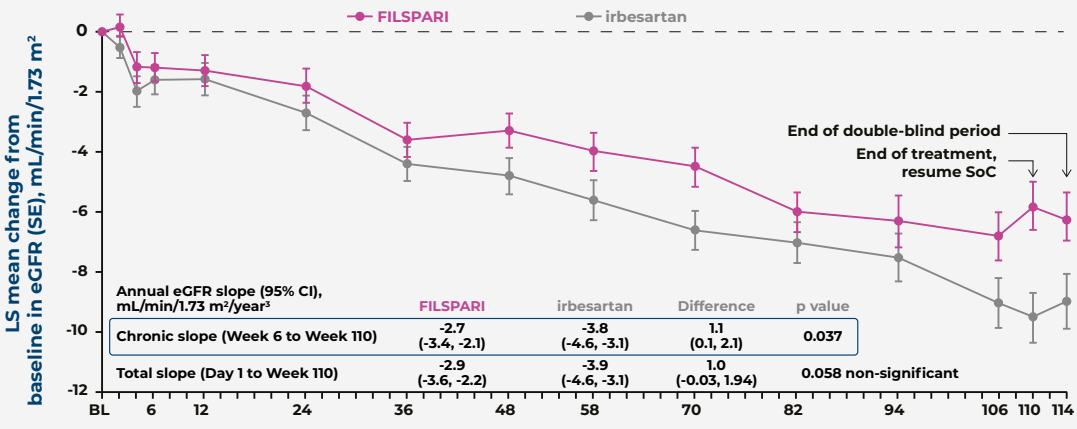
Adapted from Heerspink HJL, *et al.* 2023[15]

*Geometric LS mean ratio; 95% CI: 0.51, 0.69.[15]

FILSPARI significantly slowed the rate of eGFR decline vs irbesartan from Week 6 to Week 110[3†]

Key secondary endpoints

eGFR by visit to Week 114[3]



Annual eGFR slope (95% CI), mL/min/1.73 m²/year³	FILSPARI	irbesartan	Difference	p value
Chronic slope (Week 6 to Week 110)	-2.7 (-3.4, -2.1)	-3.8 (-4.6, -3.1)	1.1 (0.1, 2.1)	0.037
Total slope (Day 1 to Week 110)	-2.9 (-3.6, -2.2)	-3.9 (-4.6, -3.1)	1.0 (-0.03, 1.94)	0.058 non-significant

Baseline eGFR, mL/min/1.73 m² , mean (SD): **FILSPARI:** 56.8 (24.3) irbesartan: 57.1 (23.6)

Adapted from Rovin B, *et al.* 2023[3]

†Chronic slope.[3]

Absolute difference in change in eGFR from baseline to Week 110 was numerically better for FILSPARI vs irbesartan[3]

Other secondary endpoint

Under the hierarchical testing procedure, the study did not formally test the statistical hypotheses for 'other' prespecified secondary efficacy endpoints. No definite conclusions can be drawn from these data.[3]

Absolute difference in change in eGFR from baseline to Week 110:

3.7
mL/min/1.73 m^2
(95% CI: 1.5, 6.0)

irbesartan

-9.5
mL/min/1.73 m^2
(95% CI: -11.2, -7.9)

FILSPARI

-5.8
mL/min/1.73 m^2
(95% CI: -7.4, -4.2)

Statistical hypotheses for other prespecified secondary efficacy endpoints were not formally tested under the hierarchical testing procedure because the p value for the eGFR total slope was more than 0.05[3]

