Alcohol-Related Liver Disease: The Spectrum of Reversibility

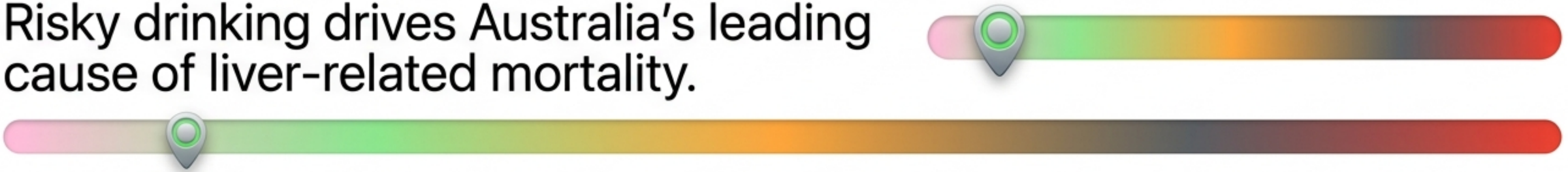
A definitive clinical pathway for early interception, diagnosis, and management in primary care.

Target Audience: Primary Care & Allied Health

Content: Current Australian Standard of Care

Read Time: ~10 minutes

Risky drinking drives Australia's leading cause of liver-related mortality.



1.4 Million

Australians exceed NHMRC guideline limits (>10 standard drinks/week).

~2,600 Deaths

Annual alcohol-attributable liver disease mortality (AIHW, 2023).

30-35%

Proportion of all Australian liver transplants due to ALD.

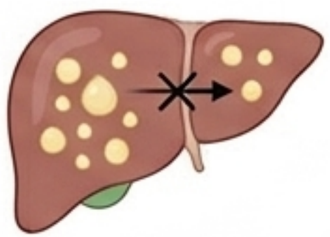
40-65 Years

Peak incidence age. Predominantly men, but female incidence is rising rapidly.

The Natural History is Highly Variable: While 90% of heavy drinkers develop steatosis, only 10-20% progress to cirrhosis over decades. The clinician's goal is to intercept the disease before irreversible structural damage occurs.

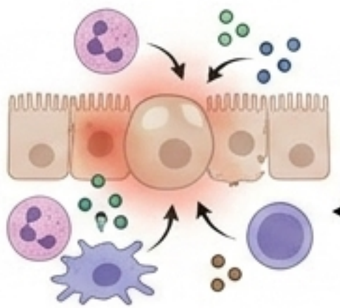
The Pathophysiology Cascade is driven by oxidative stress and inflammation.

Hepatic Steatosis



Acetaldehyde inhibits β-oxidation. Fat droplets form in hepatocytes.

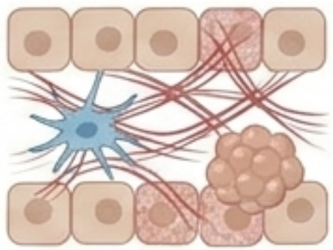
Steatohepatitis (ASH)



Gut endotoxins (LPS) activate Kupffer cells via TLR4. Cytokines recruit neutrophils. Hepatocyte ballooning occurs.

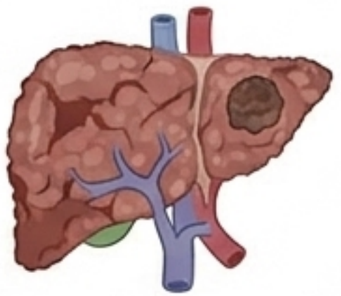
100% Reversible with Abstinence

Fibrosis & Cirrhosis



Persistent inflammation activates stellate cells. Collagen creates bridging fibrosis and regenerative nodules.

