2022 Annual Update
on Liver Disease
October 8, 2022

UT Southwestern
Medical Center

# Alcohol-associated Liver Disease

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# Disclosures

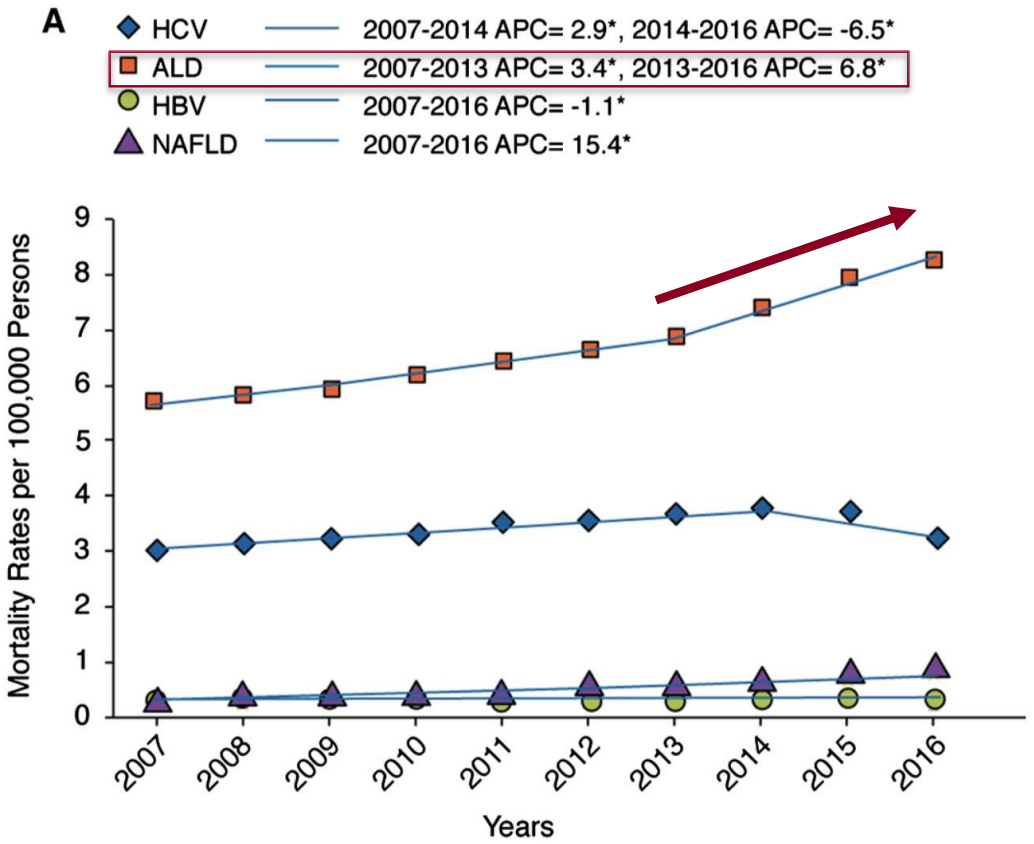
- I have no disclosures

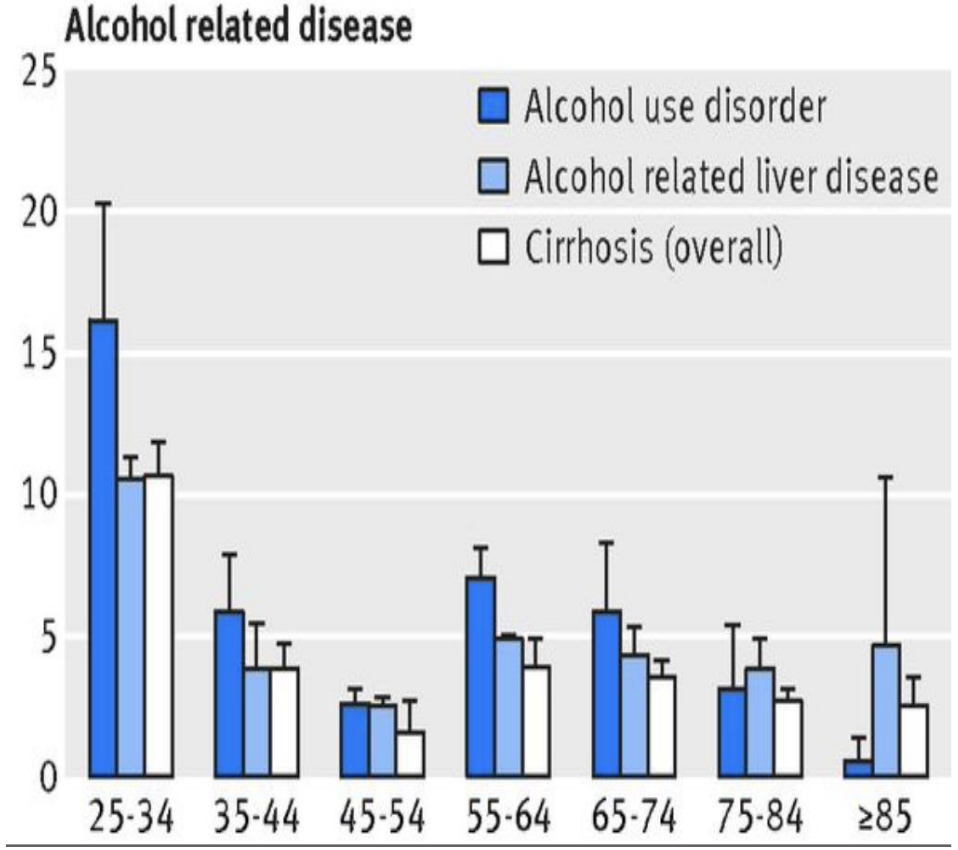
# Alcohol-associated Liver Disease (ALD)

- Epidemiology
- Pathophysiology
- Classification
- Management
- Future Directions