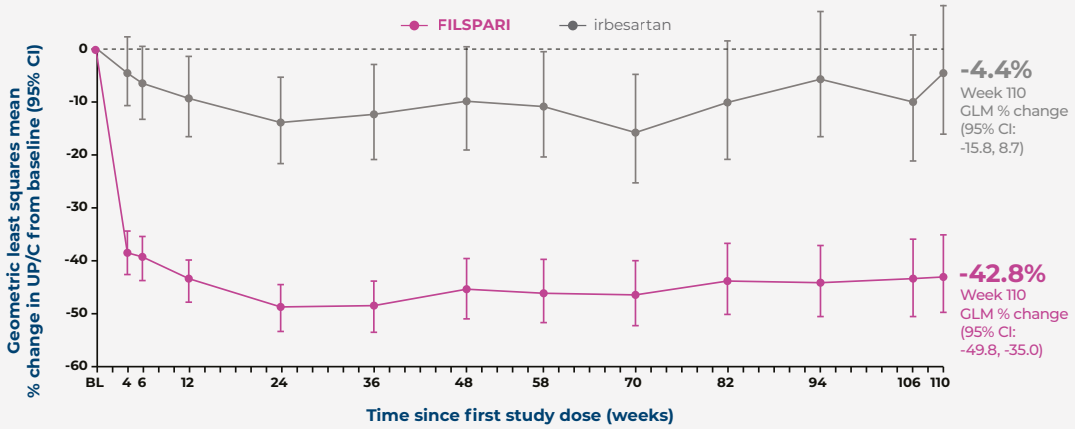
A numerical difference in proteinuria reduction for FILSPARI was observed through Week 110 vs irbesartan[3,15]

The reduction of proteinuria at Week 36 was sustained through Week 110 vs irbesartan[3]

Other secondary endpoint

Under the hierarchical testing procedure, the study did not formally test the statistical hypotheses for 'other' prespecified secondary efficacy endpoints. No definite conclusions can be drawn from these data.[3]

% Change from baseline in UP/C to Week 110[3]

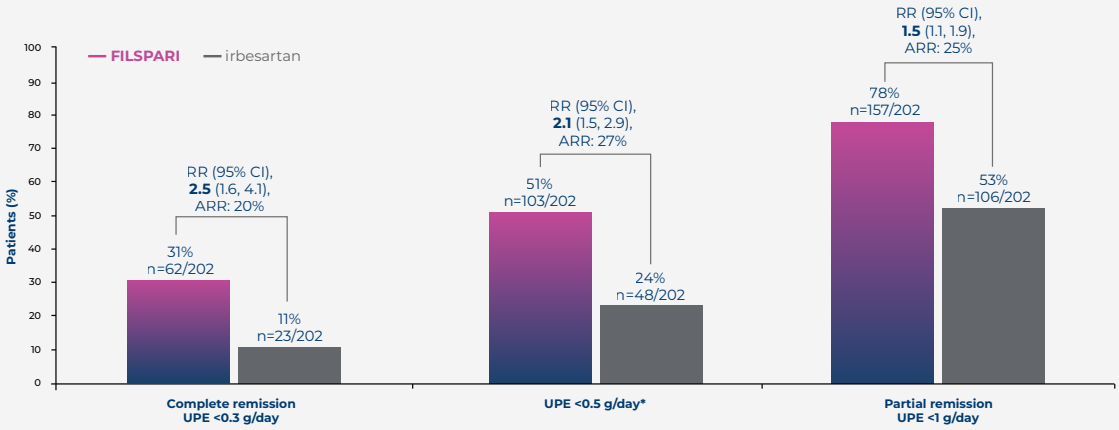


Adapted from Rovin B, *et al.* 2023[3]

Proportion of patients achieving complete (<0.3 g/day) or partial (1.0 g/day) proteinuria remission at any point up to Week 110[3,16]

Exploratory/post-hoc endpoints

Under the hierarchical testing procedure, the study did not formally test the statistical hypotheses for exploratory/post-hoc efficacy endpoints. No definite conclusions can be drawn from these data.[3]



Adapted from Rovin B, *et al*. 2023 [Suppl Appendix][16]

*The proportion of patients achieving UPE <0.5 g/day was a post-hoc assessment[16]

PROTECT study safety data[3]

