The Decompensated Cirrhotic Patient Who Recompensates: How Does It Happen?

« Le cirrhotique décompensé qui se recompense : ça arrive comment ? »

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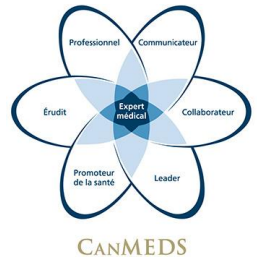
Medizinische Klinik B · Universitätsklinikum Münster

Journée André-Viallet · Montréal · September 18, 2026

Conflicts of Interest

Employment	Deputy Director, University Hospital Münster Professorship Hepatology & Rare Diseases, University of Münster
Consultancy and lectures	Gore, AstraZeneca, Roche, AbbVie, Gilead, MicroTech, Cook Medical, Ewimed, Falk, Madrigal
Stocks	None
Scientific grants	CareerS ClinicianScientist Programme and MedK Programme (University of Münster) DFG Exzellence Cluster EXC2151-390873048 BONFOR Starter Group 2021-2A-07 Ernst-und-Berta Grimmke Foundation Lfd.5/19
Other financial relationships	Keine

Compétences CanMEDS



X	**Expert médical** Apply current concepts of recompensation to the assessment and management of patients with decompensated cirrhosis.
X	**Communicateur** Explain recompensation, residual risk and treatment goals to patients and colleagues.
X	**Collaborateur** Integrate etiological treatment, portal hypertension management and interventional strategies into multidisciplinary patient care.
X	**Leader** Apply evolving concepts of recompensation to optimize long-term management strategies.
X	**Promoteur de santé** Promote etiological treatment and sustained control of modifiable disease drivers.
X	**Érudit** Critically appraise the evidence on mechanisms, predictors and therapeutic drivers of recompensation.
X	**Professionnel** Apply evidence-based criteria when defining recompensation and considering treatment withdrawal.

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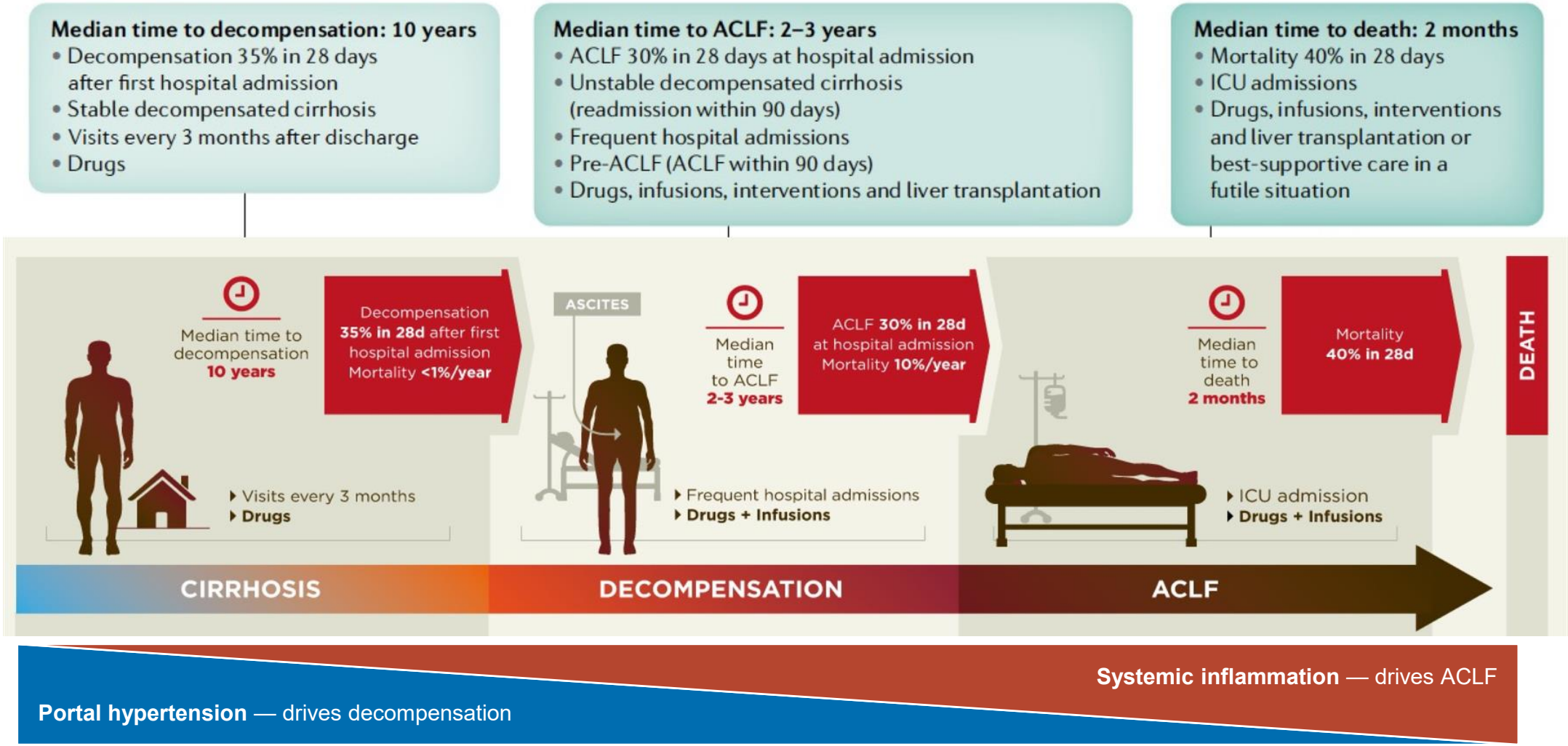
Decompensation was a one-way door



Modified after Trebicka et al. Nat Rev Gastroenterol Hepatol 2020

Three natural experiments

HBV suppression

Potent nucleos(t)ide analogues drive durable viral control.

HCV cure

Direct-acting antivirals achieve SVR in >95%.

Alcohol abstinence

Removing the driver halts and reverses injury.

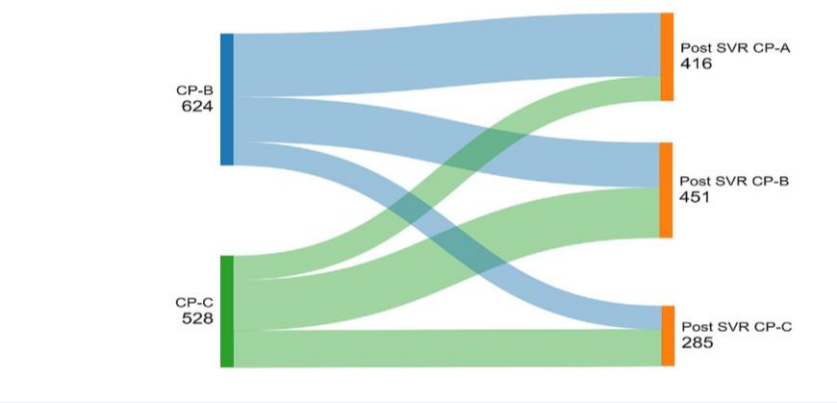
*Remove the cause — and patients show **clinical improvement, restored function, even fibrosis regression**.*

Reiberger et al. Dig Liver Dis 2023 · Piano et al. JHEP Rep 2024

What the natural experiments showed

IMPROVES

Child–Pugh class one year after HCV cure (n = 1,152)[1]

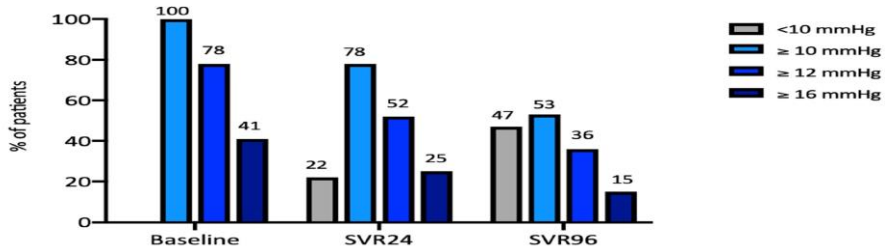


86% ascites resolved (HCV)[1] **> 90%** ↓ liver-related mortality (alcohol)[2]

7.2 → 2.6 liver-related deaths / 100 PY (HCV)[3]

DOES NOT RETURN TO NORMAL

CSPH 2 yr SVR: 53–65% persistence[5]; after alcohol recompensation: 59%[6]



Stiffness / fibrosis

6 yr SVR: F3 48%, F4 30% — fibrosis regresses only partly[4]

Varices

SVR: 17% resolved, 12% de novo[7] — persist even with HVPG < 10 mmHg[8]

Recovery is not a return to baseline.

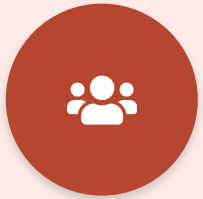
[1] Premkumar Gastro 2024 [2] Hofer Liver Int 2023 [3] Semmler CGH 2025 [4] Canillas Diagnostics 2026 [5] Lens JHEP 2020 [6] Hofer CGH 2026 [7] Giannini EJCI 2020 [8] Olivas JHEP Rep 2024

Learning objectives

OBJECTIVE 1

How does it happen?

The pathophysiology of recompensation — what function, inflammation and fibrosis reverse, and what scars persist.



OBJECTIVE 2

Who recompensates?

Identifying the patients most likely to recover — the predictors across etiologies.

OBJECTIVE 3

What drives it?

The therapeutic interventions that promote recompensation — from etiological cure to TIPS.

The current Baveno VIII definition

2016–20
First reversal reports (HBV / HCV)

2021
Baveno VII defines recompensation

2022–25
Validation across etiologies

2026
Baveno VIII — criteria refined & published

The Baveno VIII definition (2026) — all criteria required[1]

1 · Control the cause
Removal, control or suppression of the primary aetiology — SVR (HCV), HBV-DNA suppression, sustained abstinence (ALD).

