Common inflammatory diseases of the liver: hepatitis B, hepatitis C and nonalcoholic fatty liver disease

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LEARNING OBJECTIVES

- Overview epidemiology chronic viral hepatitis B and C
- Describe diagnostic approach
- Review goals of antiviral therapy
- Discuss current treatment options and criteria used to select therapy

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LEARNING OBJECTIVES

- Define NAFLD and NASH
- Describe magnitude of the problem and natural history
- Review pathogenesis
- Discuss diagnostic approach
- Review current and future management

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HEPATITIS B VIRUS (HBV) INFECTION

- HBV blood-borne DNA-virus that infects the liver
- Chronic infection leads to serious liver-related consequences, including liver cancer
- With proper screening, diagnosis, and successful treatment, **HBV infection can be controlled although not yet cured**

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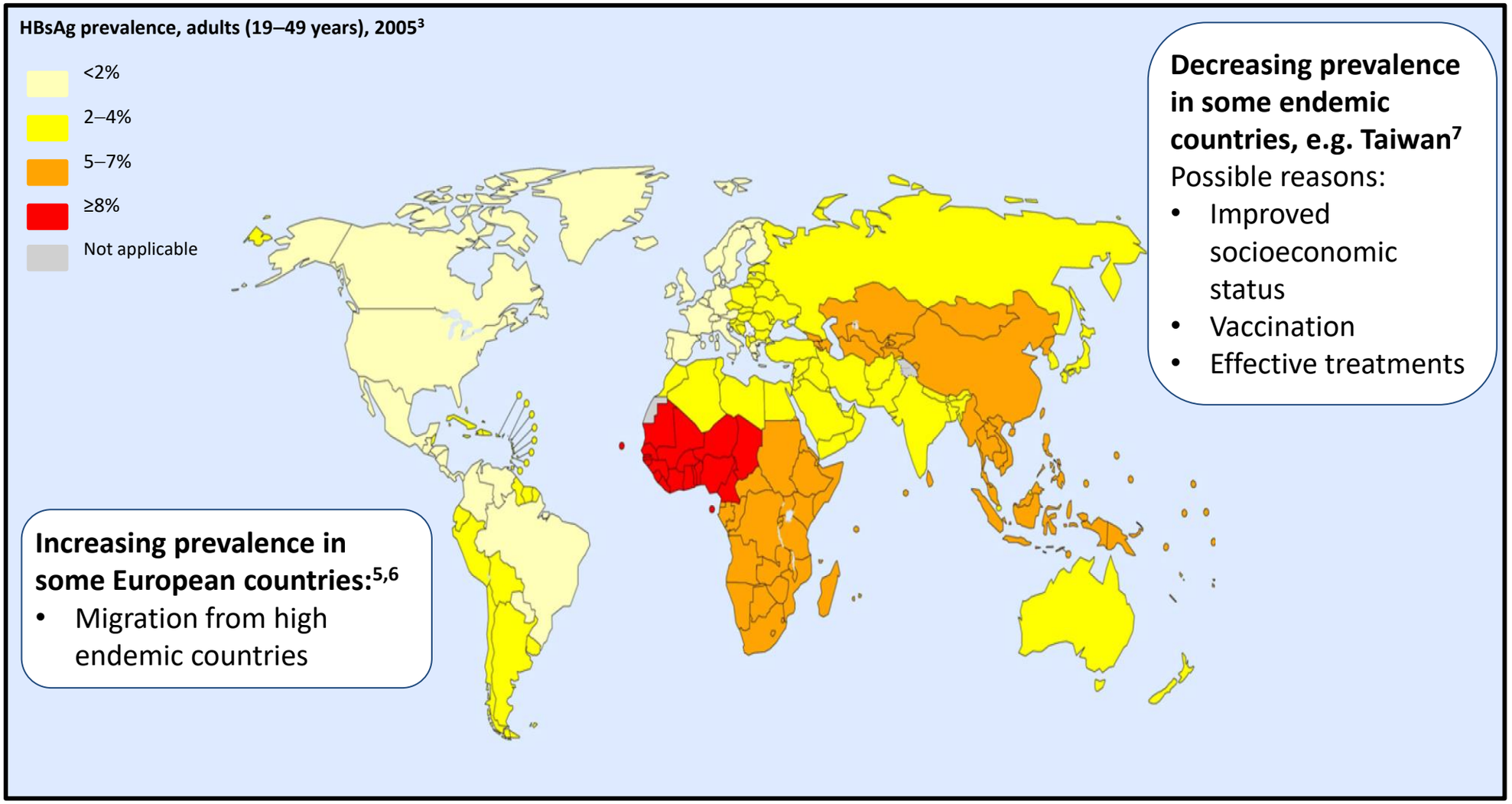
EPIDEMIOLOGY HEPATITIS B