# ALD - Epidemiology

- No. 1 cause of mortality from cirrhosis in the USA



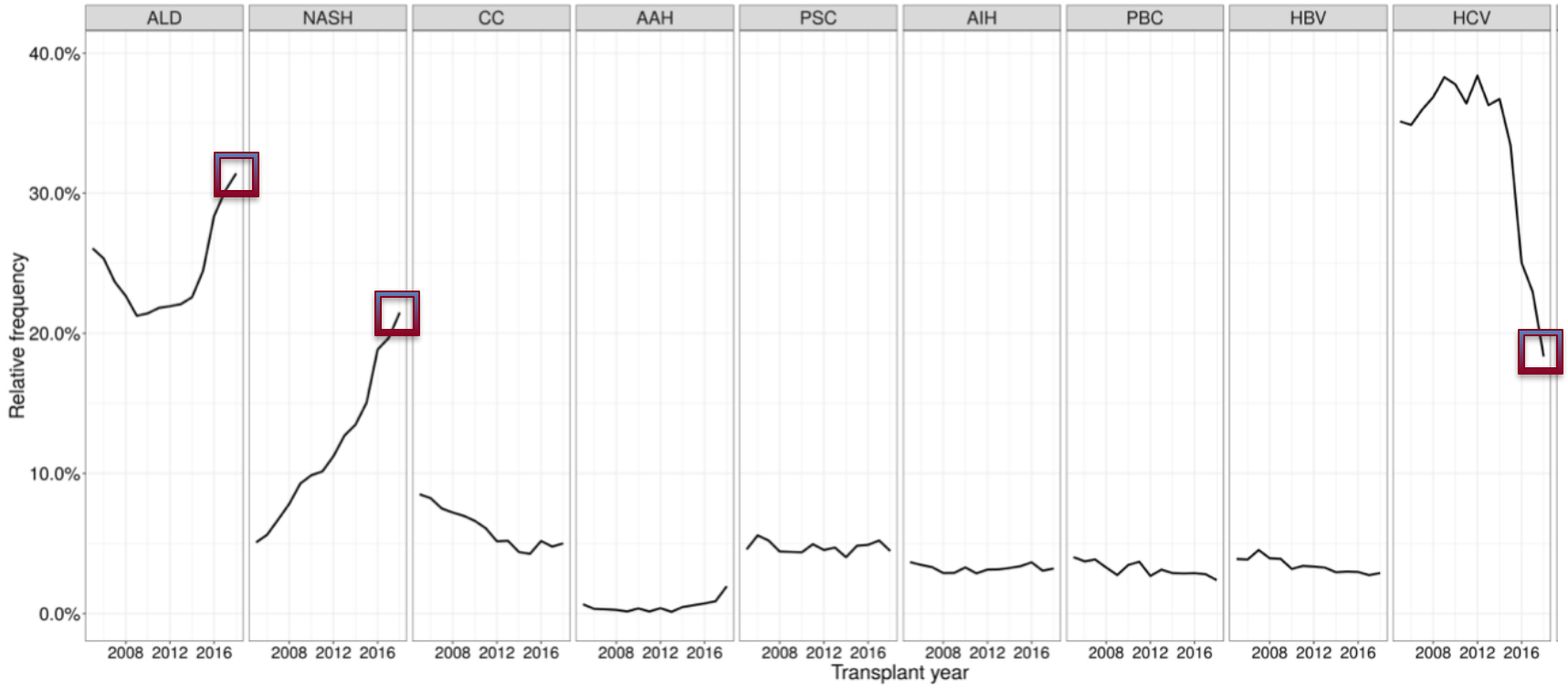






Kim D, et al. Hepatology. 2019;69:1064–1074
Tapper EB, et al. BMJ 2018;362:k2817

# ALD - Epidemiology

- No. 1 indication for liver transplantation in the USA

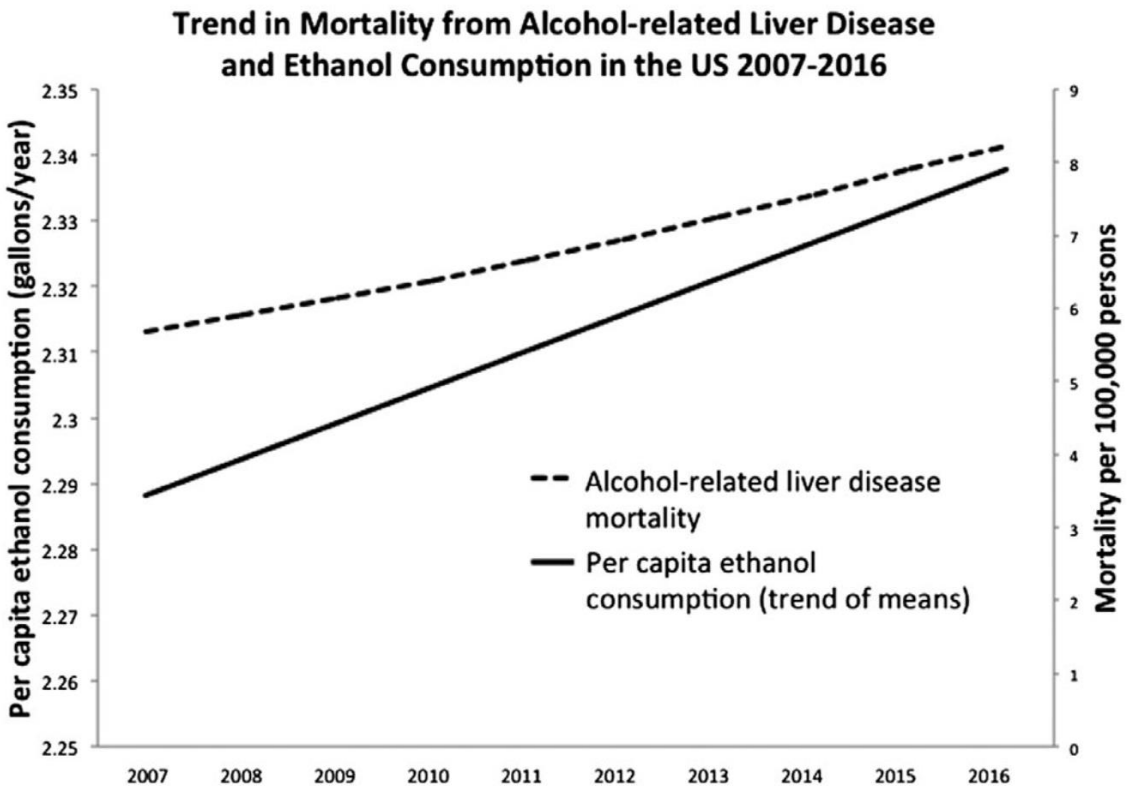





Faust N, et al. DDW April 2020. Gastroenterology 158(6):S-1265

# ALD - Epidemiology

- Change in drinking habits partly responsible




Cotter TG, Charlton M. Hepatology. 2019 Mar;69(3):931-933

# ALD - Epidemiology

- COVID-19 exacerbation of alcohol-associated liver disease

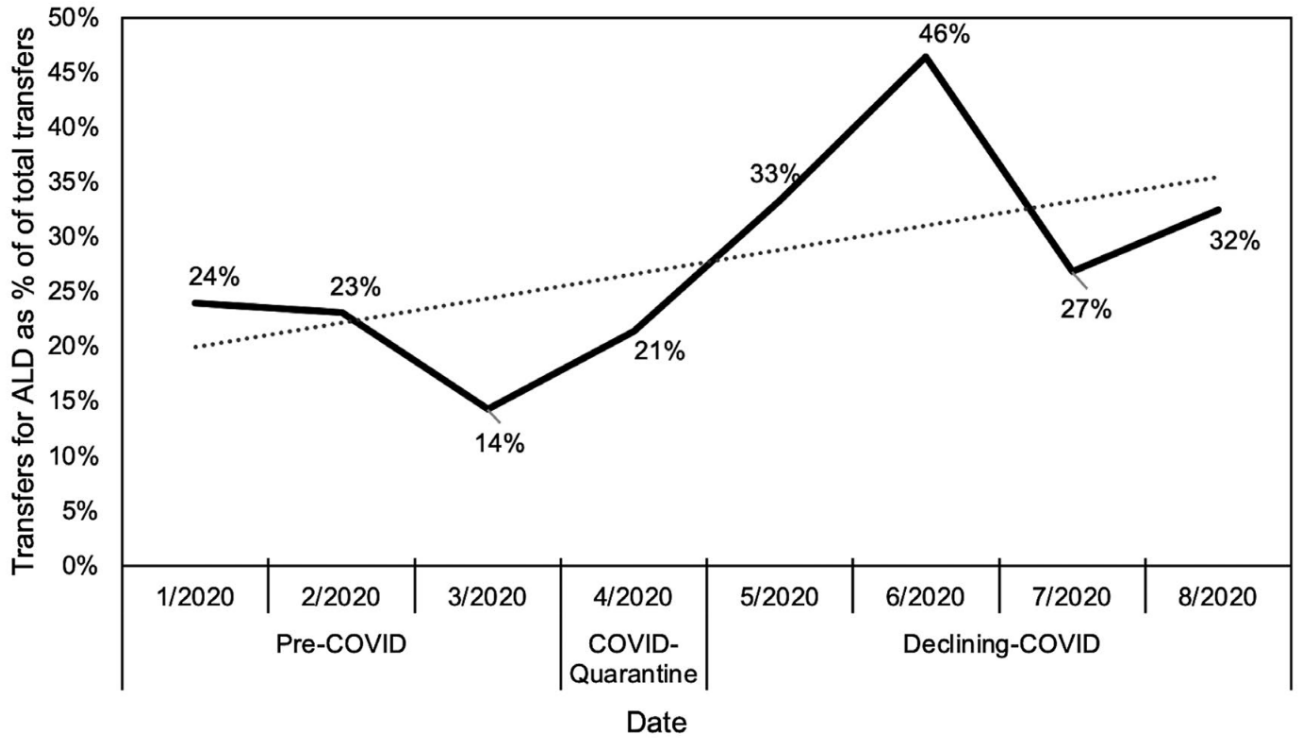
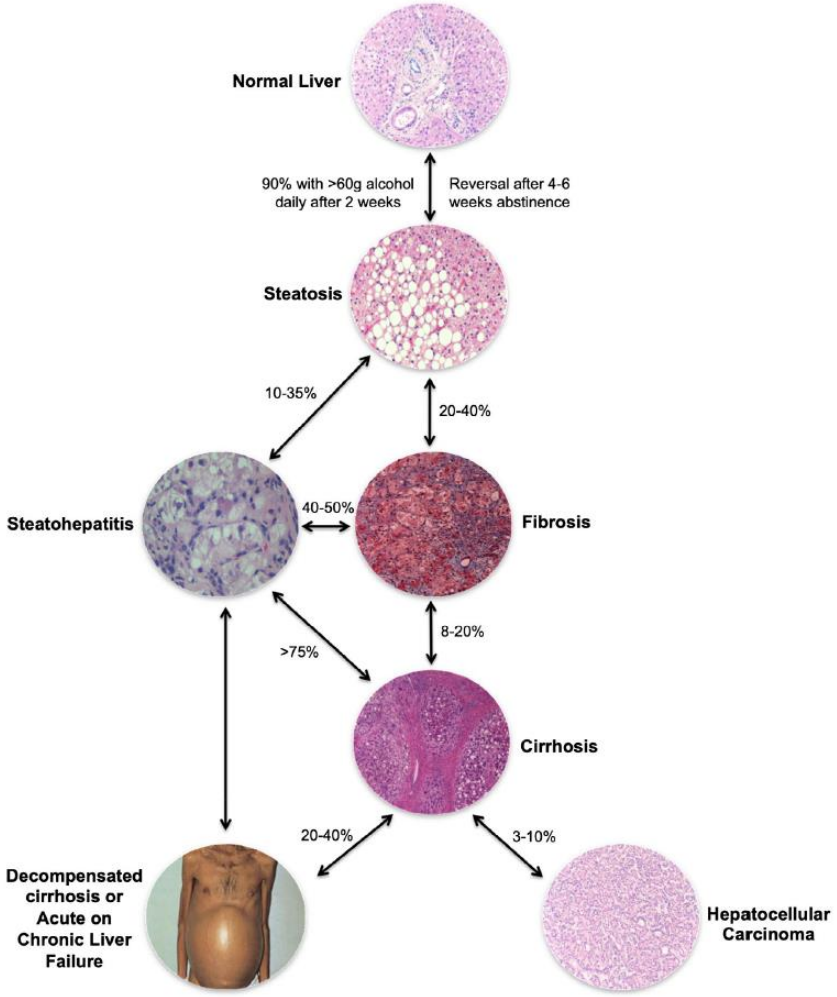



**FIG. 1.** Transfers for ALD as a percentage of total transfers in 2020.



Rutledge S, et al. Hepatol Commun. 2021 Jul;5(7):1151-1155

# ALD – Pathophysiology (Natural History)






Crabb DM, et al. Hepatology. 2020 Jan;71(1):306-333.

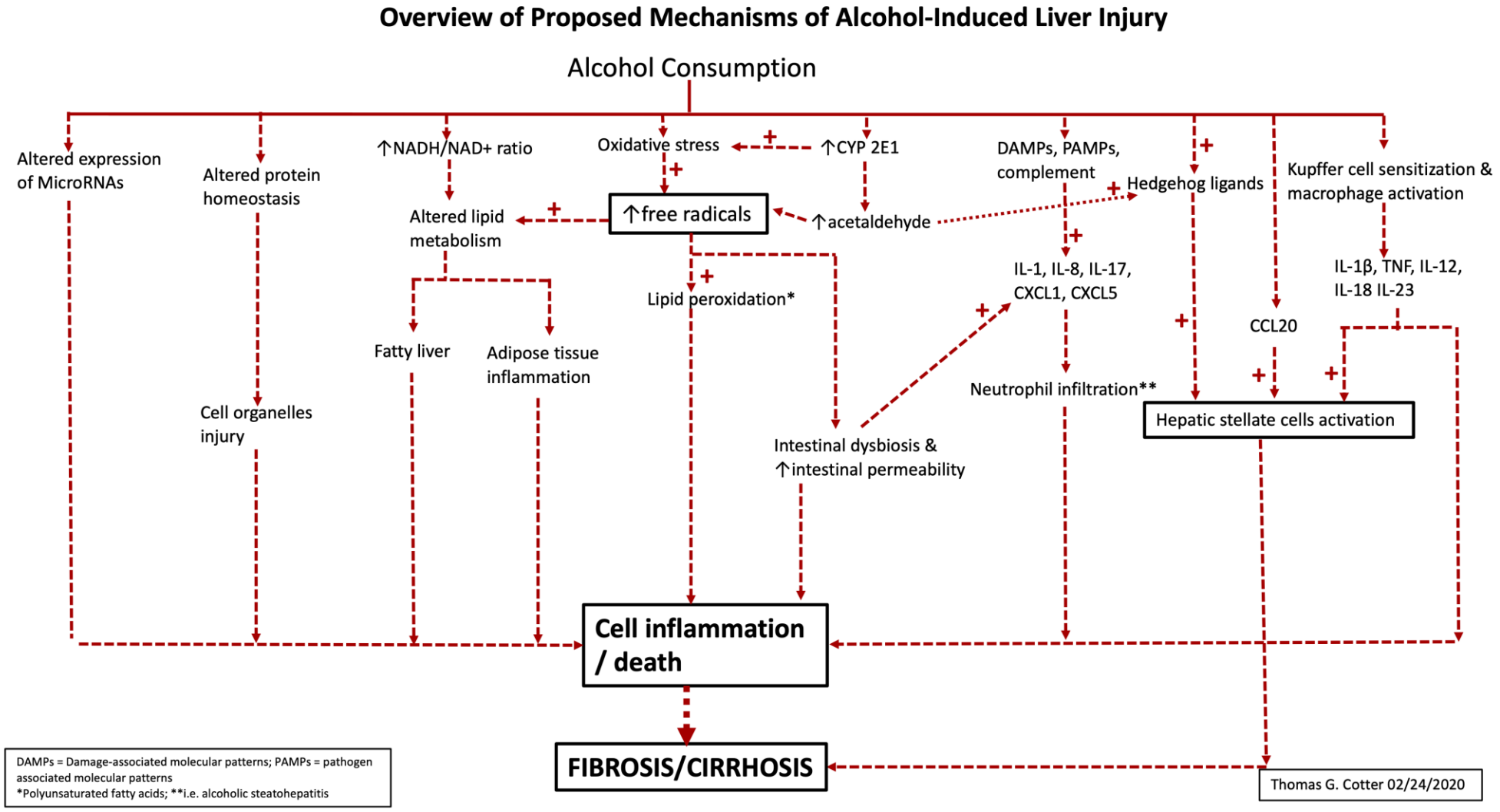
# ALD - Pathophysiology

- Alcohol-associated liver inflammation and fibrosis is infrequent, even in heavy drinkers
- Similar pathogenesis with non-alcoholic fatty liver disease (similar histology). Obesity increases the risk of alcohol-associated liver damage.
- Genetic risk factors, including polymorphisms in lipid droplet proteins are important, as well as likely other yet undetermined genes and environmental factors.



Bellentani S, et al. The Dionysos Study Group. Gut 1997;41:845-850.