2 · Clear the events
Ascites resolved off diuretics; no HE off lactulose / rifaximin / LOLA; no rebleeding after variceal bleeding, regardless of NSBB.

3 · Restore function
Liver function improved to a Child–Pugh A5/A6 range.

Sustained > 6 consecutive months *— durability is now explicit (7.4d), and function is anchored to Child–Pugh A5/A6.*

[1] Berzigotti, …, Praktiknjo et al. JHEP 2026 (Baveno VIII) *[2] de Franchis et al. JHEP 2022 (Baveno VII)*

What recompensation is NOT

Not 'controlled on therapy'

Ascites dry on diuretics, or HE-free on lactulose / rifaximin / LOLA, is compensated decompensation — not recompensation. Withdraw the drugs once the cause is controlled and function is CTP A (7.6–7.7).

Not cirrhosis regression

Recompensation is a clinical transition back to a compensated stage — it does not mean the cirrhotic architecture has reversed histologically (7.2).

Not one event 'fixed' by TIPS

After TIPS, resolved ascites alone or absence of rebleeding alone doesn't count — all criteria (aetiologic control, function, > 6 months) must still be met (7.8).

Incorrect definitions may explain varying rates.

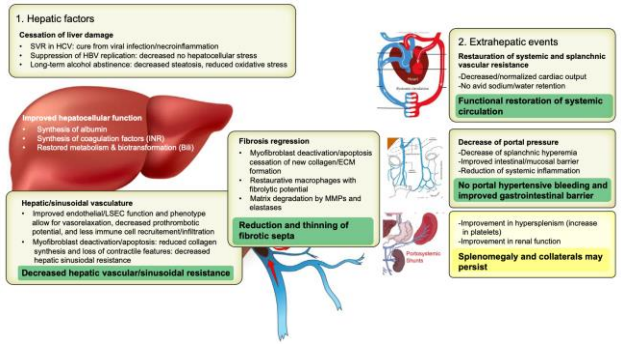
Baveno VIII (JHEP 2026), statements 7.2 / 7.6–7.8 · de Franchis et al. JHEP 2022

1

HOW DOES IT HAPPEN?

The pathophysiology of recompensation

Function & inflammation recover quickly — architecture persists



Liver function — Recovers quickly — bilirubin, INR and albumin improve[1].

Systemic inflammation — Recovers quickly — systemically measured: circulating IL-6, IL-1β and IL-10 fall toward compensated levels[2].

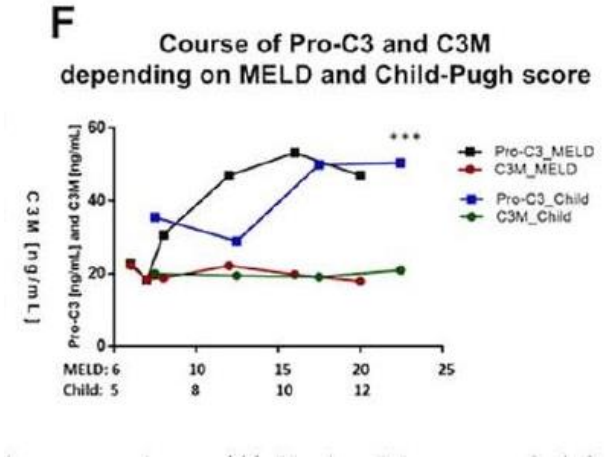
Fibrosis / ECM architecture — Persists — the mature matrix regresses slowly, if at all.

Portal hypertension — Persists with it — CSPH resolves in only ~41%; varices can outlast even a normalised pressure[3].

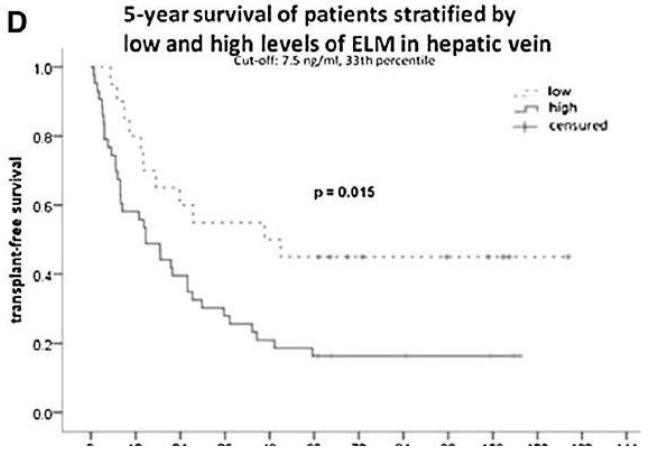
Etiology and inflammation can be treated quickly — so why do they stay vulnerable? Two things outlast etiological cure.

[1] Wang et al. JHEP 2022 · Hofer et al. CGH 2026 [2] Tonon et al. JHEP 2025 [3] Olivas et al. JHEP Rep 2024

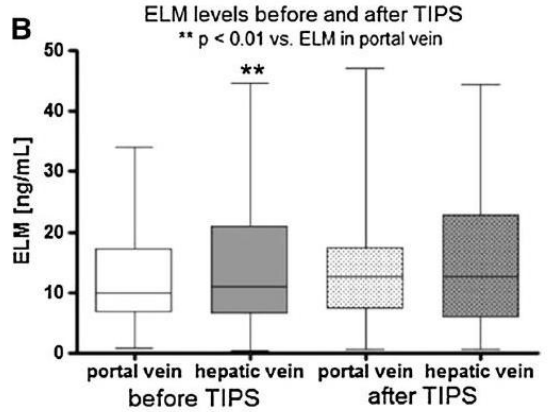
Fibrosis accumulates and is resistant



Formation outruns degradation: PRO-C3 rises while C3M stays flat as liver disease progresses — net collagen deposition[1]; collagen IV remodelling predicts mortality[3].



And it costs survival: high hepatic-vein elastin halves 5-year transplant-free survival (p = 0.015) — cross-linked elastin shields collagen from proteases[2].



And it barely moves: elastin fragments are unchanged even after TIPS radically removes portal hypertension — the shield outlasts what we can fix fast[2].

WHY IT STAYS				
	Stiffness feedback YAP-TAZ keeps HSCs on	**Cross-linking** LOX / LOXL2 hide MMP cleavage sites	**Elastin shield** embedded alongside collagen I/III	**Paucicellular scar** no local proteases left

[1] Praktiknjo et al. HepCom 2018 [2] Nielsen et al. Dig Dis Sci 2015 [3] Lehmann, Praktiknjo et al. Liver Int 2019

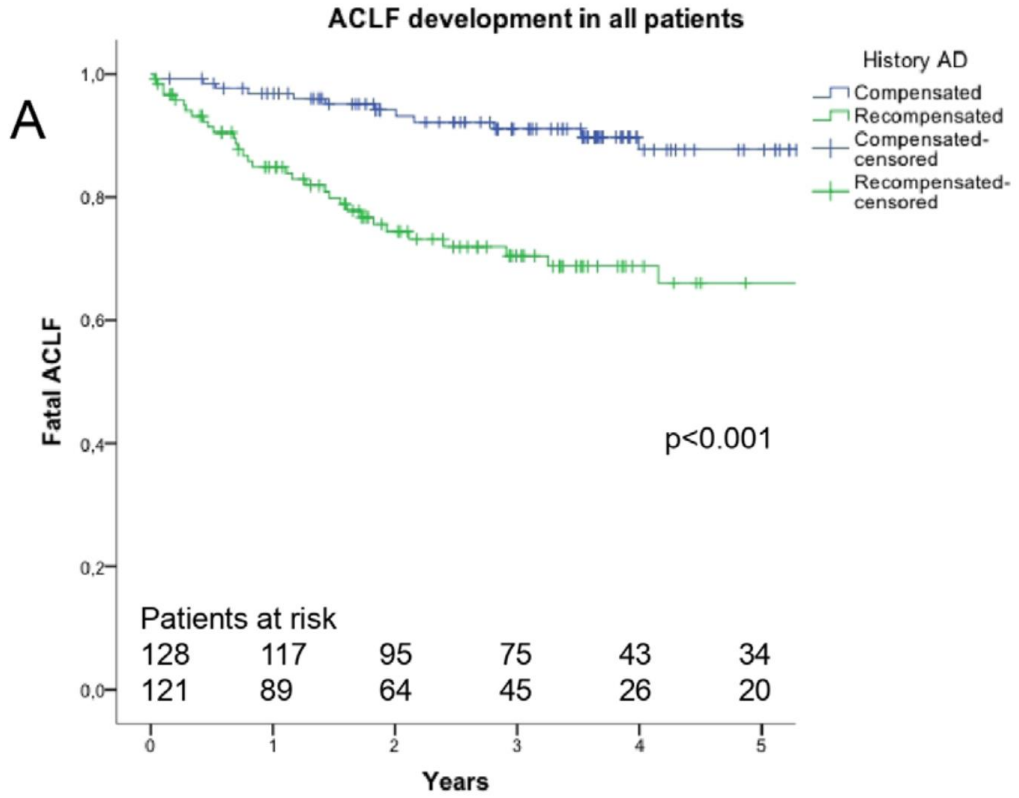
Prior decompensation leaves an immune scar

ON THE SURFACE — THEY LOOK ALIKE

Same clinical phenotype · all outpatients · same liver function tests.