Patients with TEAEs, n (%)	FILSPARI (n=202)	irbesartan (n=202)
Any TEAEs	187 (93)	177 (88)
Most common TEAEs (≥10% of patients in either group)		
COVID-19	53 (26)	46 (23)
Hyperkalaemia	32 (16)	26 (13)
Peripheral oedema	31 (15)	24 (12)
Dizziness	30 (15)	13 (6)
Headache	27 (13)	26 (13)
Hypotension	26 (13)	8 (4)
Serious TEAEs	75 (37)	71 (35)
Serious TEAEs in ≥5 patients in either group		
COVID-19	42 (21)	38 (19)
Chronic kidney disease	6 (3)	6 (3)
TEAEs leading to treatment discontinuation	21 (10)	18 (9)
TEAEs leading to death	0	1 (<1)[†]

Adapted from Rovin B, *et al*. 2023

[†]One patient in the irbesartan group died due to a severe adverse event of cardiorespiratory arrest that was considered not to be related to study drug.[3]

Safety profile of FILSPARI[1]

Safety outcome

The most common adverse drug reactions observed during clinical trials[1]



Hypotension
10.8%

Hyperkalaemia
9.6%

Dizziness
7.8%

Peripheral oedema
5.4%

The most common serious adverse reaction reported was acute kidney injury (0.9%)[1]

Adverse drug reactions observed during clinical trials[1]

Safety outcome

System organ class	Very common	Common	Uncommon
Blood and lymphatic system disorders	—	—	Anaemia
Metabolism and nutrition disorders	—	Hyperkalaemia	—
Nervous system disorders	—	Dizziness Headache	—
Vascular disorders	Hypotension	Orthostatic hypotension	—
Renal and urinary disorders	—	Renal impairment Acute kidney injury	—
General disorders and administration site conditions	—	Oedema peripheral Fatigue	—
Investigations	—	Blood creatinine increased Elevated transaminase*	—

Refer to the SmPC for full safety information before prescribing

*Elevated transaminase includes preferred terms of ALT increased, AST increased, gamma-glutamyltransferase increased, and hepatic enzyme increased.[1]

Special warnings and precautions for use[1]



Women of childbearing potential (WOCBP)

FILSPARI treatment must only be initiated in WOCBP when the absence of pregnancy has been verified, and effective contraception is practiced. Exclude pregnancy before, during and for 1 month after treatment with FILSPARI has stopped. WOCBP have to use effective contraception during and up to 1 month after treatment has stopped.[1]



Liver function

Elevations in ALT or AST of at least 3× ULN have been observed with FILSPARI. To reduce the risk of potential serious hepatotoxicity, serum aminotransferase levels and total bilirubin should be monitored prior to initiation of treatment and then continuously monitored every 3 months. If patients develop sustained, unexplained, clinically significant ALT and/or AST elevation, or if elevations are accompanied by an increase in bilirubin >2 × ULN, or if ALT and/or AST elevation is accompanied by signs or symptoms of hepatic injury (e.g, jaundice), FILSPARI therapy should be discontinued. Consider re-initiation of FILSPARI only when hepatic enzyme levels and bilirubin return to pretreatment values and only in patients without clinical symptoms of hepatotoxicity. Avoid initiation of FILSPARI in patients with elevated aminotransferase (>2 × ULN) prior to drug initiation. Clinical experience with moderate hepatic impairment is limited, therefore FILSPARI should be used with caution in these patients.[1]



Breastfeeding

Physicochemical data suggest excretion of FILSPARI in human milk. A risk to newborns/infants cannot be excluded. FILSPARI should not be used during breastfeeding.[1]



Fertility

There are no data on the effects of FILSPARI on human fertility.[1]