# ALD - Pathophysiology



# ALD - Pathophysiology

- Dose-dependent relationship between alcohol and liver disease
- >30g of alcohol daily. Risky drinking (longer duration; binge drinking; drinking outside of meals). But we can't predict who is at risk

| *Alcohol intake (g/day)* | *No (n=6442)* | *NCLD (n=57)* | *Cirrhosis (n=35)* | *Multivariate analysis (odds ratio) (95% CI)* | |
|---|---|---|---|---|---|
| | | | | *For NCLD[a]* | *For cirrhosis[a]* |
| Teetotallers | 2501 (99.9) | 0 (0) | 1 (0.04) | — | — |
| 0.1–30 | 2666 (99.3) | 13 (0.5) | 4 (0.15) | —[b] | —[b] |
| 31–60 | 745 (97.2) | 14 (1.8) | 8 (1.0) | 7.5 (3.5 to 15.9) | 10.9 (3.6 to 33.5) |
| 61–90 | 276 (93.0) | 14 (4.7) | 7 (2.3) | 20.2 (9.4 to 43.3) | 25.0 (7.9 to 79.3) |
| 91–120 | 132 (91.6) | 5 (3.5) | 7 (4.9) | 15.1 (5.3 to 42.8) | 52.9 (16.6 to 169) |
| >120 | 122 (86.5) | 11 (7.8) | 8 (5.7) | 35.8 (15.7 to 81.6) | 62.3 (20.1 to 193) |

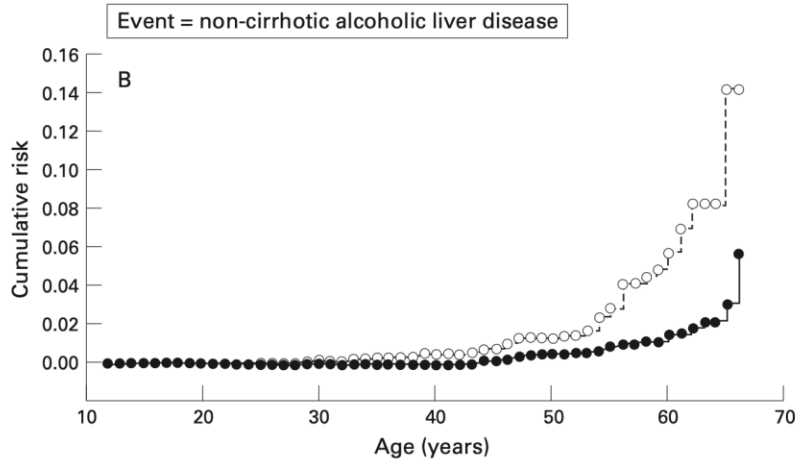


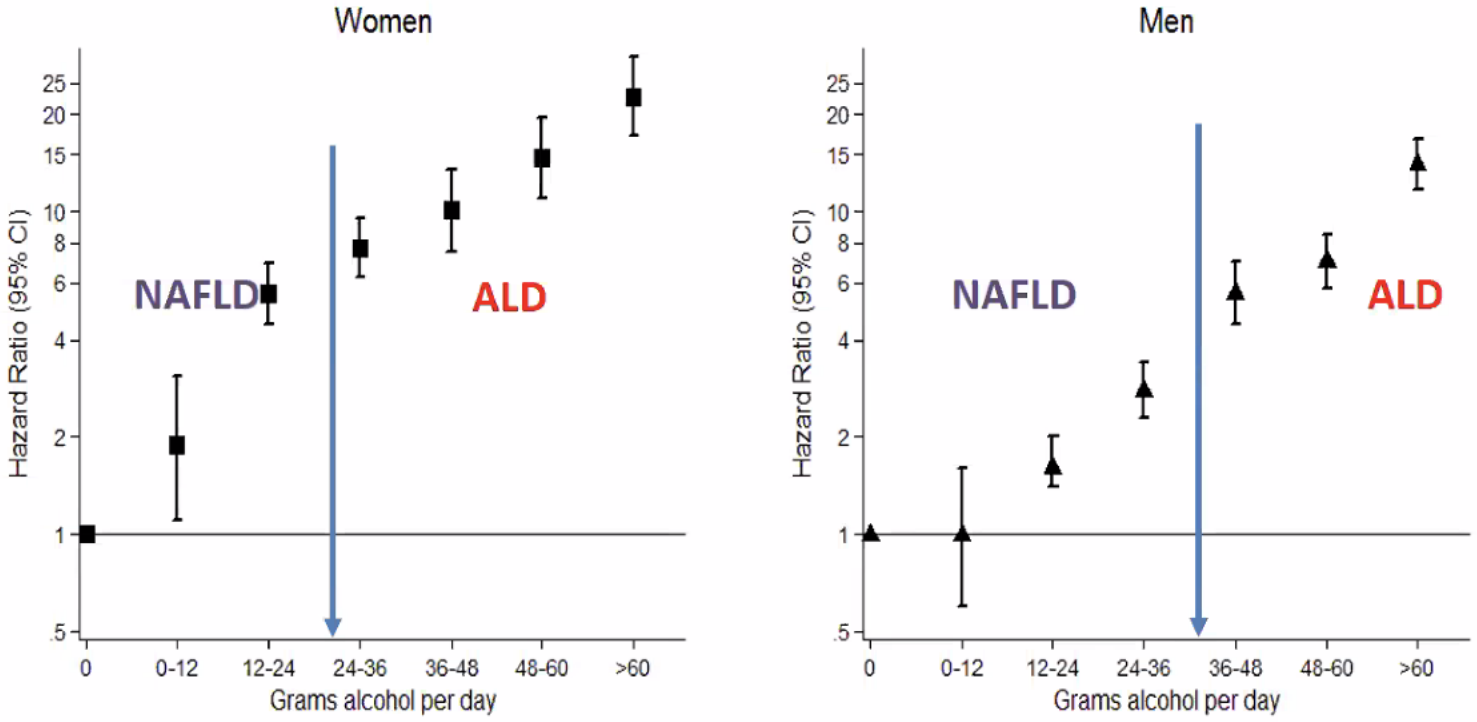

*Figure 1: Cumulative hazard risk curves for the presence of non-cirrhotic alcohol induced liver disease (NCLD) (B) and cirrhosis or hepatocellular carcinoma (A) as a function of age and drinking pattern in subjects with an alcohol intake greater than 30 g/day. Patients drinking only at mealtimes (●) were compared with those drinking outside mealtimes as well (○). The difference in risk of developing either NCLD or cirrhosis or HCC between people drinking alcohol only at mealtimes and those drinking outside mealtimes as well, calculated using log rank and Breslow tests, was significant ($p<0.001$).*



Bellentani et al. Gut 1997;41:845-840 (The Dionysus Study); Askgaard G et al. J Hepatol. 2015 May;62(5):1061-7; Lelbach WK. Ann N Y Acad Sci 1975 Apr 25;252:85-105

# ALD - Pathophysiology

- Increased risk of cirrhosis related to consumption of alcoholic beverages






Rehm J, et al. Drug Alcohol Rev. 2010 Jul;29(4):437-45

# ALD

US National Recommendations on ‘safe’ alcohol consumption

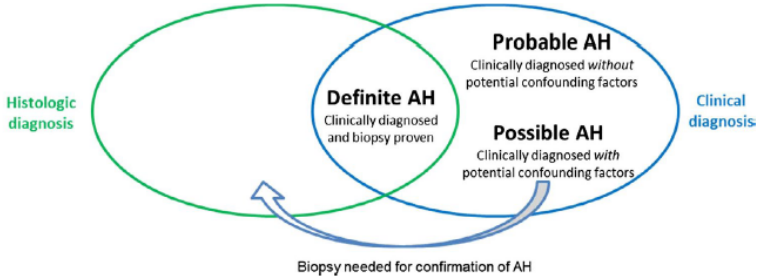
- US HHS & USDA: upper limit = 1 standard drink (14g) daily for women; 2 standard drinks (28g) daily for men
- NIAAA: binge drinking = 4 drinks for women, 5 drinks for men (in ~2 hours) (BAC 0.08 g/dL)



Rehm J, et al. Drug Alcohol Rev. 2010 Jul;29(4):437-45

# ALD - Classification

- Alcohol-associated steatosis: identified on US, CT or MRI of the liver. Absence of jaundice or stigmata of advanced liver disease. Reversible.
- Alcohol-associated cirrhosis: Observed on imaging or biopsy +/- portal hypertension. **Compensated vs. Decompensated** (jaundice, ascites, HE, HRS, variceal bleed).
- Alcohol-associated hepatitis




**Clinical diagnosis of AH**
- Onset of jaundice within prior 8 weeks
- Ongoing consumption of >40 (female) or 60 (male) g alcohol/day for ≥6 months, with <60 days of abstinence before the onset of jaundice
- AST >50, AST/ALT >1.5, and both values <400 IU/L
- Serum total bilirubin >3.0 mg/dL

**Potential confounding factors**
- Possible ischemic hepatitis (e.g., severe upper gastrointestinal bleed, hypotension, or cocaine use within 7 days) or metabolic liver disease (Wilson disease, alpha 1 antitrypsin deficiency)
- Possible drug-induced liver disease (suspect drug within 30 days of onset of jaundice)
- Uncertain alcohol use assessment (e.g., patient denies excessive alcohol use)
- Presence of atypical laboratory tests (e.g., AST <50 or >400 IU/L, AST/ALT <1.5), ANA >1:160 or SMA >1:80.



Crabb DM, et al. Hepatology. 2020 Jan;71(1):306-333.

# ALD – Management (Alcohol Use Disorder)

- Alcohol use disorder (AUD): chronic, relapsing brain disorder.

- DSM-5: 2 or more indicates AUD:

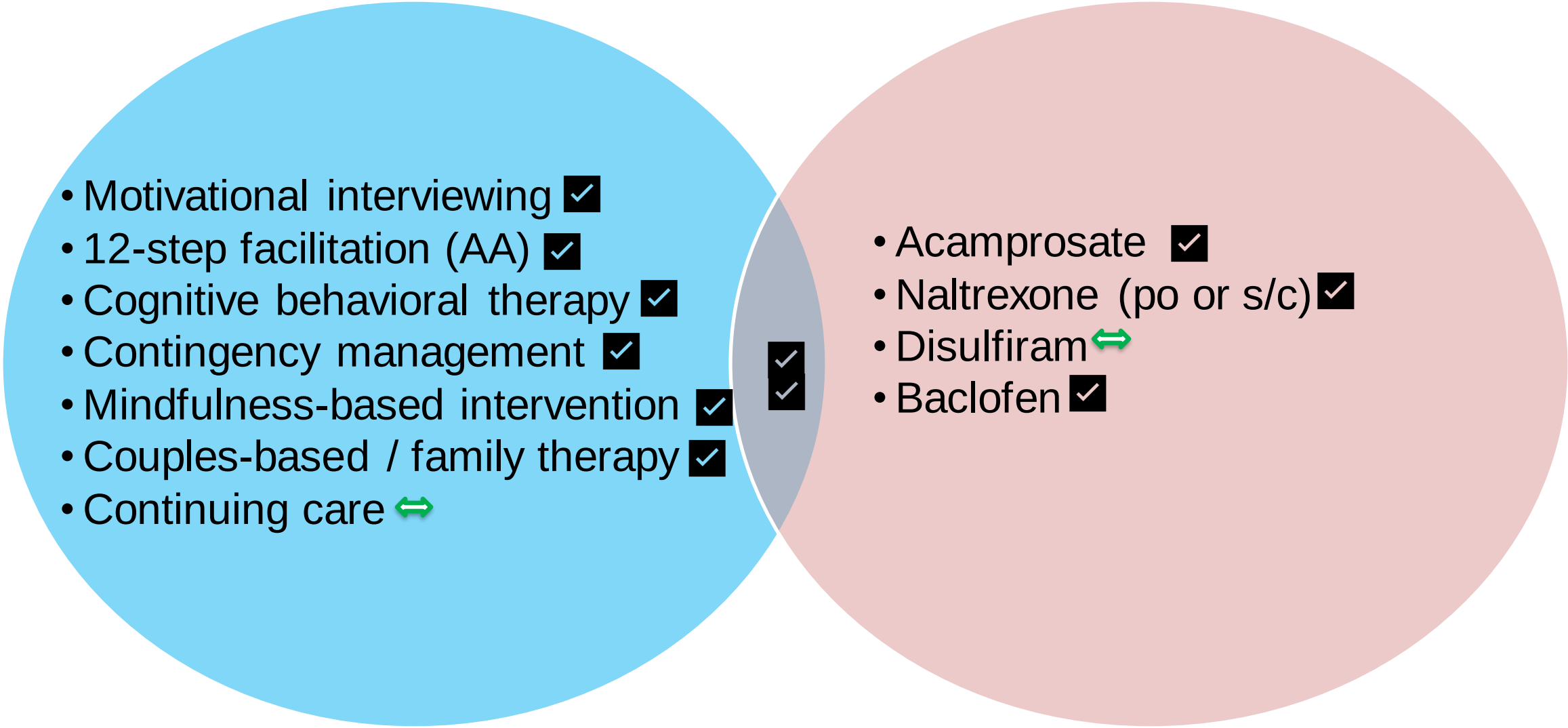
1. Had times when you ended up drinking more, or longer, than you intended?
2. More than once wanted to cut down or stop drinking, or tried to, but couldn't?
3. Spent a lot of time drinking? Or being sick or getting over other aftereffects?
4. Wanted a drink so badly you couldn't think of anything else?
5. Found that drinking—or being sick from drinking—often interfered with taking care of your home or family?
6. Continued to drink even though it was causing trouble with your family or friends?
7. Given up or cut back on activities that were important to you, or gave you pleasure, in order to drink?
8. More than once gotten into situations while or after drinking that increased your chances of getting hurt?
9. Continued to drink even though it was making you feel depressed or anxious?
10. Had to drink much more than you once did to get the effect you want?
11. Found that when the effects of alcohol were wearing off, you had withdrawal symptoms?