UNDERNEATH — THEY ARE NOT

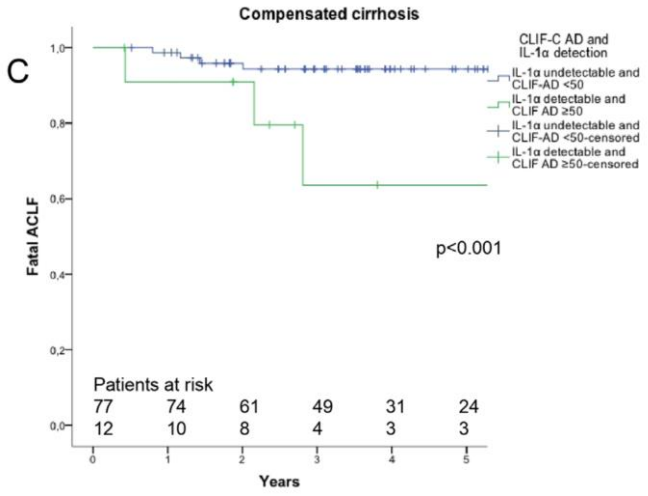
Immunologically different — a distinct inflammasome program persists.



Previous acute decompensation independently predicts fatal ACLF — even after clinical recompensation. The immune system remembers.

Monteiro, …, Praktiknjo. Gut 2021

Two inflammasome programs



NEVER-DECOMPENSATED COMPENSATED

IL-1α

caspase-4/11 program

The program of a liver that has not decompensated.

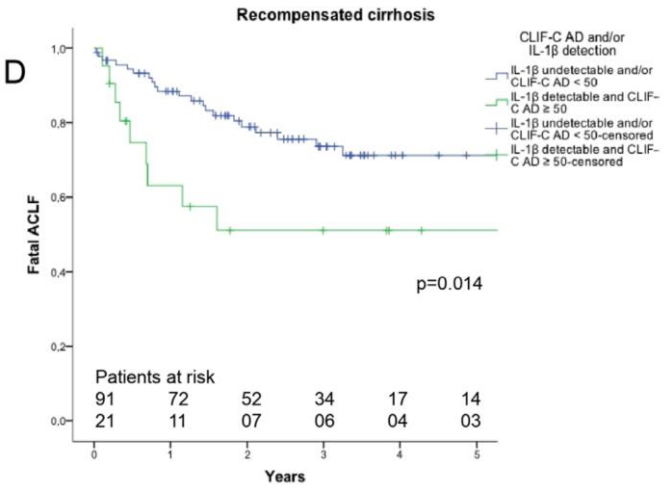
≠

RECOMPENSATED AFTER PRIOR AD

IL-1β

caspase-1 program

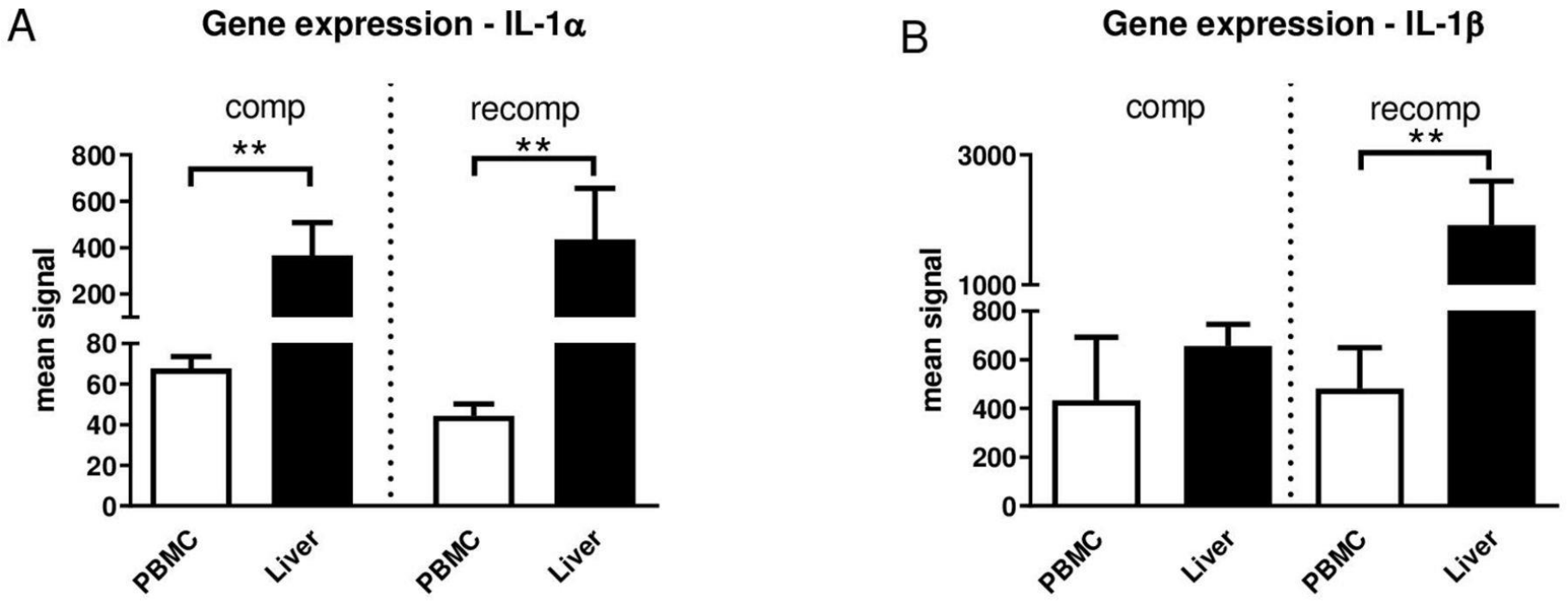
The program linked to higher ACLF in the recompensated liver.



Two different inflammasome programs — so a recompensated liver is a different biological state, not a restored one.

Monteiro, …, Praktiknjo. Gut 2021

The immune scar is in the liver



IL-1α predominant in liver vs. peripheral immune cells — in compensated AND recompensated.
IL-1β predominant in the liver ONLY of recompensated patients — not in compensated.
→ ***Hepatic inflammation persists — IL-1β is the signature unique to recompensation, while systemic inflammation has normalised.***

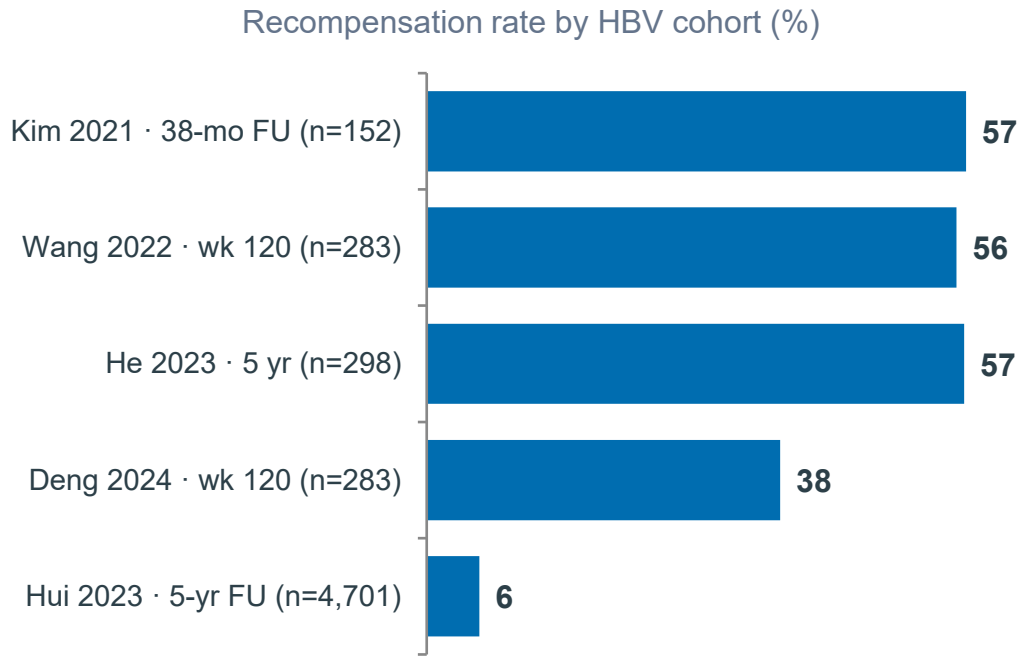
Praktiknjo et al. Gut 2021

2

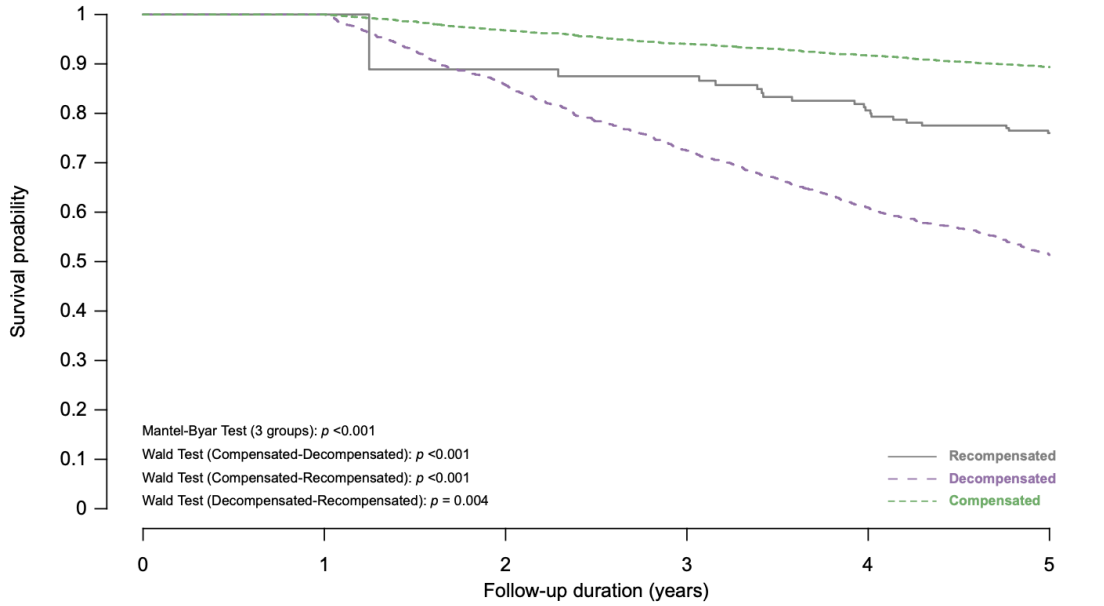
WHO RECOMPENSATES?