EASL CPG HBV. J Hepatol 2017;67:370–98
Schweitzer A, et al. Lancet 2015;386:1546–55

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EPIDEMIOLOGY HBV

- Worldwide ≈260 million chronic HBsAg
- US ~1 to 2 million persons chronic HBV
  - Asian & Pacific Islanders >50% US patients with HBV
  - Overall ~65% unaware HBV infection
- Worldwide HBV infection is the most common cause for hepatocellular carcinoma

Schweitzer A, et al. Lancet 2015;386:1546–55
Nelson NP. Et al. Clin Liver Dis 2016;20:607-628

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INTERPRETATION SCREENING TESTS HBV INFECTION

Screening Test Results				
HBsAg	Anti-HBc	Anti-HBs	Interpretation	Management
+	+	–	Chronic hepatitis B	Additional testing and management needed
–	+	+	Past HBV infection, resolved	No further management unless immunocompromised or undergoing chemotherapy or immunosuppressive therapy
–	+	–	Past HBV infection, resolved or false-positive	HBV DNA testing if immunocompromised patient
–	–	+	Immune	No further testing
–	–	–	Uninfected and not immune	No further testing

Terrault NA, et al. AASLD 2018 Hepatitis B Guidance. Hepatology 2018;67:1560-1599

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NEW NOMENCLATURE CHRONIC PHASES

	HBeAg positive		HBeAg negative		
	Phase 1	Phase 2	Phase 3	Phase 4	Phase 5
	Chronic HBV infection	Chronic hepatitis B	Chronic HBV infection	Chronic hepatitis B	Resolved HBV infection
HBsAg	High	High/ intermediate	Low	Intermediate	Negative
HBeAg	Positive	Positive	Negative	Negative	Negative
HBV DNA	>10^7 IU/mL	10^4–10^7 IU/mL	<2,000 IU/mL*	>2,000 IU/mL	<10 IU/mL‡
ALT	Normal	Elevated	Normal	Elevated†	Normal
Liver disease	None/minimal	Moderate/ severe	None	Moderate/ severe	None§
Old terminology	Immune tolerant	Immune reactive HBeAg positive	Inactive carrier	HBeAg negative chronic hepatitis	HBsAg negative / anti-HBc positive

EASL CPG HBV. J Hepatol 2017;67:370–98

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GOALS OF THERAPY

- Improve survival and quality of life by preventing disease progression and HCC development
- Prevent mother-to-child transmission
- Prevent hepatitis B reactivation
- Prevent/treat HBV-associated extrahepatic manifestations

EASL CPG HBV. J Hepatol 2017;67:370–98

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TREATMENT ENDPOINTS

Recommendations		
Main endpoint • Induction of long-term suppression of HBV DNA	I	1
Valuable endpoint • Induction of HBeAg loss (± anti-HBe seroconversion) in HBeAg-positive patients with chronic hepatitis B	II-1	1
Additional endpoint • ALT normalization (biochemical response)	II-1	1
Optimal endpoint • HBsAg loss (± anti-HBs seroconversion)	II-1	1

EASL CPG HBV. J Hepatol 2017;67:370–98

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INDICATIONS FOR TREATMENT

Recommendations		
Should be treated		
• Patients with HBeAg-positive or -negative chronic hepatitis B	I	1
• Patients with cirrhosis, any detectable HBV DNA, regardless of ALT level	I	1
• Patients with HBV DNA >20,000 IU/mL and ALT >2x ULN, regardless of severity of histological lesions	II-2	1
May be treated		
• Patients with HBeAg-positive chronic HBV infection >30 years old, regardless of severity of liver histological lesions	III	2
Can be treated		
• Patients with HBeAg-positive or -negative chronic HBV infection and family history of HCC or cirrhosis and extrahepatic manifestations	III	2

EASL CPG HBV. J Hepatol 2017;67:370–98

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THERAPY FOR TREATMENT-NAÏVE PATIENTS

- Long-term administration of potent nucleoside analogues with **high barrier to resistance** is treatment of choice
- FDA-approved: entecavir (ETV), tenofovir disoproxil fumarate (TDF) & tenofovir alafenamide (TAF)
- In specific clinical scenarios ETV or TAF may be more appropriate choices than TDF

EASL CPG HBV. J Hepatol 2017;67:370–98
Terrault NA, et al. AASLD 2018 Hepatitis B Guidance. Hepatology 2018;67:1560-1599