**>15 million US adults (5.6%).**



American Psychiatric Association: The Diagnostic and Statistical Manual of Mental Disorders (DSM–5)

# ALD – Management (Alcohol Use Disorder)






Knox J, et al. Lancet Psychiatry. 2019 Dec;6(12):1054-1067; Anton RF, et al. JAMA. 2006;295(17):2003-2017 (COMBINE)
Lieber CS, et al. Alcohol Clin Exp Res. 2003;27(11):1765-1772.

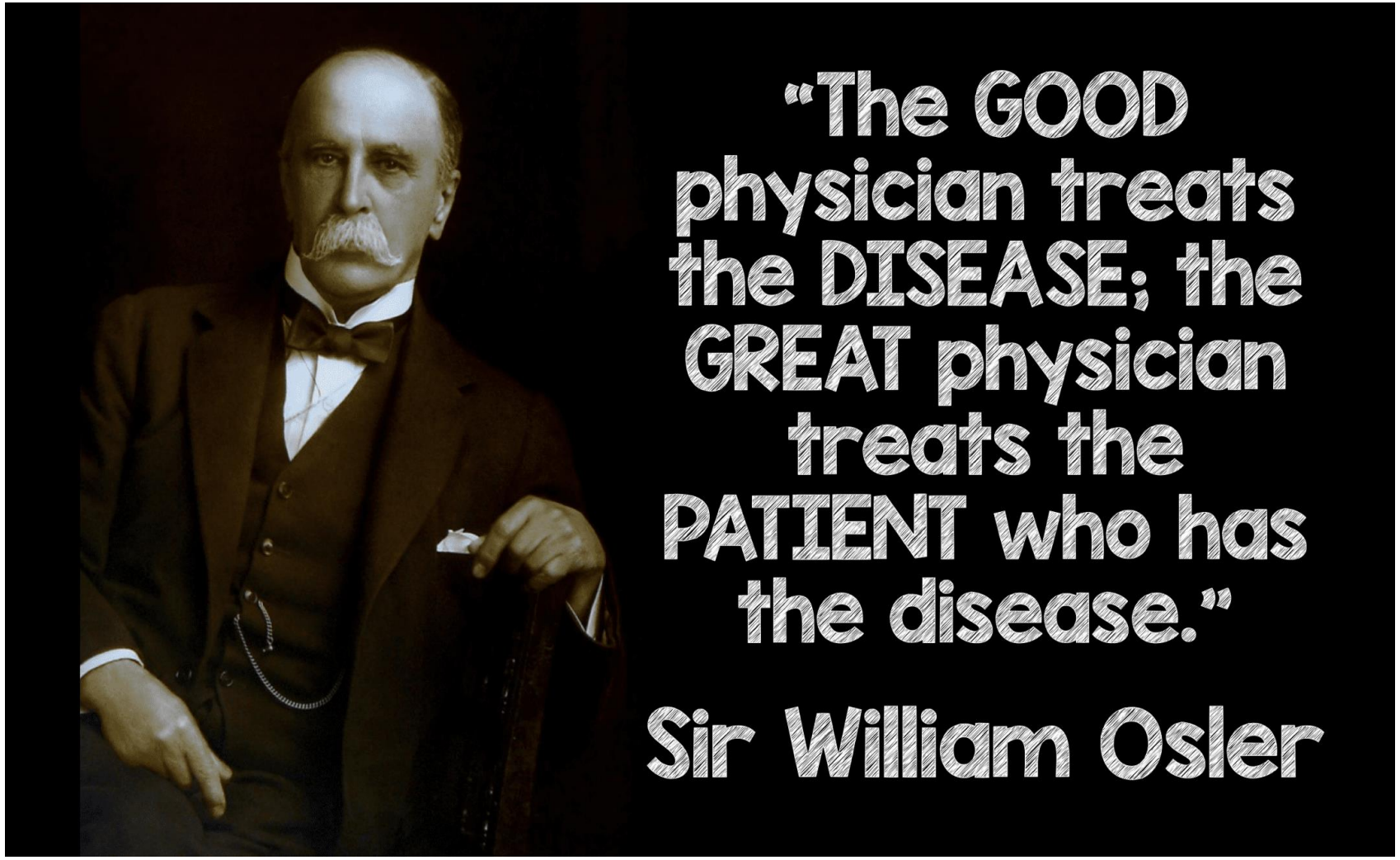
# ALD – Management (Alcohol Use Disorder)

| Drug | Mechanism of action | Dosage | FDA-Approved | Evidence | Comments |
|---|---|---|---|---|---|
| Acamprosate | ?NMDA receptor agonist | 666mg three times daily | Yes | NNT 12 | Renally excreted |
| Naltrexone | Mu opiate receptor antagonist | Oral: 50mg daily<br>IM: 480 mg q 4 wks | Yes | Oral: NNT 20<br>IM: Reduction in heavy drinking days | Opioid-free prior to initiation. Hepatically metabolized. |
| Baclofen | $GABA_B$ receptor agonist | 5mg TID, & uptitrate q 3-5 days (Max 45mg daily) | No | 68-71% vs 24-29% placebo rates for abstinence | Can lower seizure thresholds. Studied in advanced liver disease |
| Disulfiram | Inhibition of acetaldehyde dehydrogenase | 250-500mg daily | Yes | Not efficacious (?excellent adherence) | 12 hours of abstinence. Black box (hepatotoxicity) |



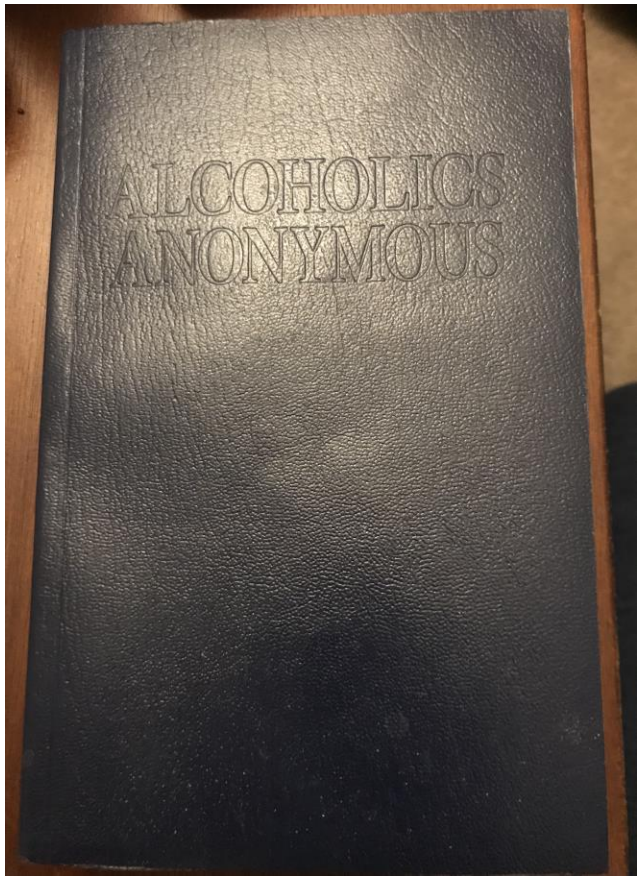
Jonas DE. Agency for Healthcare Research and Quality 2014; Addolorato G, et al. Lancet. 2007 8;370(9603):1915-22; Muller CA, et al. Eur Neuropsychopharmacol. 2015 Aug;25(8):1167-77; Rogal S, et al. Hepatology. 2020 Jun;71(6):2080-92

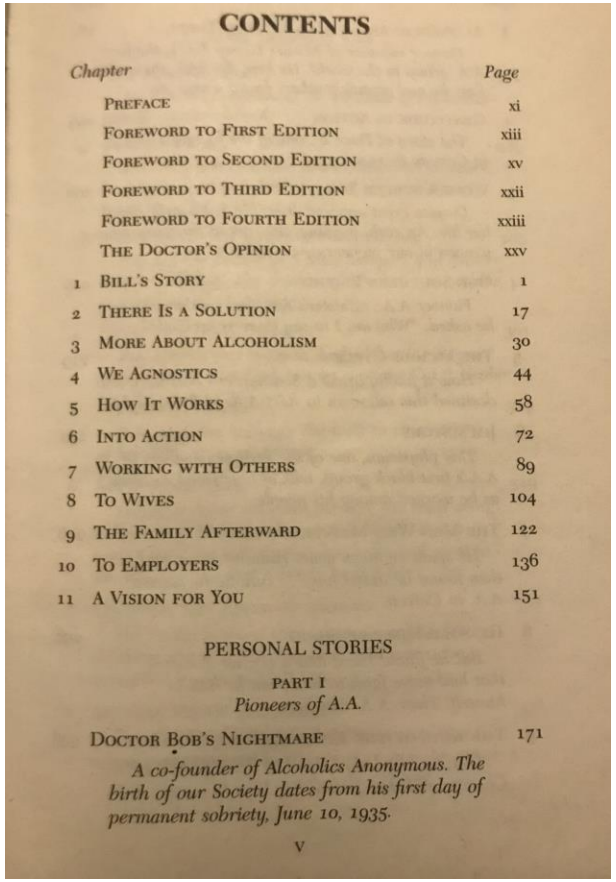
# ALD – Management (Alcohol Use Disorder)



# ALD – Management (Alcohol Use Disorder)

- Alcoholics' Anonymous



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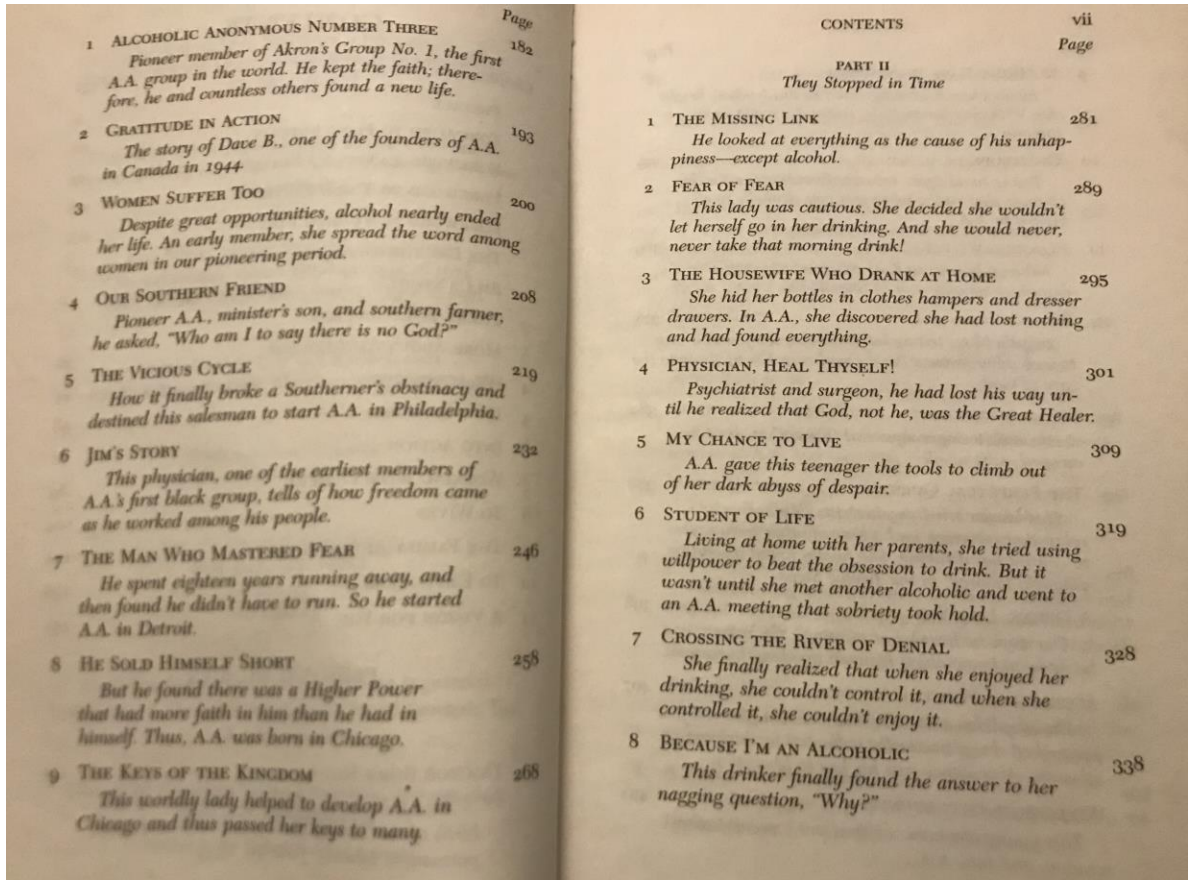