Patients, etiologies & predictors

HBV: recompensation rate and survival



Hui's 5.6% counts every NA-treated cirrhotic; the others count decompensated patients followed to a defined endpoint — read denominators carefully.



Number of Deaths						
Recompensated	0	0	1	2	11	19
Decompensated	0	0	152	299	426	527
Compensated	0	0	115	205	277	341

…and survival follows: recompensated transplant-free survival approaches the never-decompensated — n = 4,701 (Hui 2023).

Hui et al. JHEP Rep 2023 · Wang et al. JHEP 2022 · Deng et al. JHEP Rep 2024 · He et al. Hepatol Int 2023 · Kim et al. APT 2021

HBV: predictors and durability

Predictable

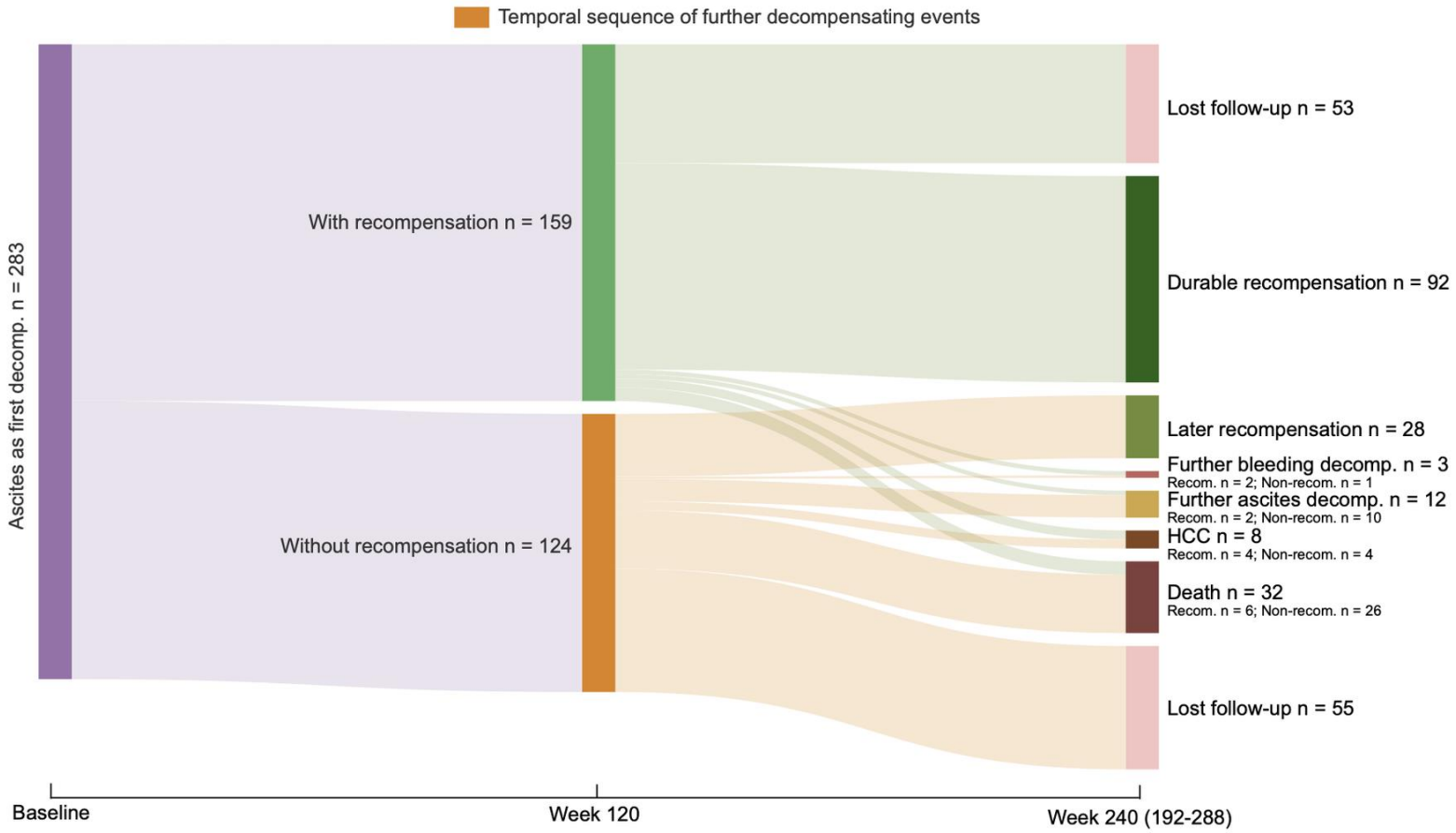
Week-24 albumin ≥ 34 g/L predicts recompensation by wk 120[1]; Brec-PAS & BC2AID scores formalise this[2].

Durable

Recompensation reached by week 120 is still maintained at week 240 (~4.6 yr) in >80% of HBV patients.[1]

Sometimes late

A minority recompensate only after week 120 — uncommon, but real; don't write patients off early.[1]



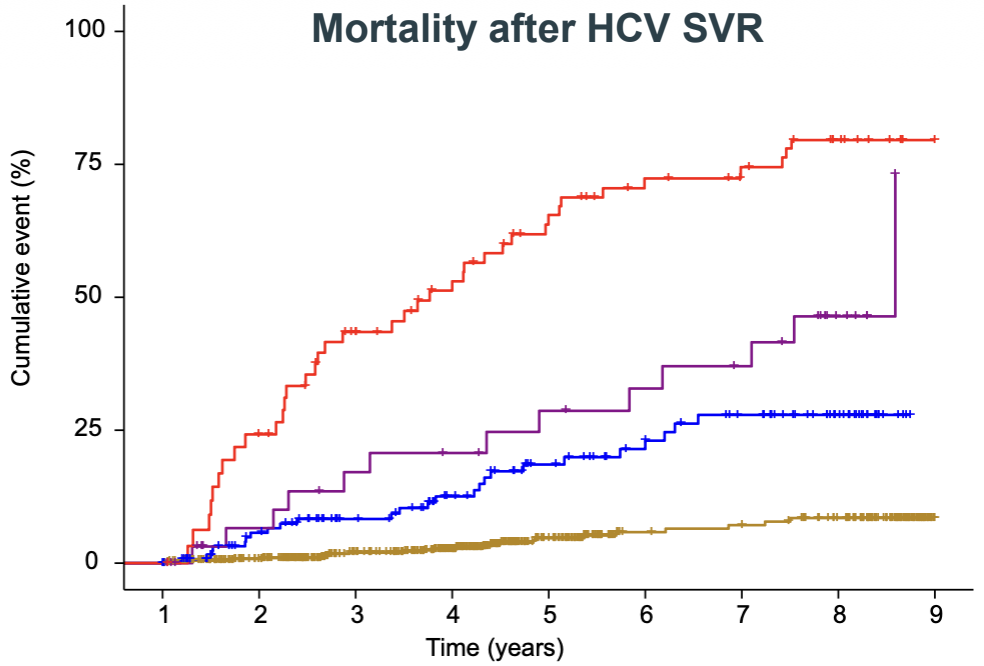
[1] Deng et al. JHEP Rep 2024 *[2] Kim et al. APT 2021*

HCV: recompensation rate and survival

63%

of decompensated HCV patients recompensated after cure (n = 137 of 353)

Two-thirds within 12 months — most recompensation occurs in the first year after SVR; the window is short.
Death and portal-vein thrombosis fall — lower liver-related death and PVT after SVR (Semmler 2025: 36.6% recompensate; Premkumar 2024: ~¼).
Fewer subsequent decompensating events (OR 0.69; OR 0.39 for the number of events).
Survival improves toward — but does not fully reach — compensated cirrhosis.



No. at risk									
Compensated cohort	663	594	470	396	221	144	141	124	1
Recompensated cohort	137	111	92	76	61	50	41	22	0
Decompensated cohort (drugs)	34	27	23	21	18	16	14	6	0
Decompensated cohort (new AD)	26	32	28	28	19	15	12	10	2

Sánchez-Torrijos et al. JHEP 2025 · Semmler et al. CGH 2025 · Premkumar et al. Gastroenterology 2024

ALD: recompensation rate, survival and predictors

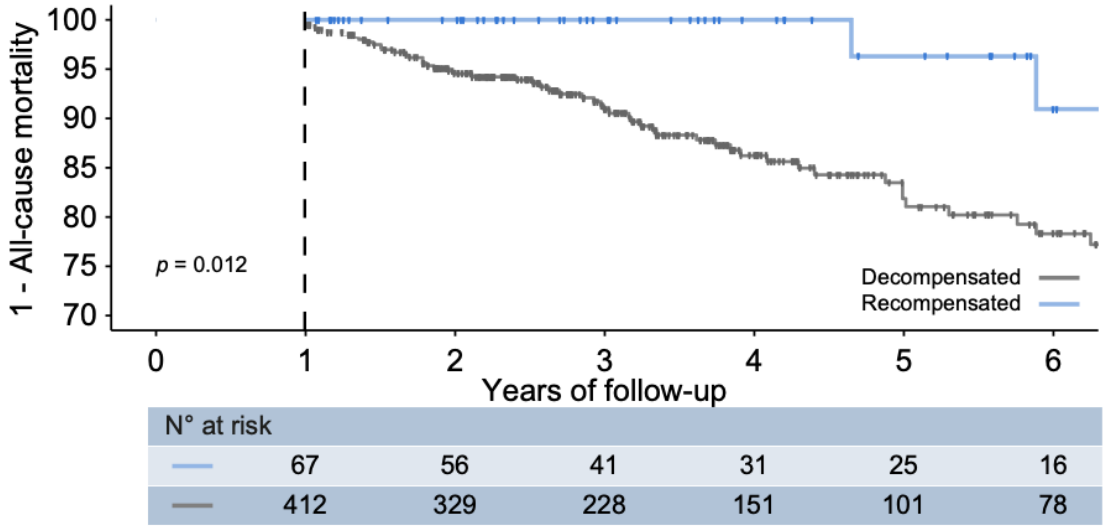
31% recompensate (197/633 · median FU 36 mo)

0.255 adjusted HR for death

Zero liver deaths / HCC if abstinent

Early abstinence wins

Abstinence < 1 month after decompensation — the dominant predictor.