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HBV AND HCV CO-INFECTION

- HCV co-infection accelerates liver disease progression and increases risk of HCC in patients with chronic HBV infection
- All patients with chronic HBV infection should be screened for HCV and other blood-borne viruses (HIV and HDV)

EASL CPG HBV. J Hepatol 2017;67:370–98

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HBV AND HCV CO-INFECTION

Recommendations		
Treatment of HCV with DAAs may cause reactivation of HBV. Patients fulfilling the standard criteria for HBV treatment should receive NA treatment	II	1
HBsAg-positive patients undergoing DAA therapy should be considered for concomitant NA prophylaxis until 12 weeks after completion of DAA treatment, and monitored closely	II-2	2
HBsAg-negative, anti-HBc-positive patients undergoing DAA therapy should be monitored and tested for HBV reactivation in case of ALT elevation	II	1

EASL CPG HBV. J Hepatol 2017;67:370–98

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ACUTE HBV INFECTION

Recommendations		
More than 95% of adults with acute HBV hepatitis do not require specific treatment	II-2	1
Only patients with severe acute hepatitis B, characterized by coagulopathy or protracted course, should be treated with NAs and considered for liver transplantation	II-2	1

EASL CPG HBV. J Hepatol 2017;67:370–98

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HBV AND IMMUNOSUPPRESSION

Recommendations		
All candidates for chemotherapy and immunosuppressive therapy **should be tested** for HBV markers prior to immunosuppression	I	1
All HBsAg-positive patients should receive ETV, TDF, or TAF as treatment or prophylaxis	II-2	1
HBsAg-negative, anti-HBc-positive subjects should receive anti-HBV prophylaxis if they are at high risk of HBV reactivation	II-2	1

EASL CPG HBV. J Hepatol 2017;67:370–98

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FUTURE TREATMENT HBV

- Several novel direct-acting antivirals and immunotherapeutic agents are in preclinical and early clinical development
- Combinations of antiviral and immune modulatory therapy, targeting multiple steps in the HBV lifecycle, will likely be needed to achieve an HBV ‘cure’

EASL CPG HBV. J Hepatol 2017;67:370–98

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HEPATITIS C VIRUS (HCV) INFECTION

- HCV blood-borne RNA-virus that infects liver
- Chronic infection can lead to serious liver-related consequences (cirrhosis, HCC)
- With proper screening, diagnosis, and successful treatment, **HCV infection can be cured**

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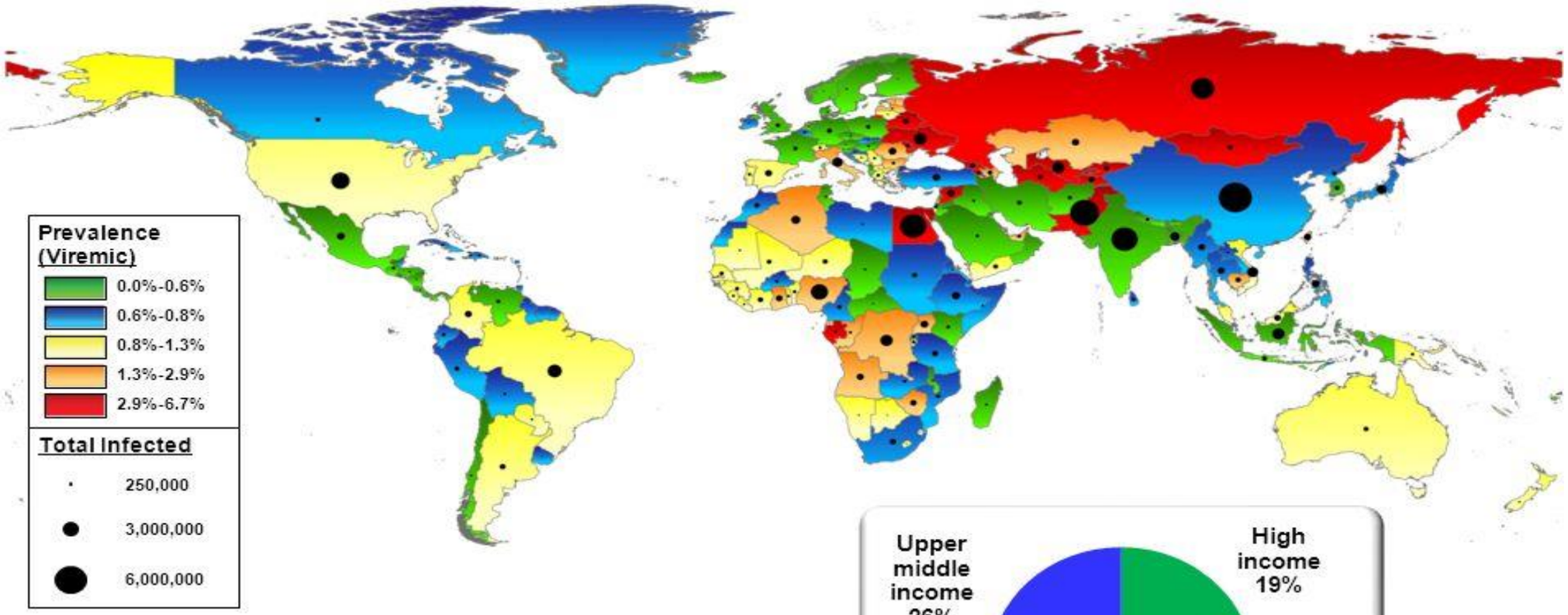
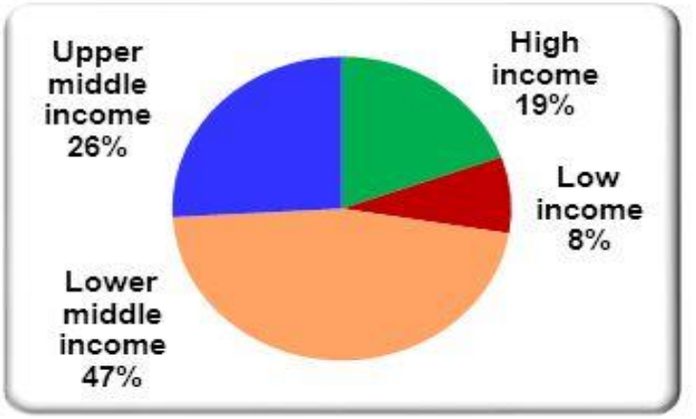
HEPATITIS C GLOBAL EPIDEMIOLOGY