Decompensation & HCC

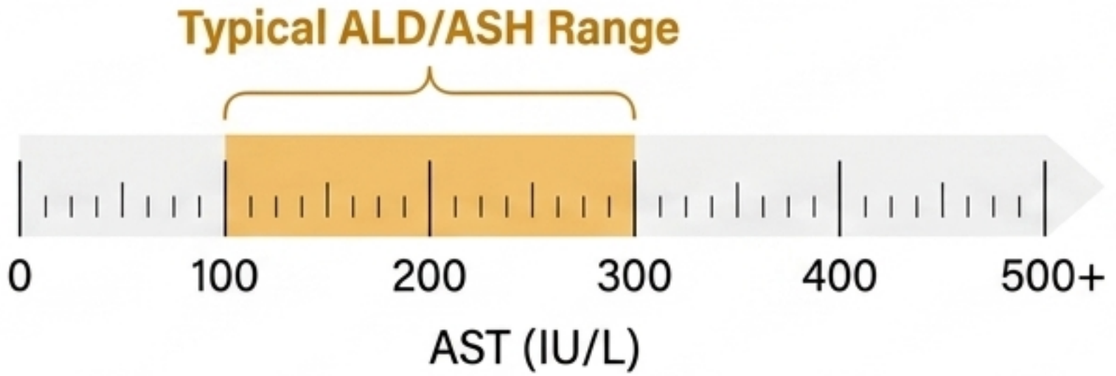


Sinusoidal remodelling drives portal hypertension and a 1-6% annual HCC risk.

PREMIUM CLINICAL DASHBOARD

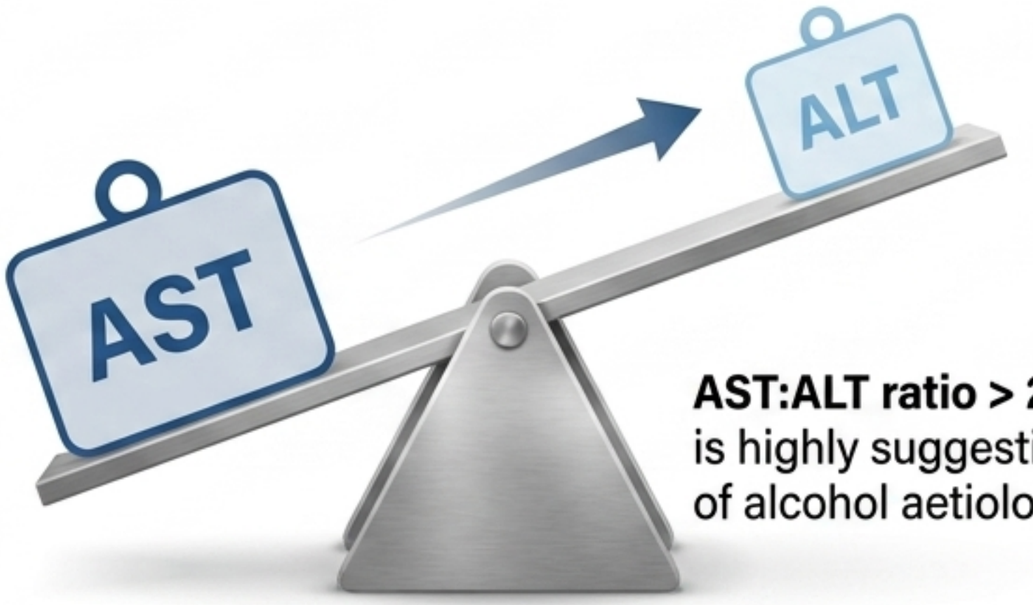
The LFT Fingerprint of ALD exhibits a distinct AST:ALT inversion

The Diagnostic LFT Scale



GGT: The most sensitive marker of recent heavy drinking

The Rule-In Ratio



AST:ALT ratio > 2 is highly suggestive of alcohol aetiology

"Red Flag" Alert Box

AST > 300 IU/L is **atypical for ALD alone.** Do not assume ALD. Immediately evaluate for alternative aetiologies: Acetaminophen overdose, viral hepatitis, ischaemic hepatitis, or autoimmune hepatitis.