Cohort (Hofer 2026): n=633, 17 centres · recompensation = Child-Pugh A and/or MELD<10 · median follow-up 36 mo after abstinence · 123 deaths (56% liver-related). The benefit is large but conditional on sustained abstinence — relapse erases it.

Hofer et al. JHEP 2026

Baveno-defined recompensation: HBV, HCV & ALD — predictors

Etiology	Key predictor(s)	Tool / score
HBV	Early treatment; wk-24 albumin ≥34 g/L; ALT, INR, bilirubin	BC2AID[1]; Brec-PAS[2]
HCV	Lower Child-Pugh; fewer prior decompensations; albumin	Child-Pugh components
Alcohol	EARLY abstinence (<1 mo); lower MELD/HVPG	Random-survival-forest[3]

Baveno VIII (7.9–7.12): lower severity (CTP / MELD), no further decompensation, no high-risk varices, and a shorter time to aetiologic therapy all favour recompensation.

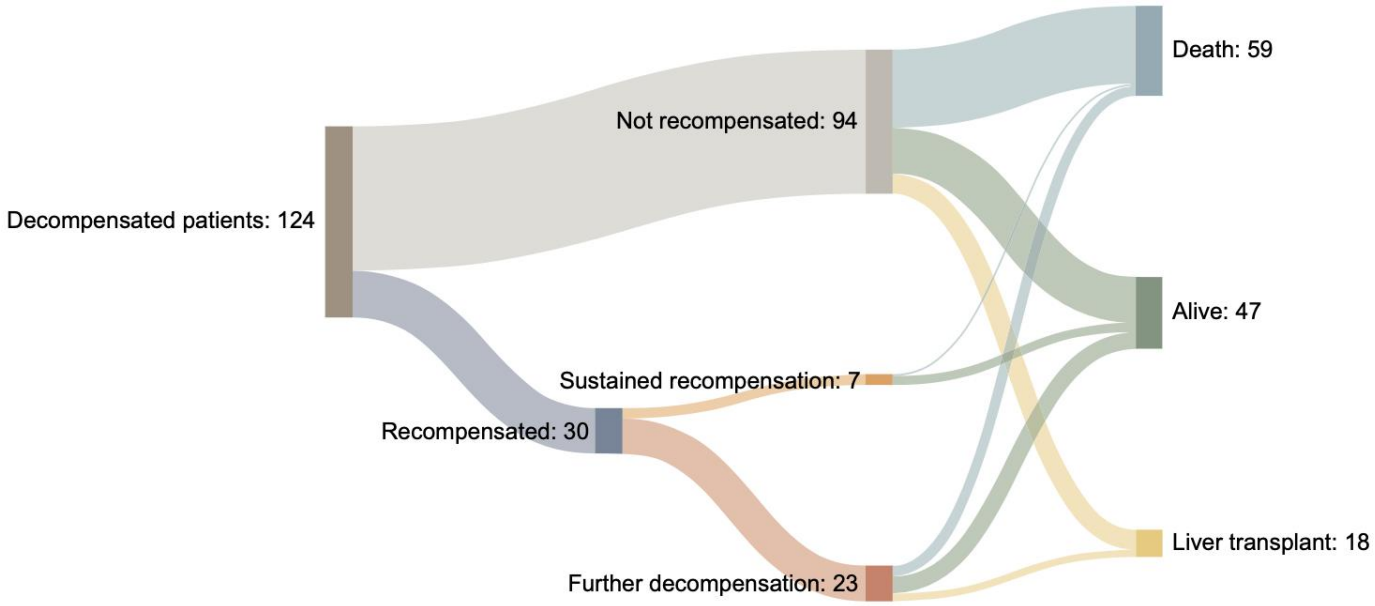
[1] Kim et al. APT 2021 [2] Deng et al. JHEP Rep 2024 [3] Hofer et al. JHEP 2026 · Baveno VIII (JHEP 2026)

3

WHAT DRIVES IT?

Therapeutic interventions — aetiologic and beyond

MASLD: recompensation rate and durability



24% 2-year cumulative recompensation · 124 pts

73% re-decompensation

But it rarely lasts — the survival signal vanished after adjustment.

No definition of controlled etiology!

Predictors: lower MELD-Na · higher albumin · single decompensation.

Cohort & definition: 124 pts · mean age 69 · 61% Child-Pugh B · MELD-Na 11 · enrolled at first decompensation · median follow-up 2.1 yr. Recompensation = Baveno VII — but MASLD has no discrete etiological-cure threshold; recovery tracked baseline liver function, which is why the definition is softest here.

Jiménez-Masip et al. HepCom 2026 · Feng et al. Trends Endocrinol Metab 2025

MASLD: impact of weight loss and predictors

18.6% recompensate (64/344 · median 15 mo · Baveno VII)

MASLD 17.7% · MetALD 19.4% · 3.9-yr follow-up

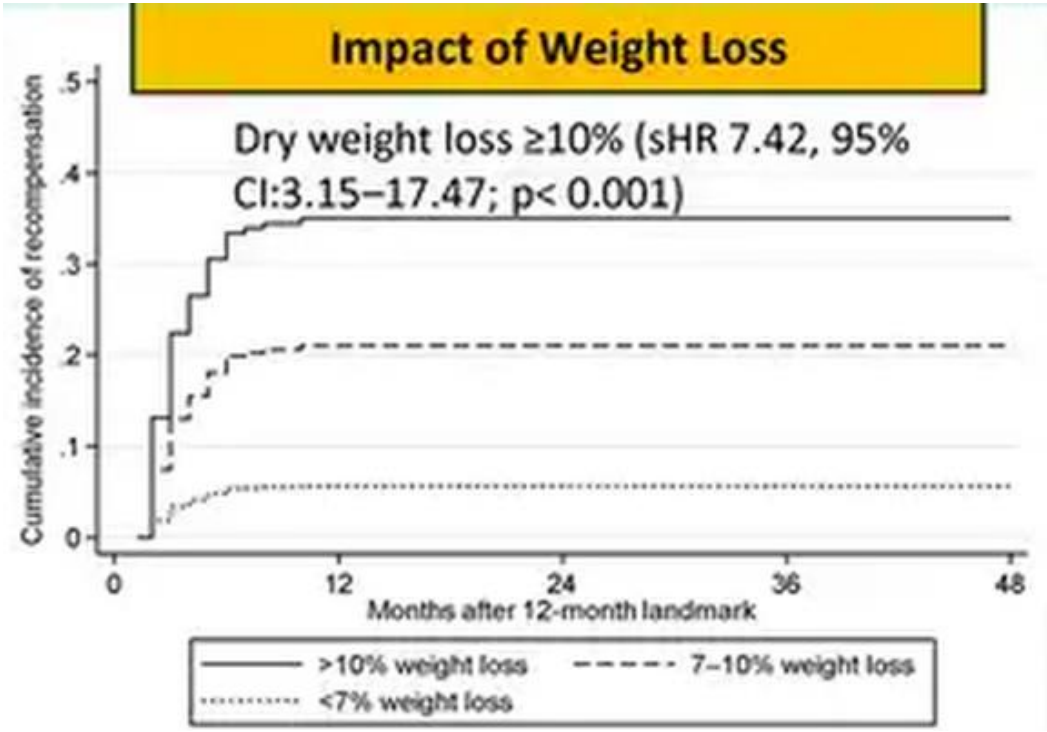
PREDICTORS (MELD-Na-adjusted)

Dry-weight loss ≥ 10% — sHR 7.4 (p < 0.001)

No large varices — sHR 2.0

PEth-verified abstinence — sHR 1.9

Glycemic control — sHR 1.4



Cohort : prospective, n = 344 (158 MASLD, 186 MetALD) · integrated weight-loss, metabolic & alcohol programme · mortality 12.0% (MASLD) vs 22.6% (MetALD).