An estimated 70 million (56 million – 90 million) individuals are infected with HCV (viremic), a prevalence of 1% (0.8%-1.2%) in 2016



In 2015, 500K patients were treated and cured with DAAs.



Source: Polaris Observatory (http://www.polarisobservatory.com/)

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HEPATITIS C IN THE UNITED STATES

- Prevalence HCV viremia ~1%
- Viremic population ~3 million persons
- Genotype distribution:

1a	46%	3	9%
1b	26%	4	6%
2	11%	6	1%

Lancet Gastroenterol Hepatol 2017;2:161-76

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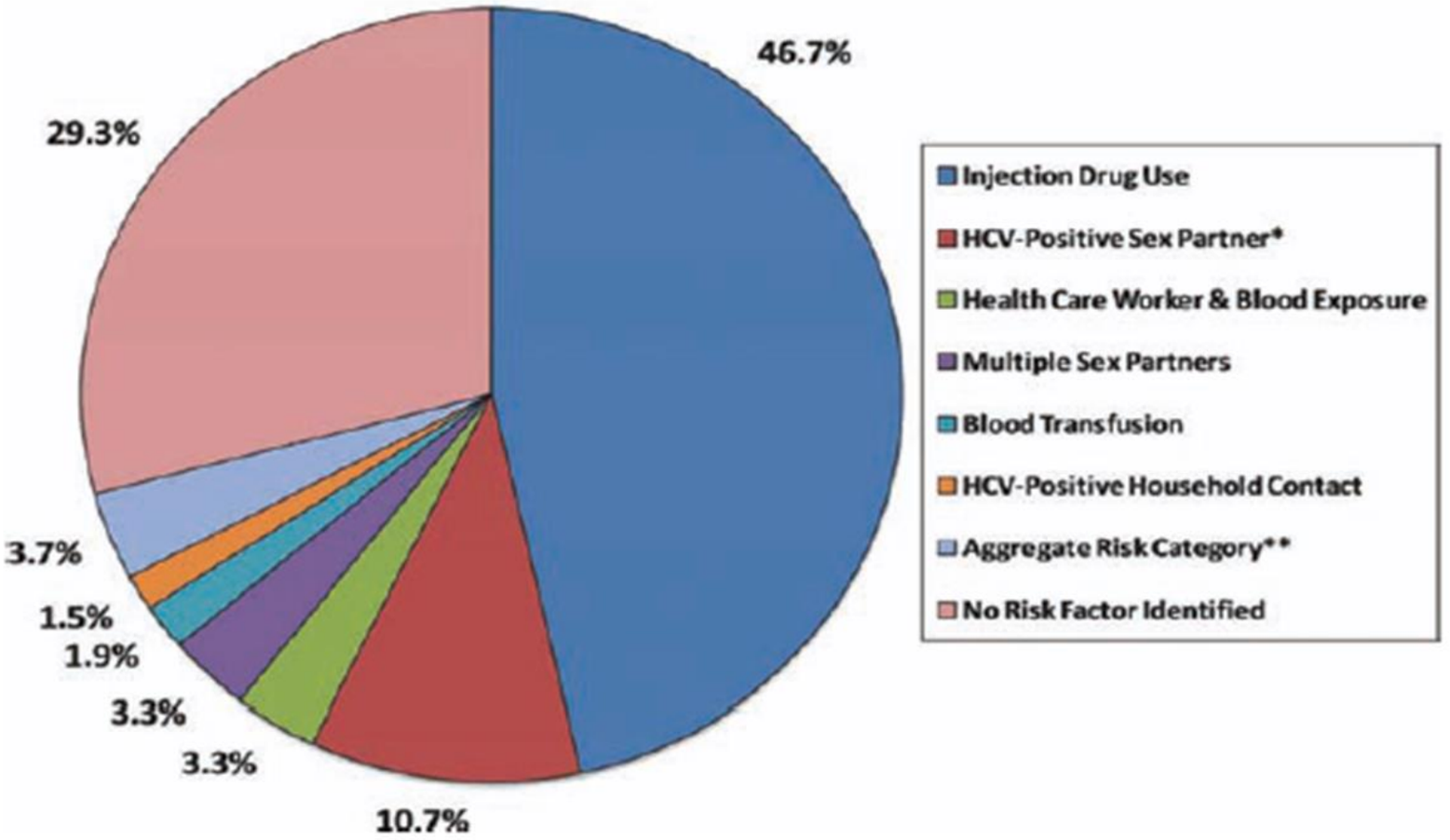
HEPATITIS C RISK FACTORS UNITED STATES