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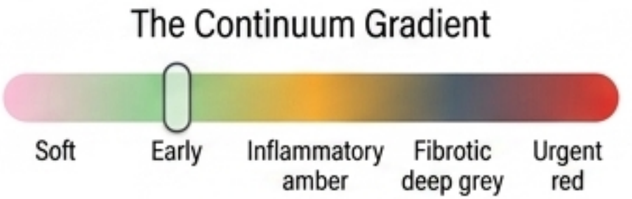
The Screening Arsenal: Match the tool to the clinical setting.

Tool	Items	Threshold	Optimal Setting
AUDIT-C	3 Items	≥3 (women), ≥4 (men)	Primary care - rapid initial screen. Embedded in GP software.
Full AUDIT	10 Items	≥8 (hazardous), ≥20 (dependence)	Confirmed risky drinking; quantifies risk and guides intervention intensity.
FAST	4 Items	≥3 total	Emergency Departments.
CAGE	4 Items	≥2 positive	Quick bedside screen (less sensitive).

Recommended Approach: Use AUDIT-C as the frontline trigger. If positive, cascade to Full AUDIT to dictate the intervention tier.

Brief interventions effectively intercept mild to moderate risky drinking.



NHMRC Risk Thresholds

Low Risk	≤4 occasion / ≤10 week
Risky	>4 occasion / >10 week
High Risk	>6 frequent / >20 week men, >15 women

The FRAMES Framework for Brief Intervention

- **F** **Feedback:** Personalize LFT/AUDIT results.
- **R** **Responsibility:** Emphasize patient autonomy.
- **A** **Advise:** Clear, non-judgemental advice to reduce/cease.
- **M** **Menu:** Offer goal-setting, self-monitoring, or referral.
- **E** **Empathy:** Reflective listening.
- **S** **Self-efficacy:** Build confidence in changing habits.

MBS Items **2700/2701** fund focused psychological strategies in GP settings.

Non-invasive evaluation stages fibrosis risk and dictates referral.

The FIB-4 Calculator

(Age × AST / Plt × $\sqrt{ALT}$)

Value: < 1.3 | **Interpretation:** Low risk. Repeat 2-3 years if drinking continues.

Value: 1.3 – 3.25 | **Interpretation:** Indeterminate. Refer for elastography.

Value: > 3.25 | **Interpretation:** High probability of advanced fibrosis/cirrhosis. Urgent hepatology referral.

Transient Elastography (FibroScan®)

(MBS 13020/55063)

Value: < 7.5 kPa | **Interpretation:** Excludes advanced fibrosis.

Value: > 12.5 kPa | **Interpretation:** Suggests established cirrhosis.

Value: > 20.0 kPa | **Interpretation:** Highly suggestive of clinically significant portal hypertension.

Clinical Pearl

Thrombocytopenia (Platelets <150 × 10^9/L) is often the earliest red flag for portal hypertension and hypersplenism.

The ALD Clinical Continuum: From silent steatosis to systemic failure.

Stage 1: Steatosis

Setting: Primary Care

Presentation: Usually asymptomatic, incidental fatty liver on US. AST/ALT often normal.

Stage 2: Steatohepatitis (ASH)

Setting: Hospital/Hepatology

Presentation: RUQ pain, hepatomegaly, anorexia. LFTs elevated (AST 100-300).

Stage 3: Compensated Cirrhosis

Setting: Hepatology Outpatient

Presentation: Clinically silent or spider naevi, thrombocytopenia. Patients feel well.

Stage 4: Decompensated Cirrhosis

Setting: Tertiary/Transplant

Presentation: Ascites, variceal bleeding, hepatic encephalopathy, jaundice. MELD >15 carries >20% 1-year mortality.

Severe Alcoholic Hepatitis (SAH) is a distinct, high-mortality clinical syndrome.