Premkumar et al. Hepatology 2026

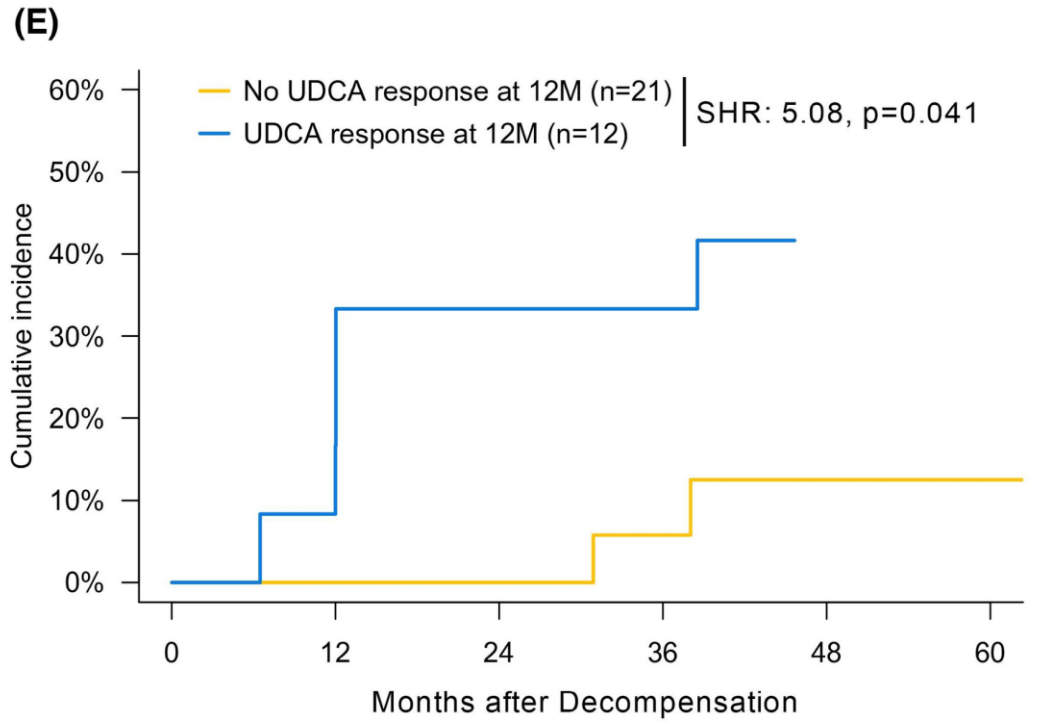
PBC: recompensation rate and predictors

PBC

~20% recompensate over 5 years[1]

Paris-II UDCA response — recompensation almost exclusively in responders (SHR 5.08, p = 0.041)[1].

Outcome benefit so far only numerical — TFS HR 0.46 (p = 0.34; n = 7)[1]. LMR predicts recompensation (AUC 0.79)[2].



Cumulative recompensation by UDCA response at 12 months[1]

[1] *Hofer et al. APT 2024* [2] *Lin et al. APT 2025*

AIH: recompensation rate, survival and predictors

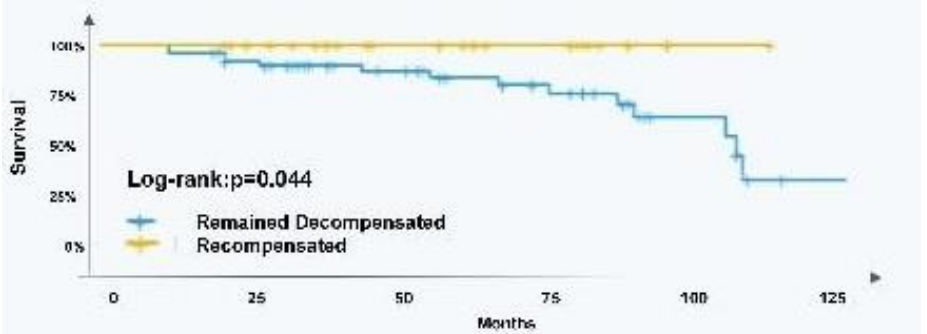
AIH

34.6% recompensate (28/81, biopsy-proven) over a median follow-up of ~5 yr (61.8 mo)

Complete biochemical response predicts it (HR 1.90); diabetes predicts failure (HR 0.58) — and survival is superior (log-rank p = 0.044).

Recompensated patients: lower IFN-γ, IL-17A, TNF-α — inflammation tracks the state.

No curative therapy → definitions are borrowed, not validated. Plausible, but on the frontier.



- **Factors associated with recompensation**

Factors	Multivariate analysis	
	HR (95%CI)	p-value
BMI	1.161 (1.022-1.326)	0.025
Diabetes	0.582 (0.294-0.896)	0.004
ALT	1.168 (1.216-1.365)	0.028
ALB	1.388 (1.195-1.635)	<0.001
CBR	1.895 (1.154-2.312)	0.014

Zhang et al. Ann Hep 2026

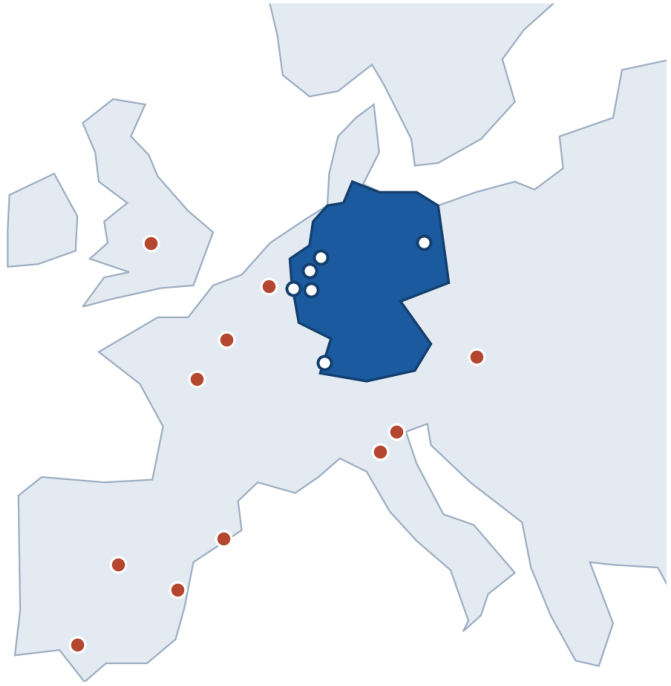
Predictors across all etiologies

Etiology	Key predictor(s)	Tool / score
HBV	Early treatment; wk-24 albumin ≥34 g/L; ALT, INR, bilirubin	BC2AID[1]; Brec-PAS[2]
HCV	Lower Child-Pugh; fewer prior decompensations; albumin	Child-Pugh components
Alcohol	EARLY abstinence (<1 mo); lower MELD/HVPG	Random-survival-forest[3]
PBC	*UDCA / Paris-II response; higher lymphocyte-to-monocyte ratio*	*LMR, AUC 0.79[4]*
AIH	*Complete biochemical response at 6 mo; no diabetes*	*IAIHG response criteria*
MASLD	*Dry-weight loss ≥10% (sHR 7.4); PEth-verified abstinence; glycemic control*	*Prospective cohort[5]*

Blue: Baveno VIII-defined recompensation (HBV, HCV, ALD) · red: beyond the definition — predictors and criteria still being validated.

[1] Kim APT 2021 [2] Deng JHEP Rep 2024 [3] Hofer JHEP 2026 [4] Lin APT 2025 [5] Premkumar Hepatology 2026 · Baveno VIII (JHEP 2026) · Zhang Ann Hep 2026

TIPS can facilitate recompensation in advanced decompensation stages



■ GCSG — Germany ● EuroTIPS centres ○ GCSG centres

EuroTIPS — derivation

242 decompensated patients

10 European centres · standardized protocol · refractory ascites / variceal bleeding

TIPS

11.2%

recompensated (27/242)

German Cirrhosis Study Group — validation

864 decompensated patients

7 centres · real-world · refractory ascites / variceal bleeding

TIPS

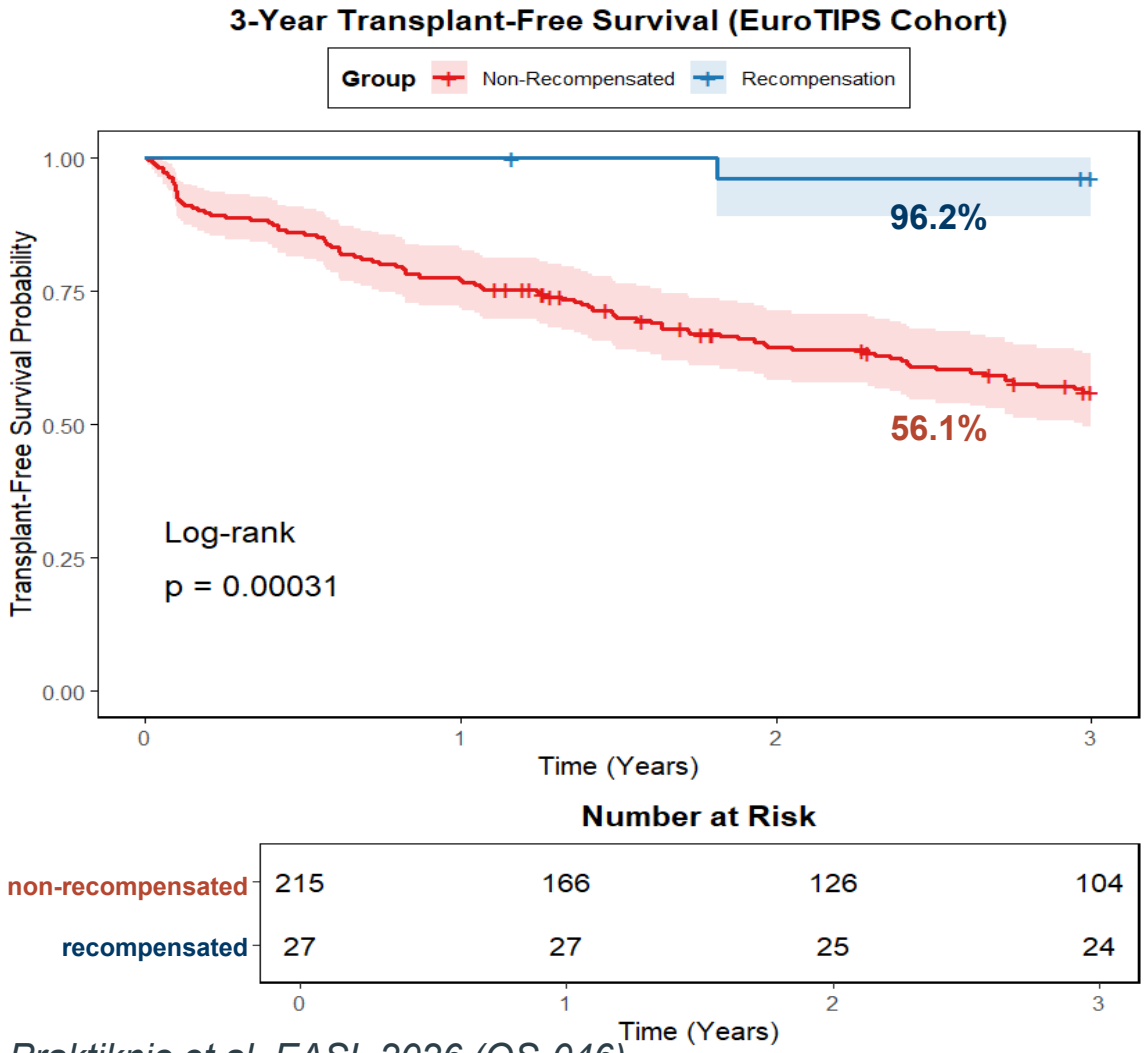
10.2%

recompensated (88/864)