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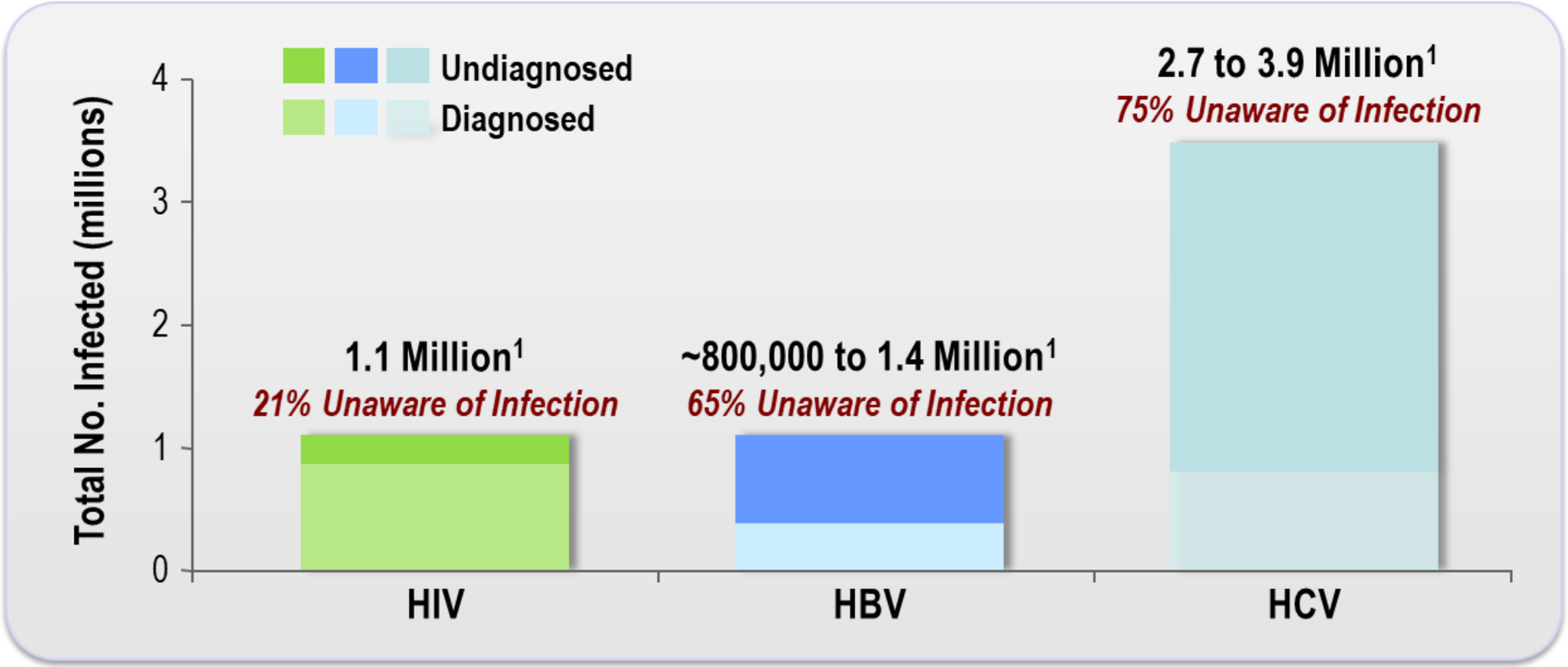
HEPATITIS C IN THE UNITED STATES