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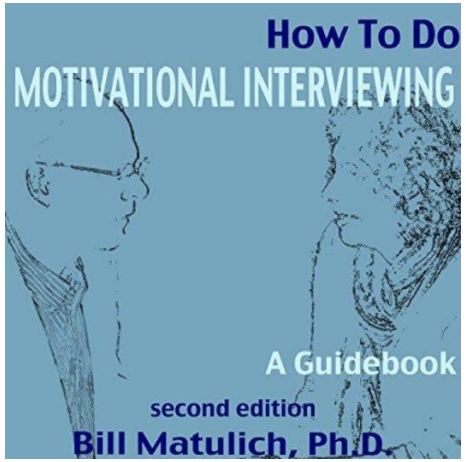
# ALD – Management (Alcohol Use Disorder)

1. We admitted we were powerless over alcohol—that our lives had become unmanageable.
2. Came to believe that a power greater than ourselves could restore us to sanity.
3. Made a decision to turn our will and our lives over to the care of God as we understood Him.
4. Made a searching and fearless moral inventory of ourselves.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. Were entirely ready to have God remove all these defects of character.
7. Humbly asked Him to remove our shortcomings.
8. Made a list of all persons we had harmed, and became willing to make amends to them all.
9. Made direct amends to such people wherever possible, except when to do so would injure them or others.
10. Continued to take personal inventory, and when we were wrong, promptly admitted it.
11. Sought through prayer and meditation to improve our conscious contact with God as we understood Him, praying only for knowledge of His will for us and the power to carry that out.
12. Having had a spiritual awakening as the result of these steps, we tried to carry this message to alcoholics, and to practice these principles in all our affairs.

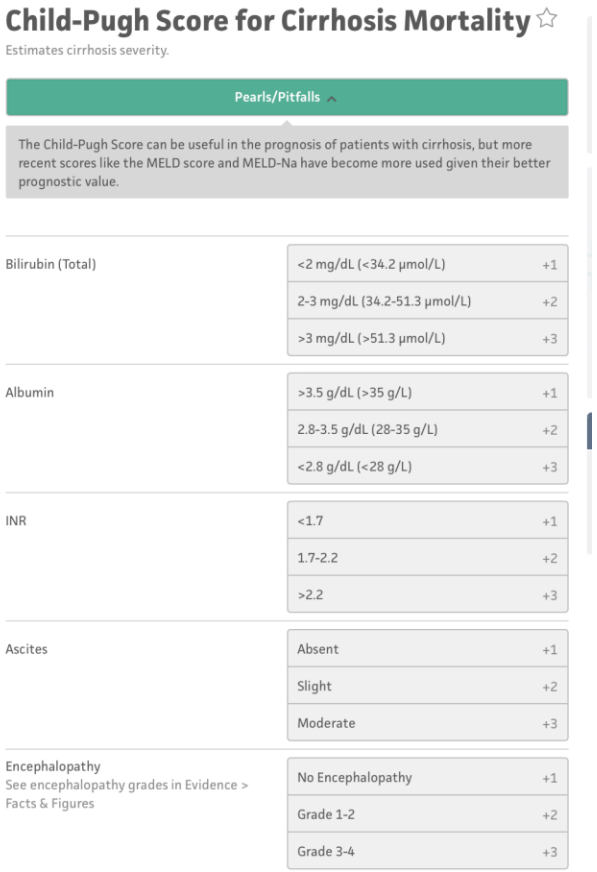
# ALD – Management (Alcohol Use Disorder)




- Motivational interviewing
- <u>Aims</u>:
  - Reduce resistance
  - Explore discrepancy between behavior and goals (i.e. resolve ambivalence)
  - Open questions, affirmation, reflective listening, and summary reflections (OARS)
  - Elicit “change talk”

# ALD – Management (alcohol-ass'd cirrhosis)

- Risk stratification: Child-Pugh Score



**Child-Pugh Score for Cirrhosis Mortality** ☆

Estimates cirrhosis severity.

Pearls/Pitfalls

The Child-Pugh Score can be useful in the prognosis of patients with cirrhosis, but more recent scores like the MELD score and MELD-Na have become more used given their better prognostic value.

| | | |
|---|---|---|
| Bilirubin (Total) | <2 mg/dL (<34.2 µmol/L) | +1 |
| | 2-3 mg/dL (34.2-51.3 µmol/L) | +2 |
| | >3 mg/dL (>51.3 µmol/L) | +3 |
| Albumin | >3.5 g/dL (>35 g/L) | +1 |
| | 2.8-3.5 g/dL (28-35 g/L) | +2 |
| | <2.8 g/dL (<28 g/L) | +3 |
| INR | <1.7 | +1 |
| | 1.7-2.2 | +2 |
| | >2.2 | +3 |
| Ascites | Absent | +1 |
| | Slight | +2 |
| | Moderate | +3 |
| Encephalopathy<br>See encephalopathy grades in Evidence > Facts & Figures | No Encephalopathy | +1 |
| | Grade 1-2 | +2 |
| | Grade 3-4 | +3 |

| | Class A | Class B | Class C |
|---|---|---|---|
| **Total points** | 5-6 | 7-9 | 10-15 |
| **1-year survival** | 100% | 80% | 45% |



Child CG, et al. The liver and portal hypertension. Philadelphia: Saunders 1964: 50-64.

# ALD – Management (alcohol-ass'd cirrhosis)

- Risk stratification: MELD score

## MELD Score (Model For End-Stage Liver Disease) (12 and older) ☆

Stratifies severity of end-stage liver disease, for transplant planning.

**INSTRUCTIONS**

Use in patients ≥12 years old. Note: As of January 2016, calculation of the MELD has changed. It now includes serum sodium level. See OPTN's announcement.

When to Use ⌄ | Pearls/Pitfalls ⌄ | Why Use ⌄

| | | |
|---|---|---|
| Dialysis at least twice in the past week<br>Or CVVHD for ≥24 hours in the past week | No | Yes |
| Creatinine<br>Cr >4.0 mg/dL is automatically assigned a value of 4.0 | Norm: 0.7 - 1.3 | mg/dL ⇋ |
| Bilirubin | Norm: 0.3 - 1.9 | mg/dL ⇋ |
| INR | Norm: 1 - 2 | |
| Sodium | Norm: 136 - 145 | mEq/L ⇋ |

### FORMULA

Per OPTN policy, January 2016 (pages 4–5):

Candidates who are at least 12 years old receive an initial MELD(i) score equal to:

**MELD(i) = 0.957 × ln(Cr) + 0.378 × ln(bilirubin) + 1.120 × ln(INR) + 0.643**

Then, round to the tenth decimal place and multiply by 10.

If MELD(i) > 11, perform additional MELD calculation as follows:

**MELD = MELD(i) + 1.32 × (137 – Na) – [ 0.033 × MELD(i) × (137 – Na) ]**

Additional rules:

- All values in US units (Cr and bilirubin in mg/dL, Na in mEq/L, and INR unitless).
- If bilirubin, Cr, or INR is <1.0, use 1.0.
- If any of the following is true, use Cr 4.0:
  - Cr >4.0.
  - ≥2 dialysis treatments within the prior 7 days.
  - 24 hours of continuous veno-venous hemodialysis (CVVHD) within the prior 7 days.
- If Na <125 mmol/L, use 125. If Na >137 mmol/L, use 137.
- Maximum MELD = 40.

### FACTS & FIGURES

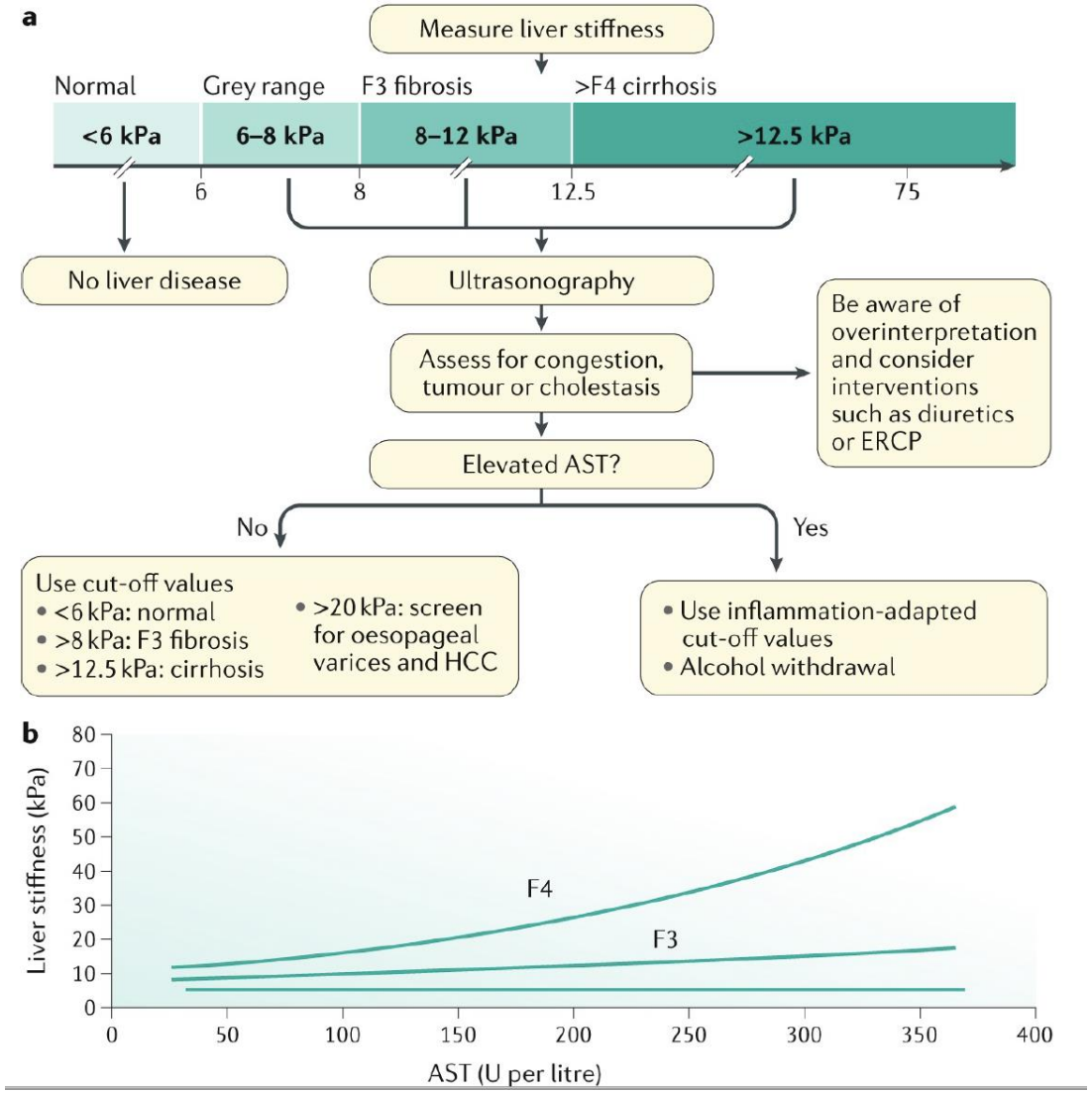
**Interpretation:**

| MELD Score | Mortality |
|---|---|
| ≤9 | 1.9% |
| 10–19 | 6.0% |
| 20–29 | 19.6% |
| 30–39 | 52.6% |
| ≥40 | 71.3% |