Two independent multicentre cohorts · viral, alcohol-related and autoimmune cirrhosis

Praktiknjo et al. EASL 2026 (OS-046)

Post-TIPS recompensation: survival and predictors



Praktiknjo et al. EASL 2026 (OS-046)

Recompensated patients were less sick at TIPS — lower MELD & Child-Pugh, higher albumin, far less prior HE.

MELD HR 0.95 (p = 0.048)
Each MELD point lowered the chance of recompensation.

Alcohol HR 0.58 (p = 0.017)
Alcohol etiology independently predicted non-recompensation — consistent with the viral > alcohol gradient.

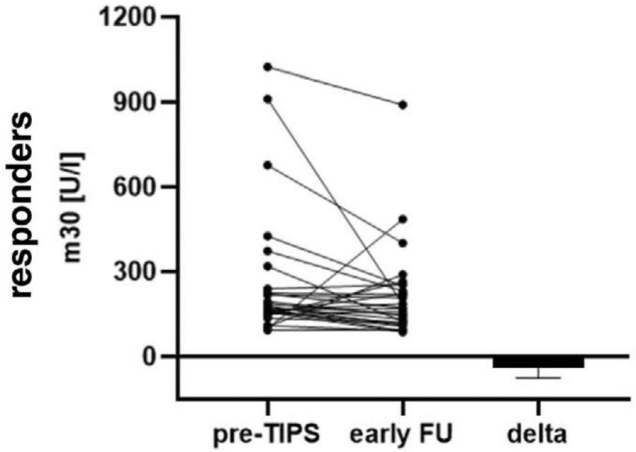
A large, reproducible signal — and the starting point (MELD, etiology) decides who gets there. Multivariable Cox, both cohorts.

TIPS reduces systemic inflammation and cell death

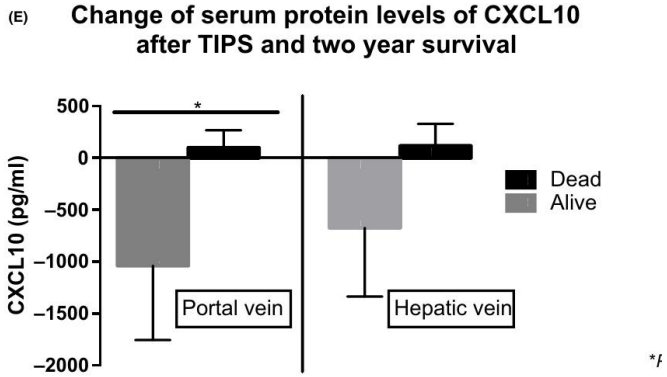
Why does TIPS facilitate recompensation in such advanced patients?

It not only abolishes portal hypertension — it also reduces systemic inflammation (CXCL10)[2] and cell death (m30)[1].

Cell death (m30) falls[1]

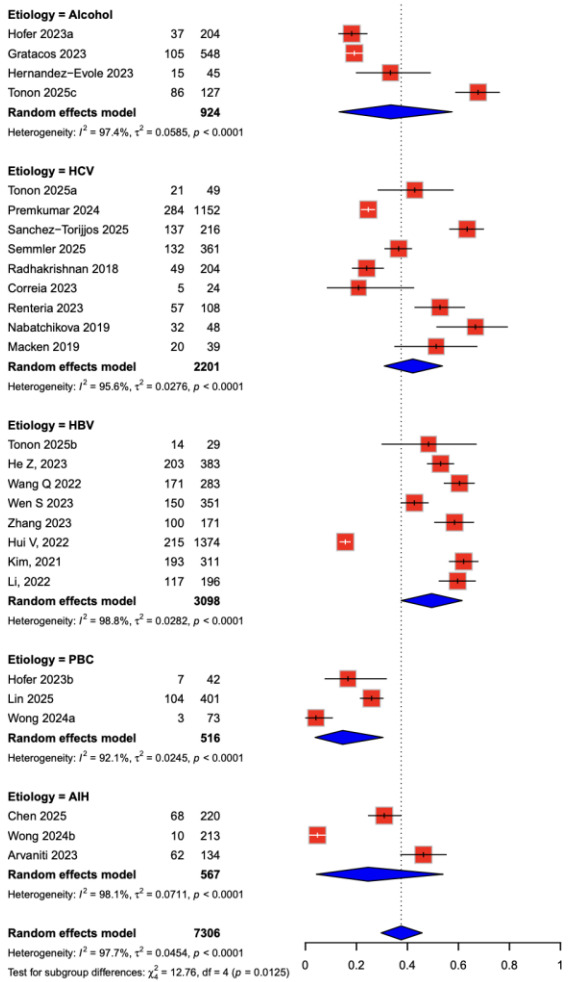


Inflammation (CXCL10) falls[2]



[1] *Piecha et al. Liver Int 2024* [2] *Lehmann, …, Praktiknjo et al. Liver Int 2017*

Metaanalysis across etiologies



≈1 in 3

35% pooled (95% CI 26–45%)

27 studies · 9,063 patients · highest HBV 50%, lowest PBC 15%

67% lower odds of death

79% lower liver-related death

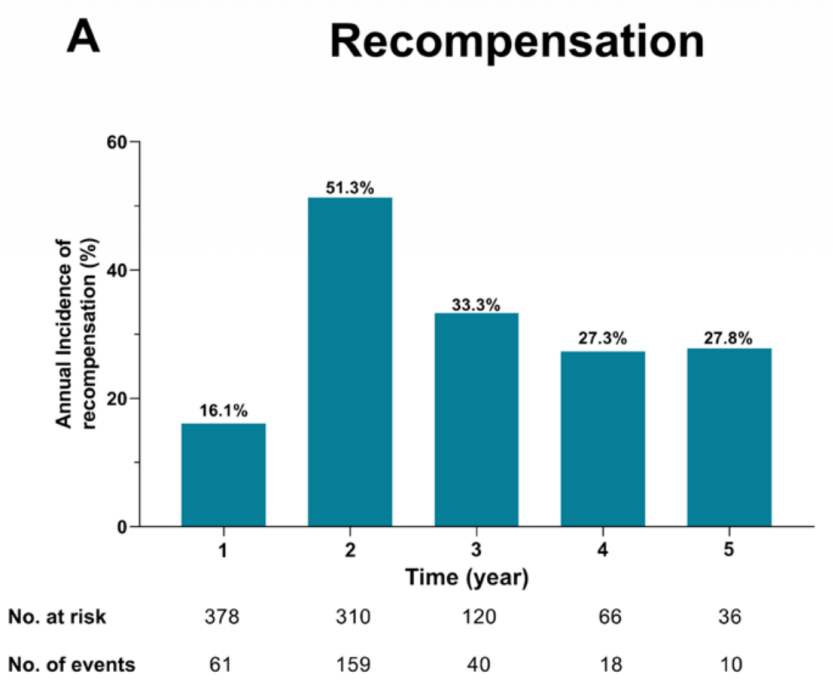
Transplant need trended lower (ns) · low-certainty evidence

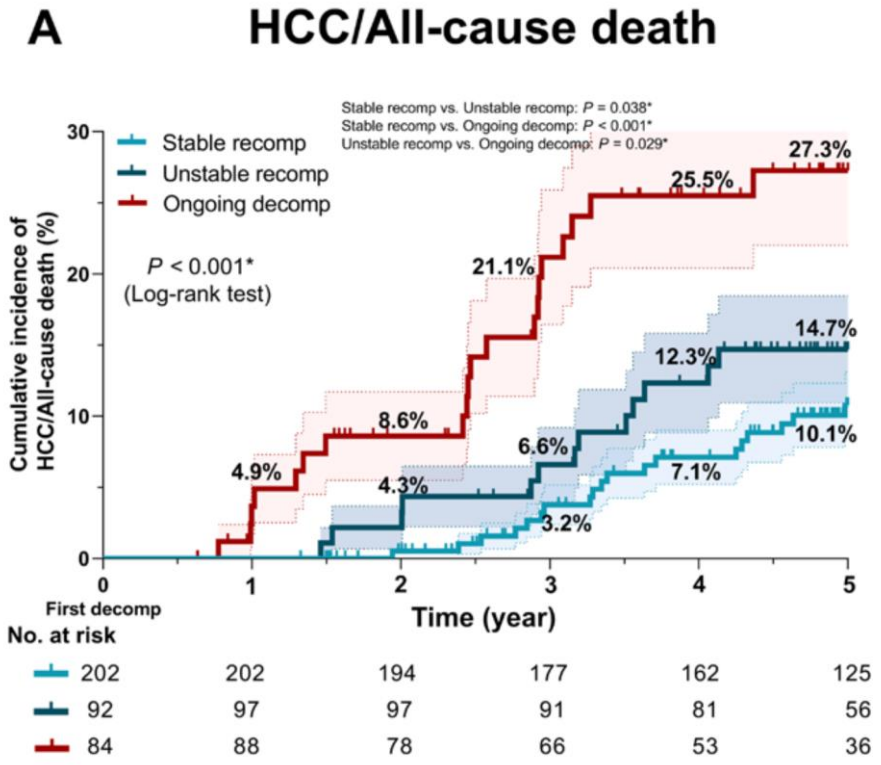
A general phenomenon — across every etiology.