- HCV is approximately 4 times as prevalent as HIV and HBV in the United States
- A 2011 study estimated that as many as 5.2 million persons are living with HCV in the United States

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CDC TESTING RECOMMENDATIONS

- Hepatitis C screening at least once in a lifetime for all adults aged ≥18 years
- Hepatitis C screening for all pregnant women during each pregnancy
- Regardless of age or setting prevalence, all persons with risk factors should be tested for hepatitis C, with periodic testing while risk factors persist

Schillie S, et al. CDC Recommendations for Hepatitis C Screening Among Adults — United States, 2020. MMWR Recomm Rep 2020;69(No. RR-2):1–17

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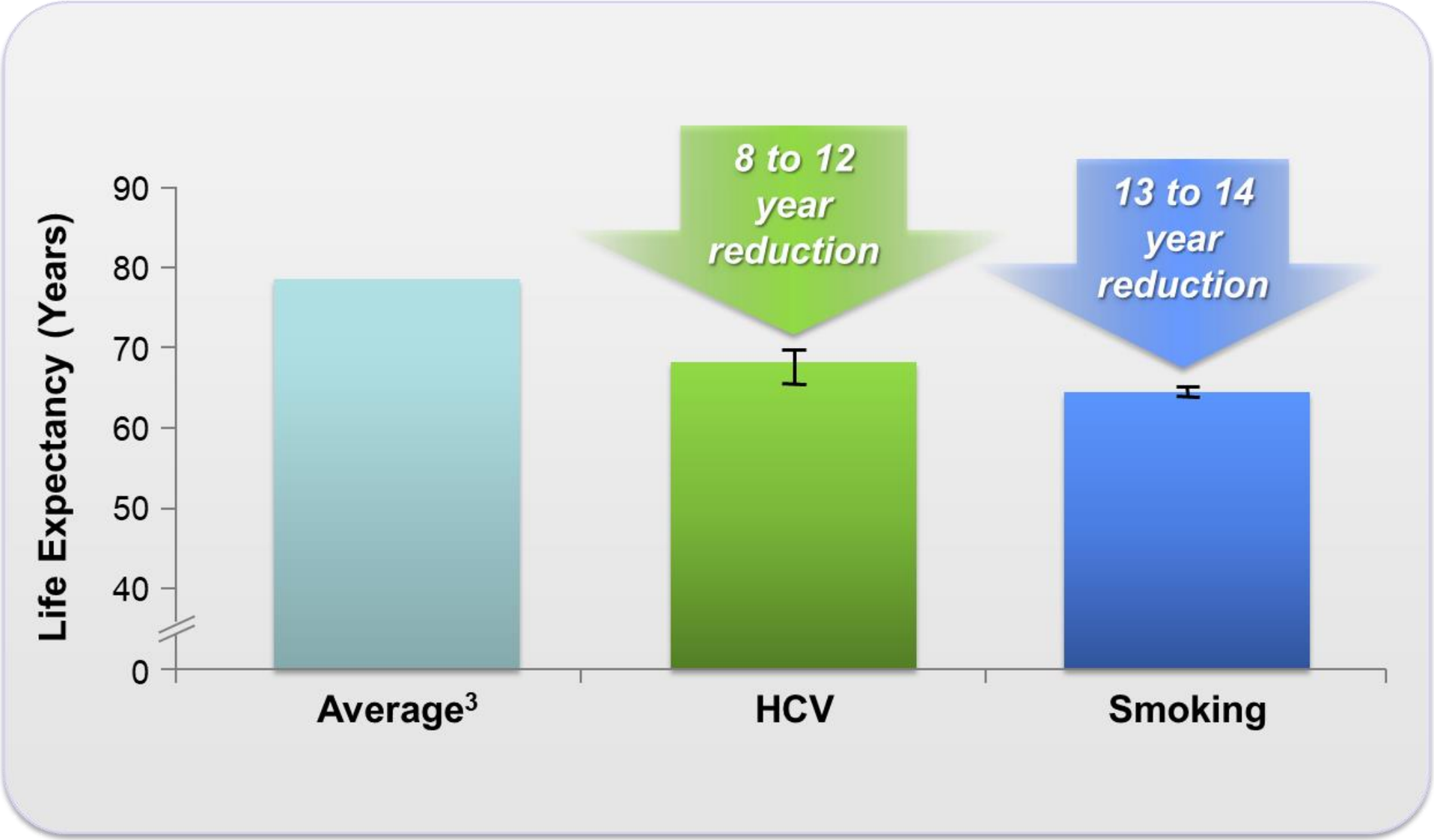
HEPATITIS C IMPACT ON LIFE EXPECTANCY