Hypotension

Hypotension has been associated with the use of renin-angiotensin-aldosterone system (RAAS) inhibitors, including FILSPARI. Hypotension may occur during treatment with FILSPARI and is reported more frequently in elderly patients. In patients at risk for hypotension, eliminating or adjusting other antihypertensive medicinal products and maintaining appropriate volume status should be considered. If hypotension develops despite elimination or reduction of other antihypertensive medicinal products, dose reduction or dose interruption of FILSPARI should be considered.[1]

A transient hypotensive response is not a contraindication to further dosing of FILSPARI; treatment can be resumed once blood pressure has stabilised. FILSPARI should be used with caution in patients with systolic blood pressure values ≤100 mmHg. FILSPARI should not be uptitrated in patients with systolic blood pressure values ≤100 mmHg.[1]



Impaired kidney function

A transient increase in serum creatinine has been associated with RAAS inhibitors, including FILSPARI. A transient increase in serum creatinine may occur, especially when initiating treatment with FILSPARI. Periodic monitoring of serum creatinine and serum potassium levels should be performed in patients at risk. FILSPARI should be used with caution in patients with bilateral renal artery stenosis. Clinical experience in patients with an eGFR <30 mL/min/1.73 m^2 is limited, therefore, FILSPARI is not recommended in these patients.[1]

Special warnings and precautions for use[1]



Fluid retention

Fluid retention has been associated with medicinal products that antagonise the ET_AR, including FILSPARI. Fluid retention may occur during treatment with FILSPARI. If fluid retention develops during treatment with FILSPARI, treatment with diuretics is recommended, or the dose of existing diuretics should be increased before modifying the dose of FILSPARI. Treatment with diuretics can be considered in patients with evidence of fluid retention before treatment initiation with FILSPARI. FILSPARI has not been studied in patients with heart failure, therefore, FILSPARI should be used with caution in patients with heart failure.[1]



Dual blockade of RAAS

There is evidence that concomitant use of ACEi, ARBs or aliskiren increases risk of hypotension, hyperkalaemia and decreased renal function. Dual blockade of RAAS through the combined use of ACEi, ARBs (partly a mechanism of FILSPARI) or renin inhibitors is not recommended. If dual blockade therapy is considered necessary, this should only occur under specialist supervision and subject to close monitoring of renal function, electrolytes and blood pressure.[1]



Effects on ability to drive and use machines

FILSPARI may have minor influence on the ability to drive and use machines. No studies on the effects of FILSPARI on the ability to drive and use machines have been performed. It should, however, be taken into account that dizziness may occur when taking FILSPARI. Patients with dizziness should be advised to refrain from driving or using machines until symptoms have subsided.[1]



Hyperkalaemia

Treatment should not be initiated in patients with serum potassium level >5.5 mmol/L. As with other medicinal products that affect the RAAS, hyperkalaemia may occur during treatment with FILSPARI, especially in the presence of renal impairment and/or heart failure. Close monitoring of serum potassium in patients at risk is recommended. If patients experience clinically significant hyperkalaemia, adjustment of concomitant medicinal products, or temporary down-titration or discontinuation is recommended. If serum potassium level is >5.5 mmol/L discontinuation should be considered.[1]



Lactose

Patients with rare hereditary problems of galactose intolerance, total lactase deficiency, or glucose-galactose malabsorption should not take this medicinal product.[1]



Sodium

This medicinal product contains less than 1 mmol sodium (23 mg) per tablet, that is to say essentially 'sodium-free'.[1]

Refer to the FILSPARI SmPC for full safety information before prescribing.