Caution: I² ≈ 98% — read 35% as a centre of gravity, not a constant.

Goh et al. CGH 2026 · Premkumar et al. Lancet Gastroenterol Hepatol 2025

Re-decompensation after recompensation





24% re-decompensate — 53% stay stably recompensated[1].
5-yr HCC / death: 14.7% vs 10.1% (p = 0.038).
Continue 6-monthly follow-up (Baveno VIII 7.13).

Cohort: HBV-related decompensated cirrhosis · n = 378 (two cohorts) · assessed every 6 months for up to 5 years · median follow-up 5.3 yr · 77.8% recompensated (Baveno VII).

Xia et al. APT 2026

4

WHY YOU SHOULD CARE?

Clinical Implications of Recompensation

Surveillance after recompensation: HCC risk decreases differently

Reduced after recompensation

HBV — falls to the compensated level (aHR 1.41, ns)[1].
Alcohol — zero HCC while abstinent (0 vs 16)[2].
Pooled — 45% lower (OR 0.55)[3].

NOT reduced

HCV — 3.9 vs 5.5 per 100 patient-yr[4] — still > 2× the surveillance threshold.
Risk ≈ decompensated[5] — carcinogenesis is uncoupled from functional recovery.

Baveno VIII (7.13–7.14): risk falls but persists — 6-monthly surveillance & hepatologist follow-up continue. No etiology has de-escalation evidence yet.

[1] Zhang et al. HepCom 2024 [2] Hofer et al. JHEP 2026 [3] Goh et al. CGH 2026 [4] Semmler et al. CGH 2025 [5] Sánchez-Torrijos et al. JHEP 2025 [6] Baveno VIII (JHEP 2026)

Non-acute decompensation is distinctly worse

1-yr transplant-free mortality²

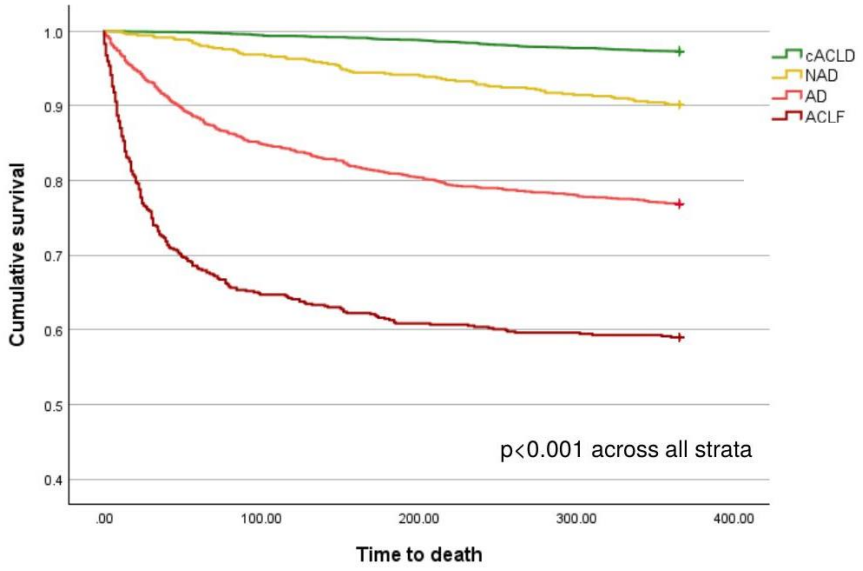
Compensated / Recompensated (off therapy) — 1.6%
recompensated ≈ compensated (HR 0.97, ns)¹

NAD grade 1 — diuretic-controlled ascites — 3.8%
'dry on spironolactone' — the state in question

NAD grade 2 — grade 1 ascites — 11.5%
minimal fluid on imaging only

NAD grade 3 — grade 2 ascites / HE 1–2 — 13.1%
the 'strict' NAD phenotype

Acute decompensation — hospitalised
the gateway to ACLF



McCoy et al. multicentre data (n = 2,077): NAD sits between cACLD and AD across the full spectrum (p < 0.001)².

A patient dry ON diuretics is NAD grade 1, not recompensated — the taper is the ladder between them (Baveno VIII 7.6–7.7).

¹ Tonon et al. JHEP 2025 ² McCoy, …, Praktiknjo. submitted

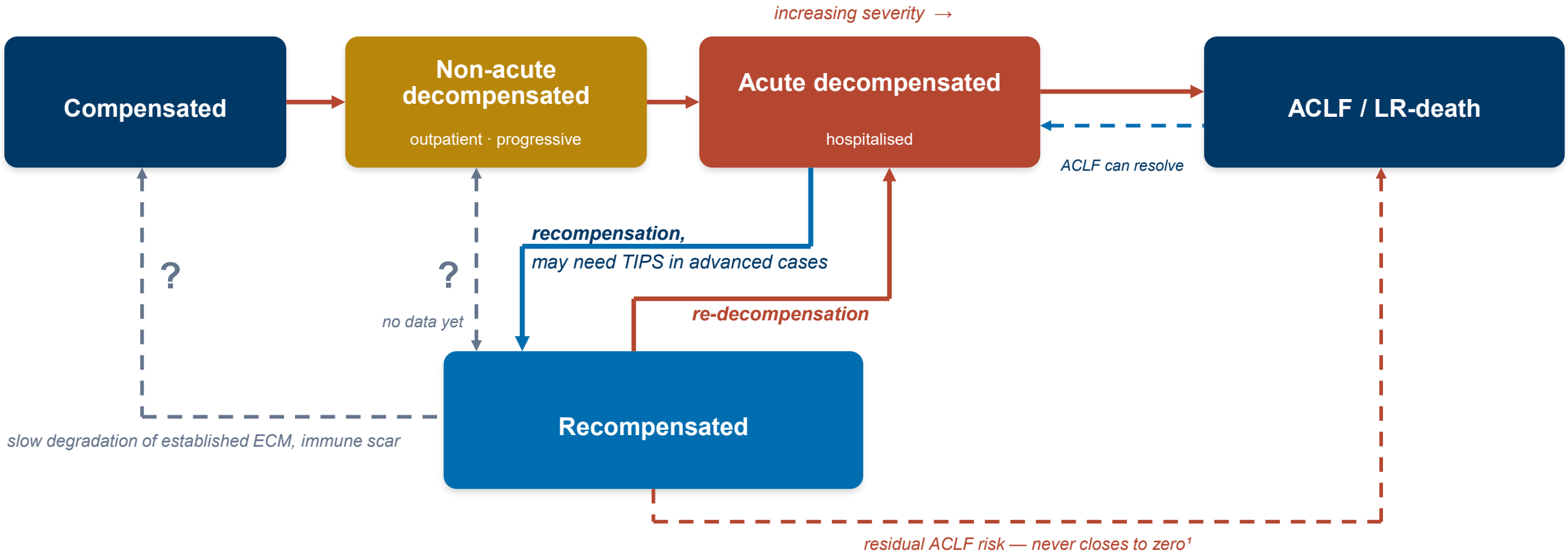
Clinical consequences

HCC surveillance	**CONTINUE**	6-monthly imaging (7.14) — risk falls but never reaches zero.
Diuretics	**ACTIVELY TAPER**	Withdrawal IS the diagnostic test (7.6) — and without ascites they only harm: AKI, hyponatraemia, falls.
Lactulose / rifaximin	**ACTIVELY TAPER**	HE must resolve OFF therapy (7.7). Persistent need = NAD, not recompensation.
NSBB	**INDIVIDUALISE**	Rule out CSPH by NITs first — LSM < 10, or LSM < 15 + SSM < 25 (7.19); otherwise continue.
Varices	**CONFIRM CSPH**	Persistent varices alone ≠ CSPH; if NITs don't rule it out, measure HVPG / EUS-PPG.
Transplant listing	**REASSESS**	Delisting is possible — but only on a durable trajectory, not one good MELD.
Follow-up interval	**DON'T RELAX**	6-monthly hepatologist follow-up (7.13) — a state to maintain, not a discharge.

Recompensation changes the odds, not the vigilance.

Baveno VIII (JHEP 2026), 7.6–7.7 / 7.13–7.19

Simplified stage model



¹ Monteiro, …, Praktiknjo. Gut 2021 ² Xia et al. APT 2026 ³ McCoy, …, Praktiknjo. submitted

Take home messages

1 · How does it happen?

Control of the cause improves function & inflammation.
Structural changes persist: established fibrosis often with CSPH, immune scar.

2 · Who recompensates?

About 1 in 3, depending on etiology. HBV, HCV and alcohol are the only etiologies with Baveno VIII criteria.
Predictors: lower baseline severity and early aetiologic control.

3 · What drives it?

Aetiologic cure; TIPS extends it to advanced stages.
Actively de-prescribe diuretics and HE drugs but keep 6-monthly surveillance.

Thank you

Questions & discussion

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