1. Ryder SD. *J Hepatol*. 2007;47(1):4-6; 2. Centers for Disease Control and Prevention. *MMWR*. 2002;51:300-303; 3. Centers for Disease Control and Prevention. *NVSS*. 2010;58(19):1-135.

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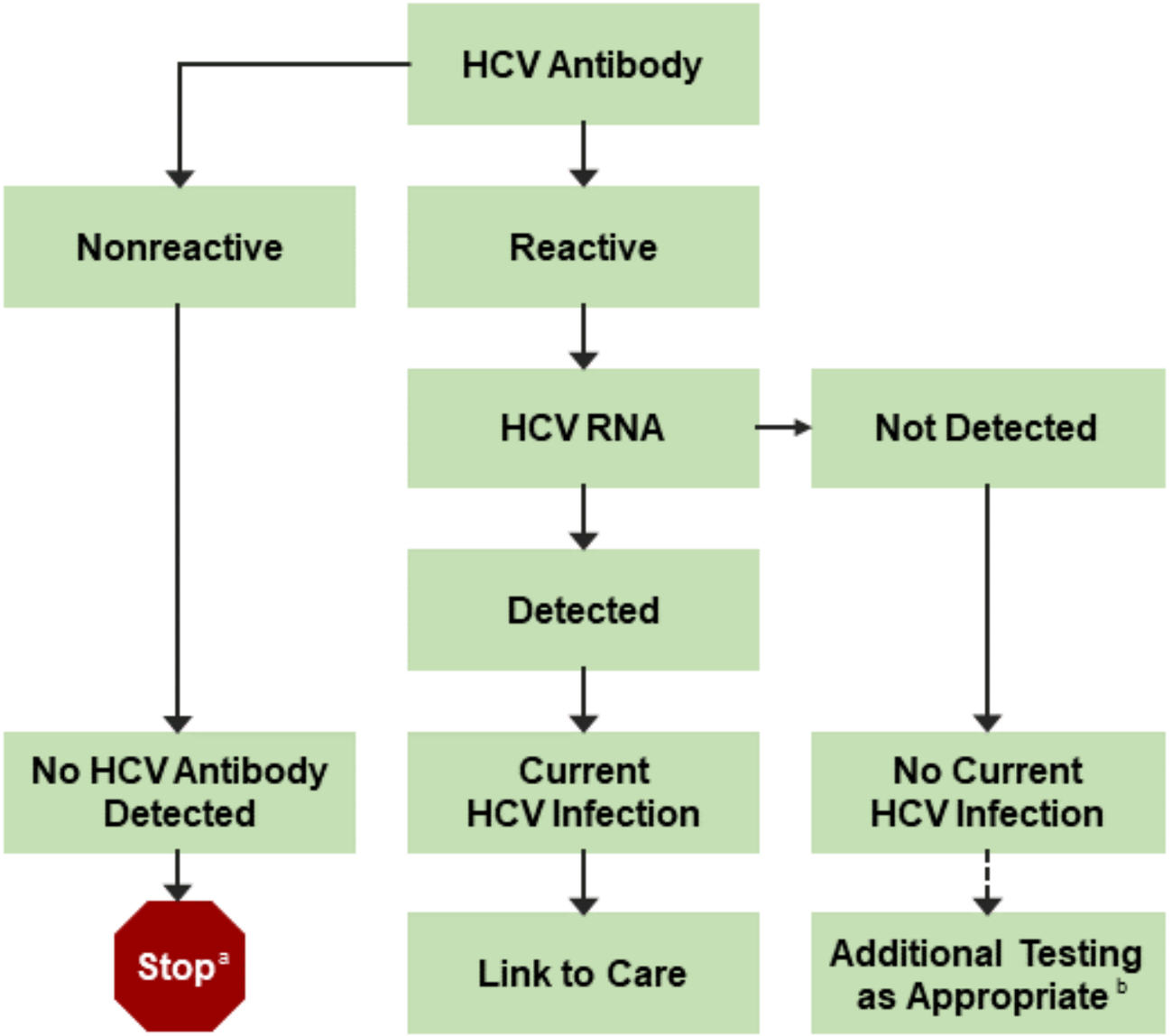
HEPATITIS C DIAGNOSIS