One tablet, once daily. One molecule addressing two critical pathways involved in IgAN disease progression[1]

200 or 400 mg tablets

1x daily

With or without food
It is recommended to swallow the tablets whole with water to avoid bitter taste

Only initiate FILSPARI in verified absence of pregnancy and while on effective contraception

Posology[1]

Stop RASi if applicable	Initiate days 1–14	Maintain days 15+ dependent on tolerability
No washout[1,4]	**1 x 200 mg/day**	**1 x 400 mg/day**

Prior to starting treatment with FILSPARI, discontinue use of ARBs, endothelin receptor antagonists (ERA) or renin inhibitors. To reduce the risk of potential serious hepatotoxicity, serum aminotransferase levels and total bilirubin should be monitored prior to initiation of treatment and then continue monitoring every 3 months

In women of childbearing potential, verify the absence of pregnancy prior to initiation/use of effective contraception[1]

Treatment with FILSPARI should be initiated at a dose of 200 mg once daily for 14 days and then increased to a maintenance dose of 400 mg once daily, dependent upon tolerability[1]

If patients experience tolerability issues (systolic BP ≤ 100 mmHg, diastolic BP ≤60 mmHg, worsening oedema, or hyperkalaemia), adjustment of concomitant medicinal products, followed by temporary down-titration or discontinuation of FILSPARI, is recommended[1]

When resuming treatment with FILSPARI after interruption, repeating the initial dosing schedule may be considered. Interruption of treatment preceded, or not by dose reduction of FILSPARI, may be considered based on persisting hypotension or changes in liver function[1]



Contraindications:[1]

- Hypersensitivity to the active substance(s) or to any of the excipients
- Pregnancy
- Coadministration of ARBs, ERAs or renin inhibitors

FILSPARI: foundation reinvented with dual action[1–3]

FILSPARI: first and only DEARA[13,14]

Innovative non-immuno-suppressive dual MoA

- FILSPARI, a **DEARA, targets two critical pathways**, the endothelin and angiotensin pathways, **involved in IgAN disease progression** in one molecule[1]

Significant and superior proteinuria reduction vs irbesartan

- **Significant and superior reduction from baseline in UP/C at Week 36 vs irbesartan** (**FILSPARI: -49.8%** [95% CI: -55.0, -44.0] **vs irbesartan: -15.1%** [95 CI: -23.7, -5.4]; p<0.0001)[3,15]

Preserved kidney function (as part of PROTECT)

- **Significantly slows the rate of eGFR decline vs** vs irbesartan[3]*
- **Chronic slope for FILSPARI vs irbesartan was -2.7 vs -3.8 mL/min/1.73 m² per year,** respectively; difference, 1.1 mL/min/1.73 m² per year (95% CI: 0.1, 2.1; p=0.037)[3]

Convenient once-daily tablet

- **One tablet, once daily**[1]
- One molecule addressing two critical pathways involved in IgAN disease progression[1]

FILSPARI key safety information

The most commonly reported adverse drug reactions with FILSPARI were **hypotension (10.8 %), hyperkalaemia (9.6 %), dizziness (7.8 %), and oedema peripheral (5.4 %).** The most common serious adverse reaction reported was **acute kidney injury (0.9 %)**[1]

FILSPARI treatment must **only be initiated in women of childbearing potential when the absence of pregnancy has been verified** and effective contraception is practiced. Women of childbearing potential have to use effective contraception during and up to 1 month after treatment has stopped[1]

Liver function should be monitored prior to initiation of treatment and then continue monitoring every 3 months. Avoid initiation of FILSPARI in patients with elevated aminotransferases (> 2 × ULN). Discontinue in patients with sustained, unexplained, clinically significant elevated aminotransferases, with or without elevated bilirubin (> 2 × ULN), or accompanied by signs and symptoms of hepatic injury[1]

*Chronic slope. Key secondary endpoints included chronic eGFR slope (rate of eGFR change over Week 6 to Week 110) and total eGFR slope (Day 1–Week 110) over the full double-blind treatment period[3]