Kamath PS, et al. Hepatology. 2001 Feb;33(2):464-70

# ALD – Management (alcohol-ass'd cirrhosis)






Seitz HK, et al. Nat Rev Dis Primers 2018 Aug 16;4(1):16

# ALD – Management (alcohol-ass'd cirrhosis)

- Treatment
  - – Abstinence (AUD mgt)
  - – MELD score 15 or above, or significant decompensation - liver transplantation evaluation
  - – Standard cirrhosis management (Volume, Infection, Bleeding, Encephalopathy, Screening
  - – Nutrition, physical therapy

# ALD – Management (alcohol-ass'd hepatitis)

- Risk Stratification

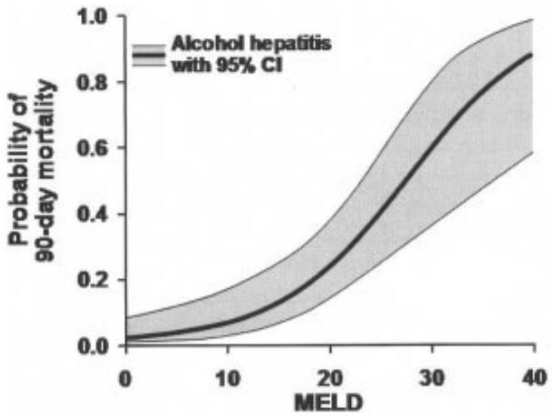



Fig. 1. Prediction of 90-day mortality in patients with AH based on MELD. The curve demonstrates probability of 90-day mortality in AH for given MELD (**black line**) with confidence intervals (**gray shading**). The probability of 90-day mortality in AH was calibrated using the data from logistic regression ($P = e^{(-4.3 + 0.16 \times \text{MELD})} / [1 + e^{(-4.3 + 0.16 \times \text{MELD})}]$). MELD, model for end-stage liver disease.

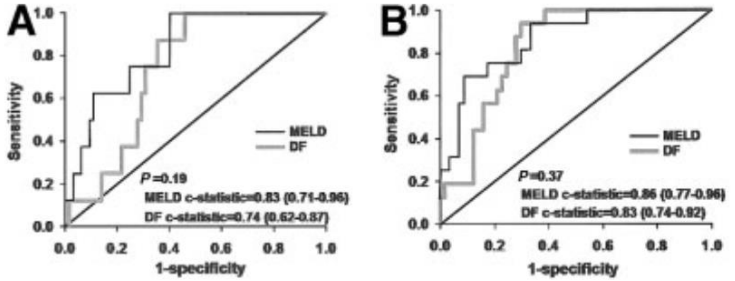



Fig. 2. Comparison of MELD and DF in predicting mortality in AH. Receiver operating characteristic curves and c-statistics were generated to compare MELD (**black curve**) and DF (**gray curve**) in predicting mortality rate in AH. Respective c-statistics and confidence intervals are indicated. MELD and DF were comparable regarding prediction of (A) 30-day mortality and (B) 90-day mortality ($P > .05$). MELD, model for end-stage liver disease; DF, Maddrey discriminant function; c-statistic, concordance statistic.

**TABLE 8. Advantages and Disadvantages of Lab-Based Prognostic Scores in Alcoholic Hepatitis**

| | Advantages | Disadvantages |
|---|---|---|
| MDF | Decades of experience in AH<br>Key inclusion criterion in most AH trials | False positives can lead to excess corticosteroid treatment |
| MELD | Extensive experience in hepatology | Uncertain threshold for initiating corticosteroids |
| ABIC | Three-tiered stratification | Uncertain threshold for initiating corticosteroids and not verified outside of Spain |
| GAHS | Improves specificity of patients with MDF ≥32 needing corticosteroids | Not verified outside of United Kingdom |
| Lille | Allows early cessation of corticosteroids | Uncertain decision making with partial response (Lille 0.46-0.56) |



Dunn. Hepatology 2005
Crabb DM, et al. Hepatology. 2020 Jan;71(1):306-333

# ALD – Management (alcohol-ass'd hepatitis)

- Lille score: to assess steroid response

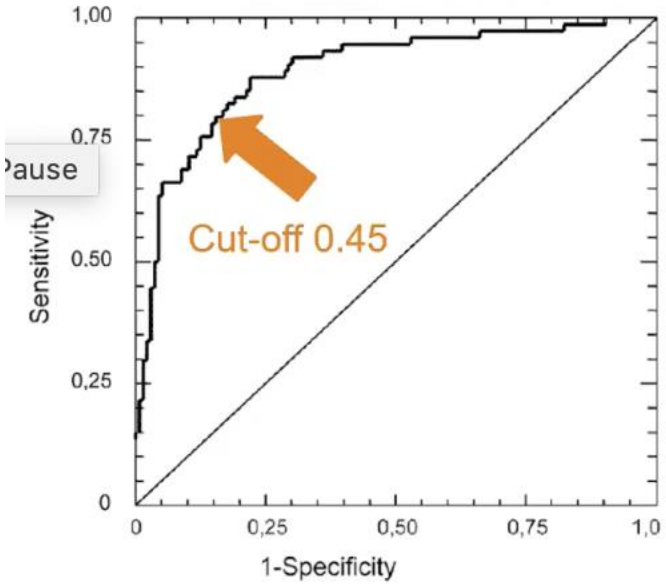
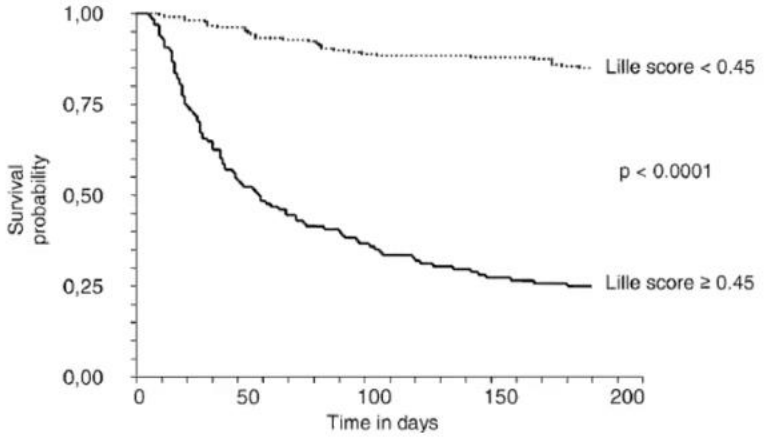



Fig. 1. Receiver operating characteristic curve for survival at 6 months in the exploratory cohort using the Lille model.




www.lillemodel.com

R = 3.19 - (0.101 x Age) + (0.0165 x Difference in bilirubin D0-D7 in µmol/l) + (0.147 x Albumin D0) - (0.206 x Renal Insufficiency) - (0.0065 x Bilirubin D0 in µmol/l) - (0.0096 x PT in seconds)



Louvet. Hepatology 2007.

# ALD – Management (alcohol-ass'd hepatitis)

- Treatment:
  - Abstinence
  - Prednisolone 40 mg once daily for 4 weeks (rule out infection, GI bleed hemostasis)
  - Nutrition

# ALD – Management (alcohol-ass'd hepatitis)

- Steroids - STOPAH trial
  - Power calculation = 28-day mortality rate at the margins from 30% (placebo) to 21% (treatment). Actual study: 28-day mortality =17% in placebo