The SAH Clinical Presentation

- Rapid onset jaundice (bilirubin >80 µmol/L)
- Fever & tender hepatomegaly
- Neutrophil leukocytosis
- History of heavy drinking (>80g/day for >6 months, often lagging 4-8 weeks post-binge)

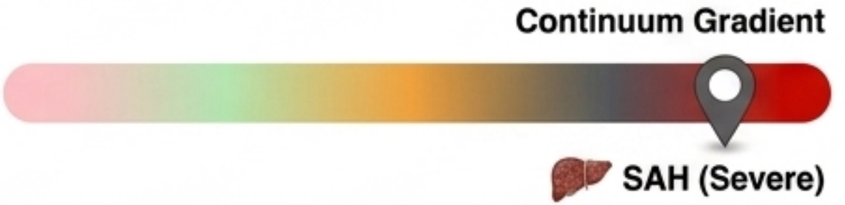
Severity Scoring Matrix

1 **Maddrey Discriminant Function (DF):**
PT difference + bilirubin.
Threshold: ≥ 32 triggers corticosteroid consideration.

2 **MELD Score:**
Bilirubin, INR, creatinine.
Threshold: ≥ 21 predicts 90-day mortality.

3 **Lille Model:**
Futility check based on Day 7 bilirubin.
Threshold: ≥ 0.45 means non-responder.

The SAH Treatment Algorithm dictates early steroids and a strict Day 7 futility gate.



The Infection Check
Blood/urine cultures, CXR, ascitic tap (PMN >250 = SBP).

Infection Present
Treat infection, STOP steroids

No Infection
Continue to Step 2.

Initiation
Prednisolone 40 mg/day PO (Prefer prednisolone over prednisone to bypass hepatic conversion).
Ensure nutritional support (35 kcal/kg/day, do NOT restrict protein).

The Day 7 Lille Score
Assess treatment response.

Lille < 0.45
Continue prednisolone to complete 28-day course.

Lille ≥ 0.45
STOP steroids (futile, <20% 6-month survival). Discuss palliative care or transplant.

Do Not Do

Pentoxifylline is obsolete. The STOPAH trial (2015) proved no mortality benefit. Do not prescribe.

Alcohol Withdrawal Syndrome (AWS) management scales with CIWA-Ar severity.

Mild

CIWA-Ar < 10

Onset: 6-12h

Symptoms: Tremor, anxiety, insomnia.

Setting: Outpatient/GP with close follow-up.

Moderate

CIWA-Ar 10-20

Onset: 12-48h

Symptoms: Pronounced tremor, diaphoresis, tachycardia, transient hallucinations.

Setting: Hospital or supervised detox unit.

Severe / DTs

CIWA-Ar > 20

Onset: 48-96h

Symptoms: Seizures, delirium tremens, autonomic instability, severe hallucinations. ⚠️

Mortality 5-15% untreated.

Setting: ICU/HDU admission.

Benzodiazepine selection in withdrawal must account for hepatic clearance.

Benzodiazepine Selection

Diazepam / Chlordiazepoxide
Standard first-line.
Caveat: Active metabolites accumulate in renal/severe hepatic impairment.

Lorazepam (The Hepatic Preference)
1-2 mg QID PRN.
Advantage: Undergoes glucuronidation only; does not accumulate in decompensated cirrhosis.

The Thiamine Imperative

300-500 mg IV TDS for 3-5 days

Never give glucose before thiamine. In dependent drinkers, giving glucose without preceding or concurrent IV thiamine can precipitate **irreversible Wernicke-Korsakoff syndrome.**

The AUD Pharmacotherapy Cheat Sheet: Match the agent to organ function.

Drug	Mechanism	Adult Dose	Hepatic Rules	Renal Rules	Key Contraindications
Naltrexone (Oral/Depot)	Opioid antagonist	50mg daily	Avoid in decompensated cirrhosis/acute failure	Standard monitoring	PBS Authority Concurrent opioids
Acamprosate	NMDA modulator	666mg TDS	Safest in liver disease (no hepatic metabolism)	Contraindicated if eGFR < 30	PBS Authority Severe renal failure
Disulfiram	Aldehyde dehydrogenase inhibitor (aversive)	200mg daily	Absolutely contraindicated in hepatic impairment	Standard monitoring	PBS General Psychosis/CVD

The Cirrhosis Care Plan requires proactive management of portal hypertension.