References and Abbreviations

ACEi, angiotensin-converting enzyme inhibitor; **ANG II,** angiotensin II; **ALT,** alanine aminotransferase; **ARB,** angiotensin II receptor blocker; **ARR,** absolute risk reduction; **AST,** aspartate aminotransferase; **AT_1,** angiotensin II type 1; **BL,** baseline; **BP,** blood pressure; **CI,** confidence interval; **DEARA,** dual endothelin angiotensin receptor antagonist; **eGFR,** estimated glomerular filtration rate; **ERA,** endothelin receptor antagonist; **ET-1,** endothelin 1; **ET_A,** endothelin type A; **GLM,** geometric least squares mean; **IgA,** immunoglobulin A; **IgAN,** immunoglobulin A nephropathy; **K+,** potassium; **LS,** least squares; **MoA,** mechanism of action; **OLE,** open-label extension; **RAAS,** renin-angiotensin-aldosterone system; **RaDaR,** UK National Registry of Rare Kidney Diseases; **RASi,** renin-angiotensin system inhibitor; **RR,** relative risk; **SD,** standard deviation; **SE,** standard error; **SoC,** standard of care; **TEAE,** treatment-emergent adverse event; **UK,** United Kingdom; **ULN,** upper limit of normal; **UP/C,** urine protein-to-creatinine ratio; **UPE,** urine protein excretion; **WOCBP,** women of childbearing potential

1. FILSPARI EU SmPC. Last accessed: October 2026. **2.** Barratt J, *et al. Kidney Int Rep.* 2023;8(5):1043–56. **3.** Rovin B, *et al. Lancet.* 2023;402(10417):2077–90. **4.** Pitcher D, *et al. Clin J Am Soc Nephrol.* 2023;18(6):727–38. **5.** Lai KN, *et al. Nat Rev Dis Primers.* 2016;2:16001. **6.** Lehrke I, *et al. J Am Soc Nephrol.* 2001;12(11):2321–9. **7.** Chan LY, *et al. J Am Soc Nephrol.* 2005;16(8):2306–17. **8.** Komers R, Plotkin H. *Am J Physiol Regul Integr Comp Physiol.* 2016;310:R877–84. **9.** Kohan DE, *et al. Compr Physiol.* 2011;1:883–919. **10.** Raina R, *et al. Kidney Dis.* 2020;6:22–34. **11.** Sharma S, Smyth B. *Kidney Blood Press Res.* 2021;46:411–20. **12.** Bagchi S, *et al.* Kidney *Int Rep.* 2021;6:1661–8. **13.** Syed YY, *et al. Drugs.* 2023;83(6):563–8. **14.** Campbell KN, *et al. Int J Nephrol Renovasc Dis.* 2023;16:281–91. **15.** Heerspink HJL, *et al. Lancet.* 2023;401(10388):1584–94. **16.** Rovin B, *et al. Lancet.* 2023;402(10417):2077–90 [Suppl Appendix]. **17.** PROTECT ClinicalTrials.gov:http://clinicaltrials.gov/ct2/show/NCT03762850. Date Accessed: October 2026.

Filspari▼ (sparsentan)

Prescribing Information – Republic of Ireland

For full prescribing information refer to the Summary of Product Characteristics (SmPC)

Active ingredient: Sparsentan

Presentation: Film-coated tablets as 200mg or 400mg.

Indication: Filspari is indicated for the treatment of adults with primary immunoglobulin A nephropathy (IgAN) with a urine protein excretion ≥1.0 g/day (or urine protein-to-creatinine ratio ≥0.75 g/g).

Dosage and Administration: Initiate at a dose of 200 mg once daily for 14 days and then increase to a maintenance dose of 400 mg once daily, dependent upon tolerability. If patients experience tolerability issues (systolic blood pressure [SBP] ≤100 mmHg, diastolic blood pressure ≤60 mmHg, worsening oedema, or hyperkalaemia), adjustment of concomitant medicinal products, followed by temporary down-titration or discontinuation of Filspari is recommended. When resuming treatment with Filspari after interruption, repeating the initial dosing schedule may be considered. Interruption of treatment preceded, or not, by dose reduction of Filspari may be considered based on persisting hypotension or changes in liver function. For oral use. Recommended to swallow whole with water.