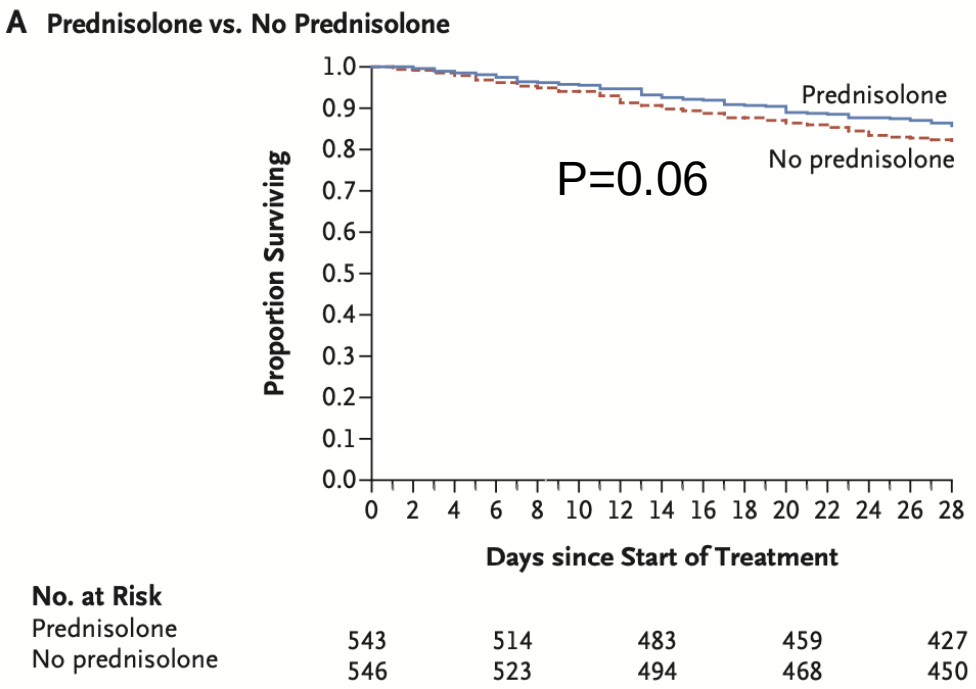



**Table 3. Analysis of Factors Associated with Mortality at 28 Days.***

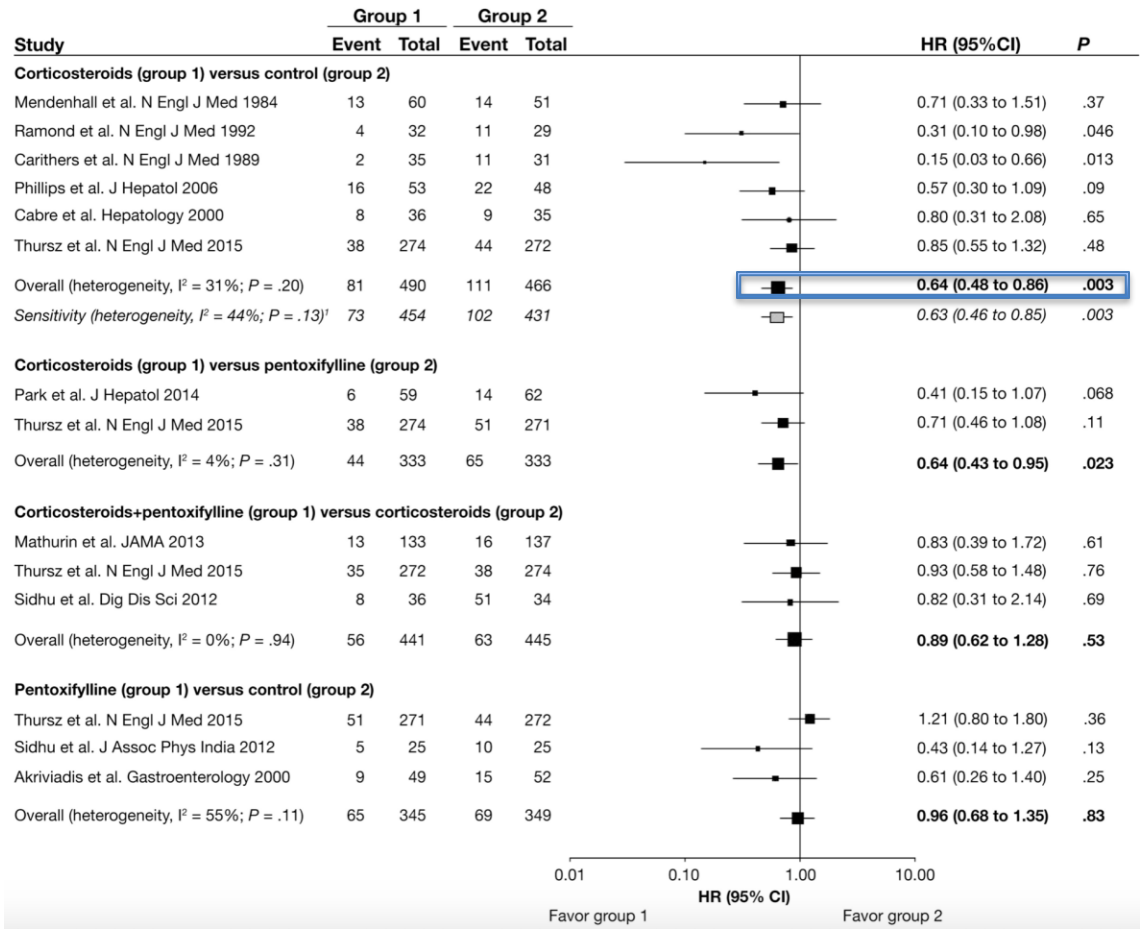
| Variable | Univariate Analysis† | | Multivariate Analysis‡ | |
|---|---|---|---|---|
| | Odds Ratio (95% CI) | P Value | Odds Ratio (95% CI) | P Value |
| Prednisolone vs. no prednisolone | | | 0.61 (0.41–0.91) | 0.02 |
| Pentoxifylline vs. no pentoxifylline | | | 1.10 (0.74–1.64) | 0.62 |
| Discriminant function | 1.02 (1.02–1.03) | <0.001 | | |
| GAHS | 2.17 (1.86–2.53) | <0.001 | | |
| MELD | 1.15 (1.12–1.18) | <0.001 | | |
| Lille§ | 1.03 (1.02–1.03) | <0.001 | | |
| Prothrombin ratio or INR | 1.38 (1.10–1.73) | 0.005 | 1.38 (1.13–1.69) | 0.002 |
| Age | 1.05 (1.03–1.07) | <0.001 | 1.05 (1.03–1.07) | <0.001 |
| White cells | 1.06 (1.03–1.08) | <0.001 | 1.03 (1.00–1.06) | 0.04 |
| Urea | 1.14 (1.10–1.18) | <0.001 | 1.06 (1.01–1.12) | 0.01 |
| Creatinine | 3.07 (2.32–4.08) | <0.001 | 1.56 (1.05–2.33) | 0.03 |
| Pyrexia | 0.66 (0.37–1.16) | 0.15 | | |
| Hypotension | 1.20 (0.79–1.83) | 0.39 | | |
| Tachycardia | 1.09 (0.71–1.65) | 0.70 | | |
| Alcohol intake | 1.00 (1.00 –1.00) | 0.37 | | |
| Albumin | 0.99 (0.96–1.02) | 0.41 | | |
| Alkaline phosphate | 1.00 (1.00–1.00) | 0.07 | | |
| Bilirubin | 1.07 (1.05–1.09) | <0.001 | 1.03 (1.01–1.06) | 0.003 |
| Hepatic encephalopathy | 3.70 (2.59–5.29) | <0.001 | 3.07 (2.05–4.60) | <0.001 |



Thursz MR, et al. Engl J Med. 2015 Apr 23;372(17):1619-28

# ALD – Management (alcohol-ass'd hepatitis)

- 11 RCTs:
  - Steroids > control: 36% mortality decrease over 28 days
  - No difference in 3- and 6-month survival
  - Not studied in severe renal failure
  - Pentoxifylline inefficient, alone or with steroids



| Study | Group 1 Event | Group 1 Total | Group 2 Event | Group 2 Total | HR (95%CI) | P |
|---|---|---|---|---|---|---|
| **Corticosteroids (group 1) versus control (group 2)** | | | | | | |
| Mendenhall et al. N Engl J Med 1984 | 13 | 60 | 14 | 51 | 0.71 (0.33 to 1.51) | .37 |
| Ramond et al. N Engl J Med 1992 | 4 | 32 | 11 | 29 | 0.31 (0.10 to 0.98) | .046 |
| Carithers et al. N Engl J Med 1989 | 2 | 35 | 11 | 31 | 0.15 (0.03 to 0.66) | .013 |
| Phillips et al. J Hepatol 2006 | 16 | 53 | 22 | 48 | 0.57 (0.30 to 1.09) | .09 |
| Cabre et al. Hepatology 2000 | 8 | 36 | 9 | 35 | 0.80 (0.31 to 2.08) | .65 |
| Thursz et al. N Engl J Med 2015 | 38 | 274 | 44 | 272 | 0.85 (0.55 to 1.32) | .48 |
| Overall (heterogeneity, $I^2$ = 31%; $P$ = .20) | 81 | 490 | 111 | 466 | **0.64 (0.48 to 0.86)** | **.003** |
| *Sensitivity (heterogeneity, $I^2$ = 44%; $P$ = .13)[1]* | *73* | *454* | *102* | *431* | *0.63 (0.46 to 0.85)* | *.003* |
| **Corticosteroids (group 1) versus pentoxifylline (group 2)** | | | | | | |
| Park et al. J Hepatol 2014 | 6 | 59 | 14 | 62 | 0.41 (0.15 to 1.07) | .068 |
| Thursz et al. N Engl J Med 2015 | 38 | 274 | 51 | 271 | 0.71 (0.46 to 1.08) | .11 |
| Overall (heterogeneity, $I^2$ = 4%; $P$ = .31) | 44 | 333 | 65 | 333 | **0.64 (0.43 to 0.95)** | **.023** |
| **Corticosteroids+pentoxifylline (group 1) versus corticosteroids (group 2)** | | | | | | |
| Mathurin et al. JAMA 2013 | 13 | 133 | 16 | 137 | 0.83 (0.39 to 1.72) | .61 |
| Thursz et al. N Engl J Med 2015 | 35 | 272 | 38 | 274 | 0.93 (0.58 to 1.48) | .76 |
| Sidhu et al. Dig Dis Sci 2012 | 8 | 36 | 51 | 34 | 0.82 (0.31 to 2.14) | .69 |
| Overall (heterogeneity, $I^2$ = 0%; $P$ = .94) | 56 | 441 | 63 | 445 | **0.89 (0.62 to 1.28)** | **.53** |
| **Pentoxifylline (group 1) versus control (group 2)** | | | | | | |
| Thursz et al. N Engl J Med 2015 | 51 | 271 | 44 | 272 | 1.21 (0.80 to 1.80) | .36 |
| Sidhu et al. J Assoc Phys India 2012 | 5 | 25 | 10 | 25 | 0.43 (0.14 to 1.27) | .13 |
| Akriviadis et al. Gastroenterology 2000 | 9 | 49 | 15 | 52 | 0.61 (0.26 to 1.40) | .25 |
| Overall (heterogeneity, $I^2$ = 55%; $P$ = .11) | 65 | 345 | 69 | 349 | **0.96 (0.68 to 1.35)** | **.83** |





Louvet A, et al. Gastroenterology. 2018 Aug;155(2):458-468

# ALD – Management (alcohol-ass'd hepatitis)

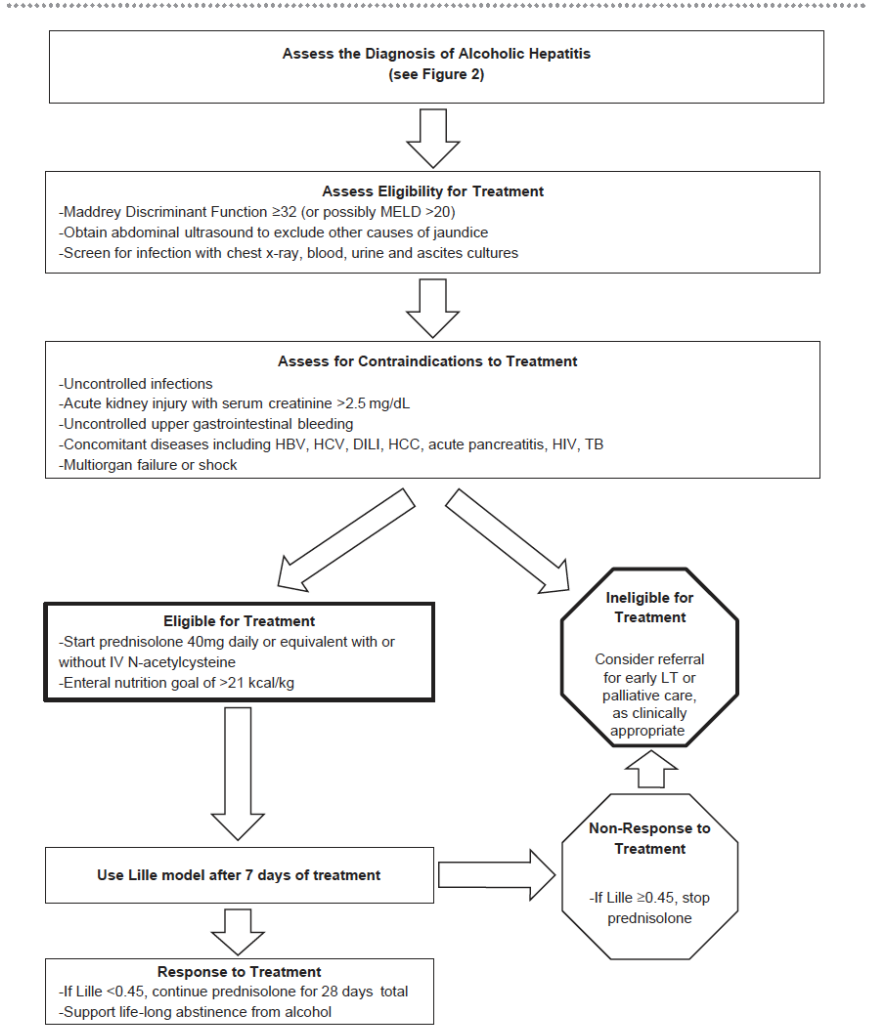



FIG. 3. Assessment of patients with alcoholic hepatitis likely to benefit from treatment with corticosteroids. Abbreviations: DILI, drug-induced liver injury; HBV, hepatitis B virus; HIV, human immunodeficiency virus; and TB, tuberculosis.