The Continuum Gradient

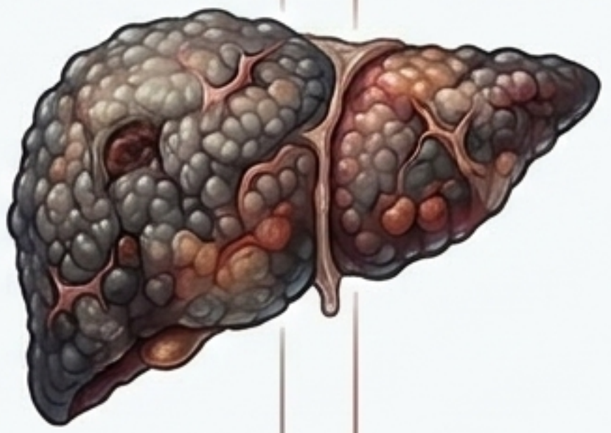
CIRRHOSIS (FIBROTIC)

Variceal Bleeding Risk

OGD (endoscopy) at diagnosis.

Prescribe non-selective β-blockers (carvedilol/propranolol) for medium/large varices.

- **Baveno VII Criteria:** FibroScan <20 kPa + Platelets >150 safely excludes high-risk varices.



HCC Surveillance

Mandatory 6-monthly ultrasound ± AFP. 1-6% annual risk.

Ascites & Hepatorenal Syndrome

Diagnostic tap to r/o SBP. Type 1 HRS requires urgent terlipressin + albumin and transplant referral.

Abstinence & Transplant

MELD >15 triggers transplant assessment. Requires strict abstinence (usually 6 months).

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Standard ALD guidelines require strict modifications in vulnerable populations

Pregnancy

Teratogenic risk (FASD)

Naltrexone/Acamprosate/Disulfiram contraindicated

Multidisciplinary care essential

GGT unreliable physiologically; use PEth/CDT

Paediatrics & Adolescents

Screen with CRAFFT/AUDIT-C

AUD pharmacotherapies NOT approved for <18

Psychosocial interventions are first-line

The Elderly

Increased vulnerability due to reduced hepatic blood flow and polypharmacy

High falls risk with benzodiazepines (reduce dose 50%, prefer short-acting oxazepam/lorazepam)

FIB-4 less reliable

Immunocompromised

HIV/HCV co-infection **accelerates fibrosis**

Careful coordination required with ARV hepatotoxicity

ALD is a leading driver of the Indigenous health gap in Australia.

The Disparity

Aboriginal and Torres Strait Islander people face a **3.1× higher rate of alcohol-attributable liver hospitalizations, younger onset of ALD,** and **higher mortality in remote areas** (NT, WA, QLD).

Barrier 1: The Geography Gap

Remote communities are often >500km from hepatology services. FibroScan is rarely available locally.

Barrier 2: The Co-infection Multiplier

Hepatitis B prevalence is 5-10× higher in Indigenous communities, severely compounding ALD fibrosis.

Barrier 3: Stigma & Trauma

Intergenerational trauma and shame-based framing inhibit engagement with standard clinical models.

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Closing the Gap requires culturally safe, community-led clinical models.

Solution 1: ACCHOs & AHWPs

Integration of Aboriginal Community Controlled Health Organisations and Health Workers builds trust. Frame conversations around culture and connection, not blame.

Solution 2: Telehealth & Biomarkers

Utilize MBS items 99-110 for remote specialist access. Substitute inaccessible FibroScan with serum biomarkers (FIB-4, ELF).