Special populations: Elderly: No dose adjustment is recommended. The increase to a maintenance dose of 400 mg once daily should be performed with caution, based on tolerability. **Hepatic impairment:** No dose adjustment recommended in patients with mild or moderate hepatic impairment. There is limited experience in patients with moderate hepatic impairment, therefore use with caution. Sparsentan has not been studied in patients with severe hepatic impairment, therefore is not recommended for use. Due to limited of clinical experience, Filspari should not be initiated in patients with AST or ALT >2 x ULN. **Renal impairment:** No dose adjustment is required in patients with mild chronic kidney disease (CKD) stage 2 (eGFR 60-89 mL/min/1.73 m²) or moderate CKD stages 3a and 3b (eGFR 30-59 mL/min/1.73 m²). As there is limited clinical experience in patients with severe kidney disease, sparsentan is not recommended in these patients. **Kidney transplant:** Sparsentan has not been studied in patients who have received a kidney transplant, therefore sparsentan should be used with caution is these patients. **Dialysis:** Sparsentan has not been studied in patients undergoing dialysis, therefore initiation is not recommended. **Children:** The safety and efficacy of Filspari in children below the age of 18 years with IgAN have not yet been established. No data are available. **Pregnancy:** Sparsentan treatment must only be initiated in women of childbearing potential when the absence of pregnancy has been verified. Exclude pregnancy before, during and for 1 month after treatment with sparsentan has stopped. The frequency of recommended pregnancy testing should be determined by the method of contraception used and the associated likelihood of contraceptive failure. Women of childbearing potential have to use effective contraception during and up to 1 month after treatment has stopped. Filspari is contraindicated during pregnancy. **Breastfeeding:** Sparsentan should not be used during breastfeeding.

Contraindications: Filspari is contraindicated in patients with hypersensitivity to sparsentan or to any of its excipients. Filspari is contraindicated during pregnancy. Do not co-administer Filspari with angiotensin receptor blockers, endothelin receptor antagonists, or renin inhibitors

Special warnings and precautions:

Women of childbearing potential: Filspari treatment must only be initiated in women of childbearing potential when the absence of pregnancy has been verified and effective contraception is practised. Exclude pregnancy before, during and for 1 month after treatment with sparsentan has stopped. The frequency of recommended pregnancy testing should be determined by the method of contraception used and the associated likelihood of contraceptive failure. Women of childbearing potential have to use effective contraception during and up to 1 month after treatment has stopped. Filspari is contraindicated during pregnancy. **Hypotension:** Hypotension may occur during Filspari treatment, and is more frequently reported in elderly patients. In patients at risk for hypotension, eliminating or adjusting other antihypertensive medicinal products and maintaining appropriate volume status should be considered. If hypotension develops despite elimination or reduction of other antihypertensive medicinal products, dose reduction or dose interruption of Filspari should be considered. A transient hypotensive response is not a contraindication to further dosing of Filspari; treatment can be resumed once BP has stabilised. If hypotension persists despite elimination or reduction of antihypertensive medicinal products, sparsentan dosing should be reduced to the initial starting dose until BP stabilises. Dose interruption of treatment with sparsentan should be considered if symptoms of hypotension persist after 2 weeks of dose reduction. Sparsentan should be used with caution in patients with SBP values ≤ 100 mmHg. Sparsentan should not be uptitrated in patients with SBP values ≤ 100 mmHg. **Impaired kidney function:** A transient increase in serum creatinine may occur, especially during treatment initiation. Periodic monitoring of serum creatinine and serum potassium levels should be performed in patients at risk. Filspari should be used with caution in patients with bilateral renal artery stenosis. Filspari is not recommended in patients with an estimated glomerular filtration rate (eGFR) <30 mL/min/1.73m². **Fluid retention:** Fluid retention has been associated with medicinal products that antagonise the endothelin type A receptor, including Filspari. If fluid retention develops, treatment with diuretics is recommended, or the dose of existing diuretics should be increased before modifying the dose of Filspari. Consider diuretic treatment in patients with existing fluid retention prior to initiation of Filspari. Filspari has not been studied in patients with heart failure, therefore should be used with caution. **Liver function:** Elevations in ALT or AST of at least 3 x ULN have been observed with Filspari. No concurrent elevations in bilirubin >2 x ULN or cases of liver failure have been observed in Filspari-treated patients. To reduce the risk of potential serious hepatotoxicity, serum aminotransferase levels and total bilirubin should be monitored prior to initiation of treatment and then every 3 months. Patients should be monitored for signs of hepatic injury. If patients develop sustained, unexplained, clinically significant ALT and/or AST elevation, or if elevations are accompanied by an increase in bilirubin >2 × ULN, or if ALT and/or AST elevation is accompanied by signs or symptoms of hepatic injury (e.g. jaundice), Filspari therapy should be discontinued. Consider re-initiation of Filspari only when hepatic enzyme levels and bilirubin return to pretreatment values and only in patients without clinical symptoms of hepatotoxicity. Avoid initiation of sparsentan in patients with elevated aminotransferase (> 2 × ULN) prior to drug initiation. There is limited clinical experience with moderate hepatic impairment, therefore, use with caution. **Dual blockade of the Renin-angiotensin-aldosterone system (RAAS):** Dual blockade of RAAS through the combined use of ACE inhibitors, angiotensin II receptor blockers (partly a mechanism of sparsentan) or renin inhibitors is not recommended. If dual blockade therapy is considered absolutely necessary, this should only occur under specialist supervision and subject to frequent close monitoring of renal function, electrolytes and BP. **Hyperkalaemia:** Treatment should not be initiated in patients with serum potassium level > 5.5 mmol/l. As with other medicinal products that affect the RAAS, hyperkalaemia may occur during the treatment with sparsentan, especially in the presence of renal impairment and/or heart failure. Close monitoring of serum potassium in patients at risk is recommended. If patients experience clinically significant hyperkalaemia adjustment of concomitant medicinal products, or temporary down–titration or discontinuation is recommended. If serum potassium level is > 5.5 mmol/l discontinuation should be considered. **Excipients:** Contains lactose. Patients with rare hereditary problems of galactose intolerance, total lactase deficiency, or glucose-galactose malabsorption should not take this medicinal product. Filspari contains less than 1 mmol sodium (23 mg) per tablet, and is essentially 'sodium-free'.

Overdose: Sparsentan has been administered in doses of up to 1600 mg/day in healthy subjects without evidence of dose limiting toxicities. Patients who experience overdose (possibly experiencing signs and symptoms of hypotension) should be monitored closely and appropriate symptomatic treatment given.

Undesirable effects: Very common (≥ 1/10): Hypotension. Common (≥ 1/100 to < 1/10): hyperkalaemia, dizziness, headache, orthostatic hypotension, renal impairment, acute kidney injury, peripheral oedema, fatigue, blood creatinine increased, elevated transaminase. Uncommon (≥ 1/1,000 to < 1/100): anaemia.

Please consult the SmPC for further information on undesirable effects.

Legal category: POM

MA Number: EU/1/23/1788/001 (200mg tablet), EU/1/23/1788/002 (400mg tablet), EU/1/23/1788/003 (400mg tablet)

Date of authorisation: 19/04/2024

MA Holder: Vifor France, 100-101 Terrasse Boieldieu, Tour Franklin La Défense 8, 92042 Paris la Défense Cedex, France.

Document number: IE-SPT-2600010

Date of preparation: July 2026

Additional information is available on request.

▼ This medicine is subject to additional monitoring. Adverse events should be reported. Reporting forms and information can be found at http://www.hpra.ie/homepage/about-us/report-an-issue. Adverse events should also be reported to Vifor Fresenius Medical Care Renal Pharma, care of Vifor Pharma Ltd.
Tel: + 441276 853633. E-mail: medicalinfo_UK@viforpharma.com

CSL