Crabb DM, et al. Hepatology. 2020 Jan;71(1):306-333

# ALD – Management (alcohol-ass'd hepatitis)

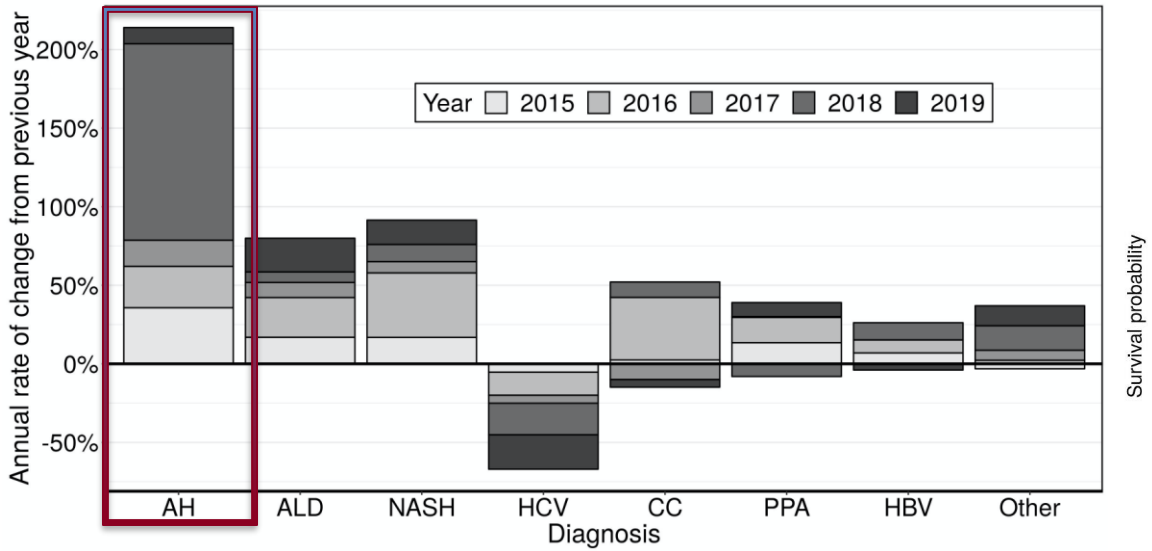
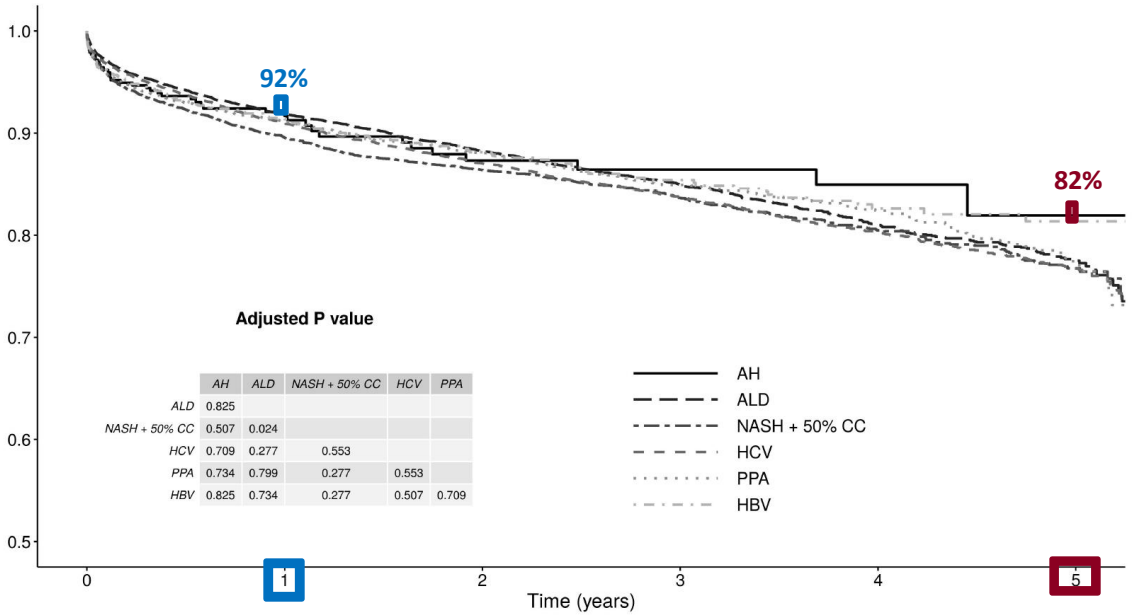



Figure 1: Annual rate change (%) in deceased-donor liver transplantation by primary listing indication from 2014 to 2019. There has been a 5-fold increase in alcoholic hepatitis cases over this time frame, but the rate of change slowed down to only 10% from 2018 to 2019. (*Abbreviations*: AH: Alcoholic Hepatitis; ALD: Alcohol-related Liver Disease; CC: Cyroptogenic Cirrhosis; HBV: Hepatitis B Virus infection; HCV: Hepatitis C Virus infection; NASH: Non-Alcoholic Steatohepatitis; PPA: Primary Biliary Cholangitis / Primary Sclerosing Cholangitis / Autoimmune Hepatitis.)




| | AH | ALD | NASH + 50% CC | HCV | PPA |
|---|---|---|---|---|---|
| ALD | 0.825 | | | | |
| NASH + 50% CC | 0.507 | 0.024 | | | |
| HCV | 0.709 | 0.277 | 0.553 | | |
| PPA | 0.734 | 0.799 | 0.277 | 0.553 | |
| HBV | 0.825 | 0.734 | 0.277 | 0.507 | 0.709 |



Cotter TG, et al. Am J Transplant. 2021 Mar;21(3):1039-1055

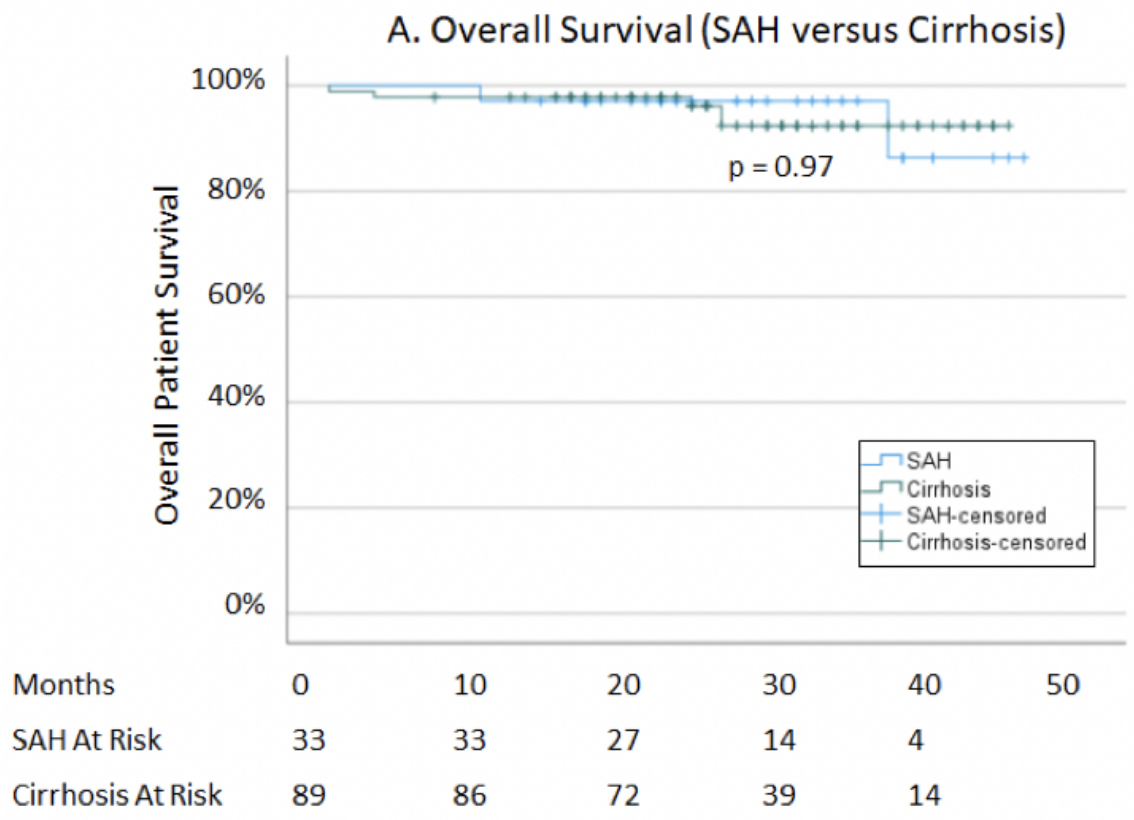
# ALD – Management (alcohol-ass'd hepatitis)

- **UT Southwestern Medical Center early LT criteria for AH:**

1. First episode of AH.
2. Addiction Psychiatry evaluation.
3. Acceptable social support and insight (Social Worker and/or psychological assessment).
4. Written commitment to engage in post-liver transplant alcohol counseling. 5. If history of psychiatric disease, Psychologist or Psychiatrist evaluation.
6. No current abuse of other substances.

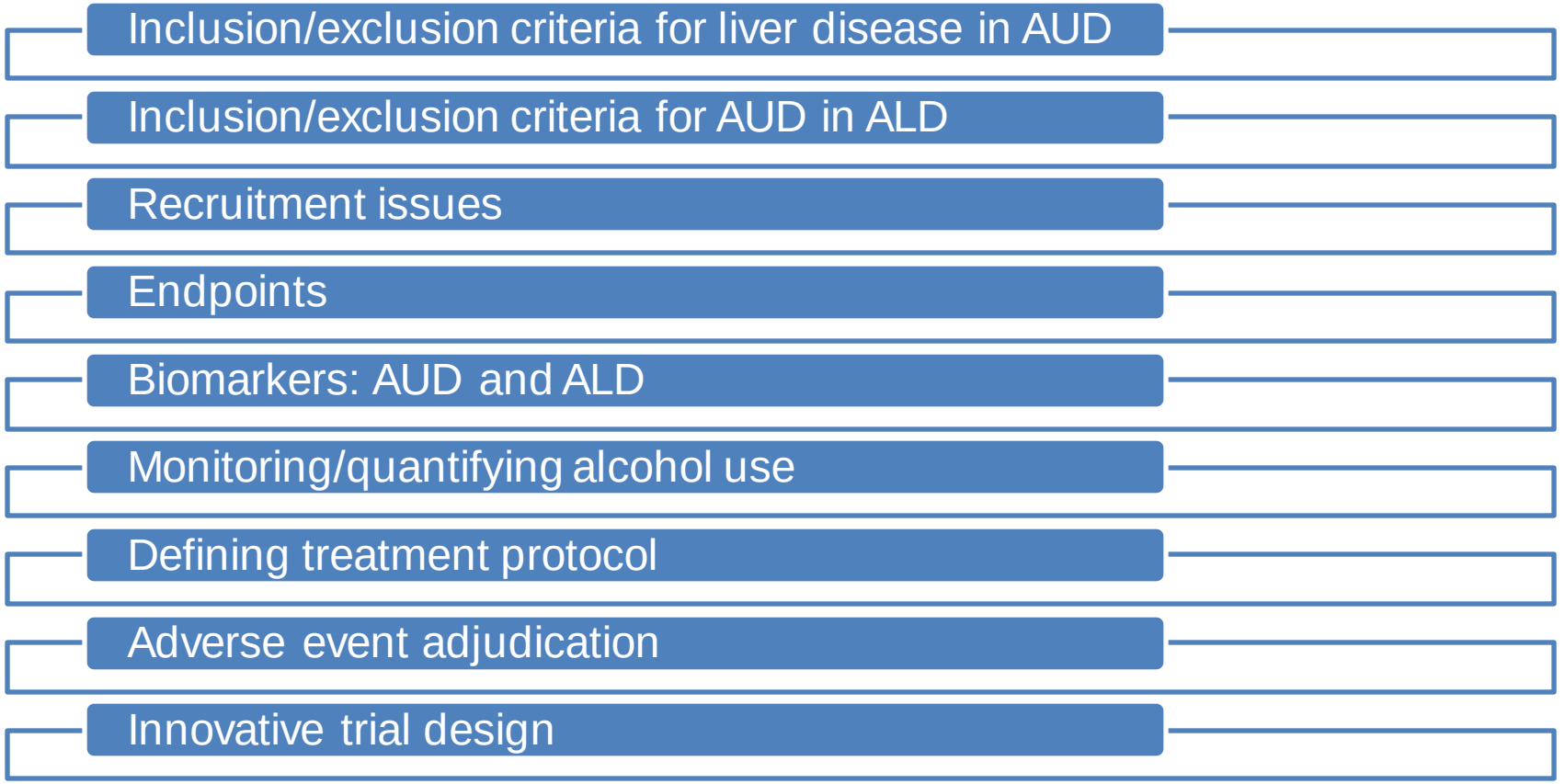
# ALD – Management (alcohol-ass'd hepatitis)

- UT Southwestern Medical Center early LT for AH outcomes:



# ALD – Future Directions

- NIAAA: Improving integrated care for AUD-ALD patients



# THANK YOU!

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