Solution 3: Universal HBV Screening

Mandatory HBsAg, anti-HBc, and anti-HBs checking. Initiate NIP-funded catch-up vaccinations or PBS-listed antivirals immediately.

Solution 4: MBS 715 Health Checks

Embed screening and brief interventions into every annual Indigenous health check.

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Objective biomarkers validate self-reporting and monitor treatment adherence.

EtG (Urine)
Detects very recent intake.

EGG (Urine)
Detects *vew* recent intake.

PEth (Phosphatidylethanol)
High Sensitivity/Specificity. Direct metabolite, definitive proof, but emerging availability.

CDT (Carbohydrate-deficient transferrin)
High Specificity. Best currently available marker for heavy drinking (MBS 66832).

GGT
Moderate Specificity. Easily confounded by obesity, cholestasis, or drugs.

Note: Self-report (AUDIT, Timeline Followback) remains the cornerstone of primary care monitoring.

The Primary Care Monitoring Schedule: A structured rhythm for ALD review.

Baseline

LFTs, FBC (Platelets), INR, FIB-4, US, HBV/HCV serology, AUDIT-C.

3 Months

Repeat LFTs/Platelets/INR.

Re-calculate FIB-4.

Check PEth/CDT for adherence.

Review pharmacotherapy side effects.

6 Months

If cirrhosis confirmed → Initiate **HCC Surveillance** (US ± AFP).

Consider FibroScan repeat.

12 Months

Comprehensive review.

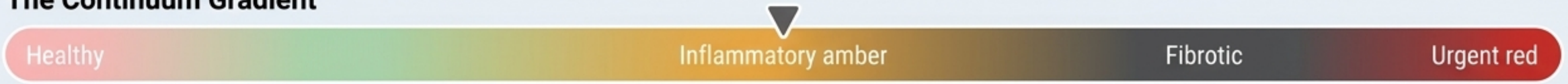
If LFTs normalized, step down to annual monitoring + 6-monthly HCC check.

If deteriorating, refer to hepatology.

Ongoing

Annual AUDIT-C, bone density (DEXA) every 2 years if on steroids.

The Continuum Gradient

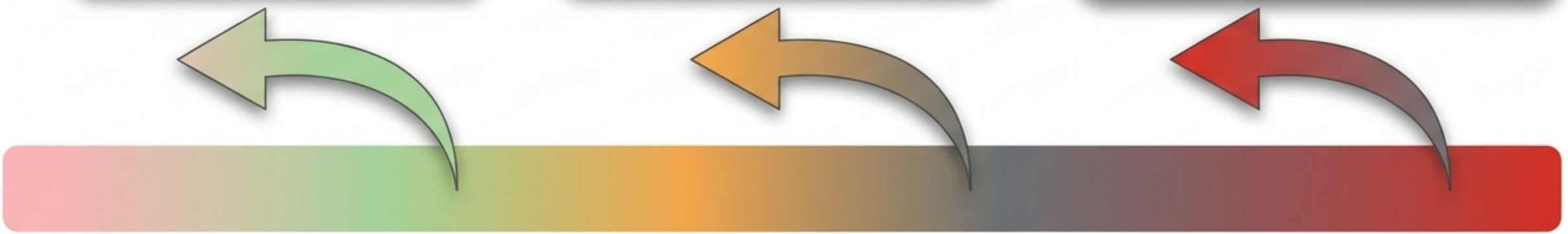


Complete abstinence alters the disease trajectory at every single stage.

At Steatosis:
Abstinence completely reverses histological damage.

At Advanced Fibrosis:
Abstinence halts progression and prevents portal hypertension.

At Decompensated Cirrhosis:
Abstinence reduces portal pressure, improves synthetic function, and provides a distinct survival benefit.



Healthy pink

Steatosis (Green)

Inflammatory Amber

Fibrotic Grey

Decompensated Cirrhosis (Red)

There is no safe level of ongoing alcohol intake for patients with established liver disease. Empathic, sustained intervention in primary care saves lives.