MMWR Morb Mortal Wkly Rep 2013:62:362-5

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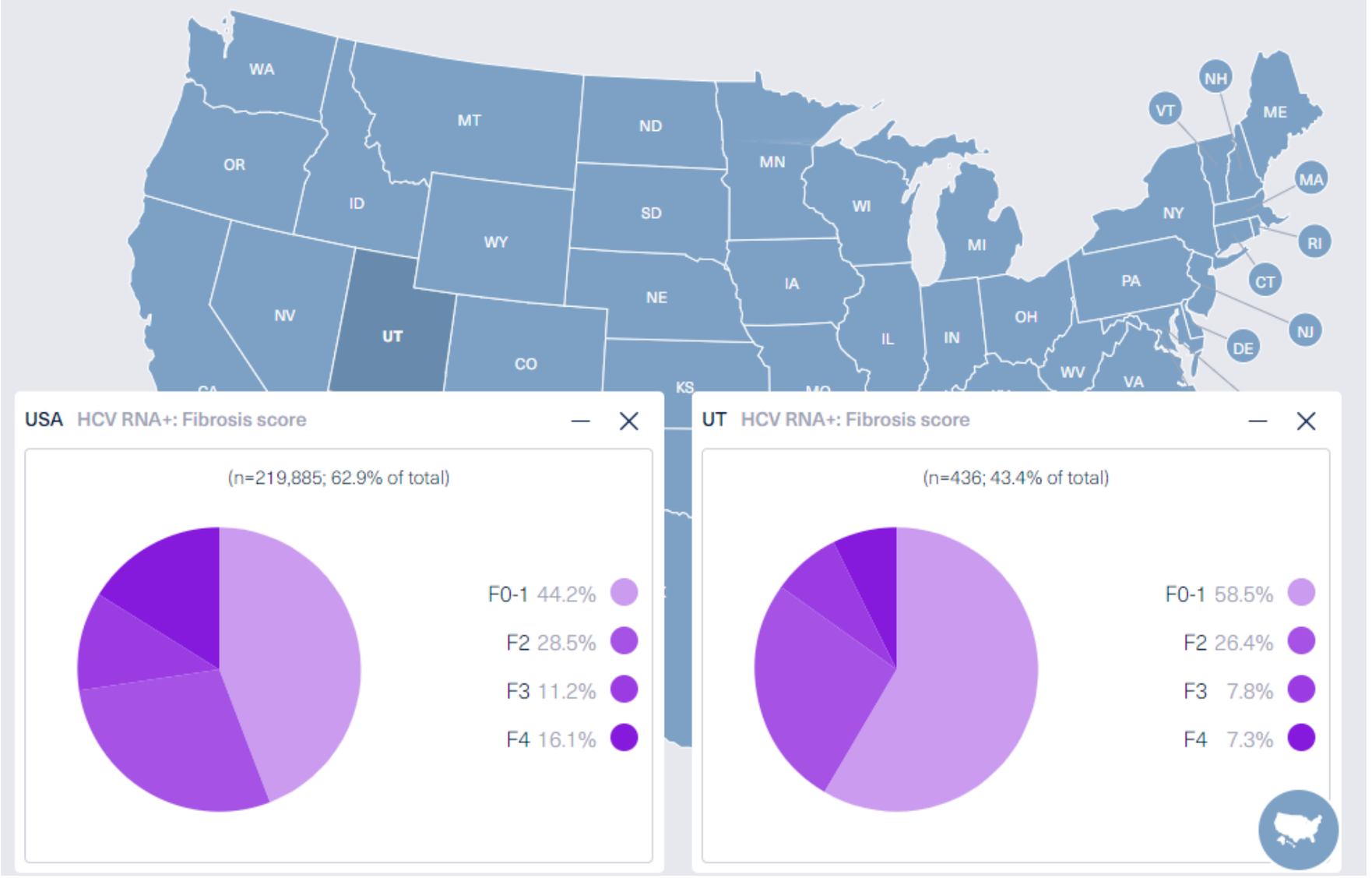
HEPATITIS C IN THE UNITED STATES
DISTRIBUTION OF LIVER FIBROSIS (FIB-4, 2016)



www.mappinghepc.com/maps

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HEPATITIS C TREATMENT GOALS

Eradicate HCV infection

Slow disease progression

Improve histology

Reduce risk of HCC

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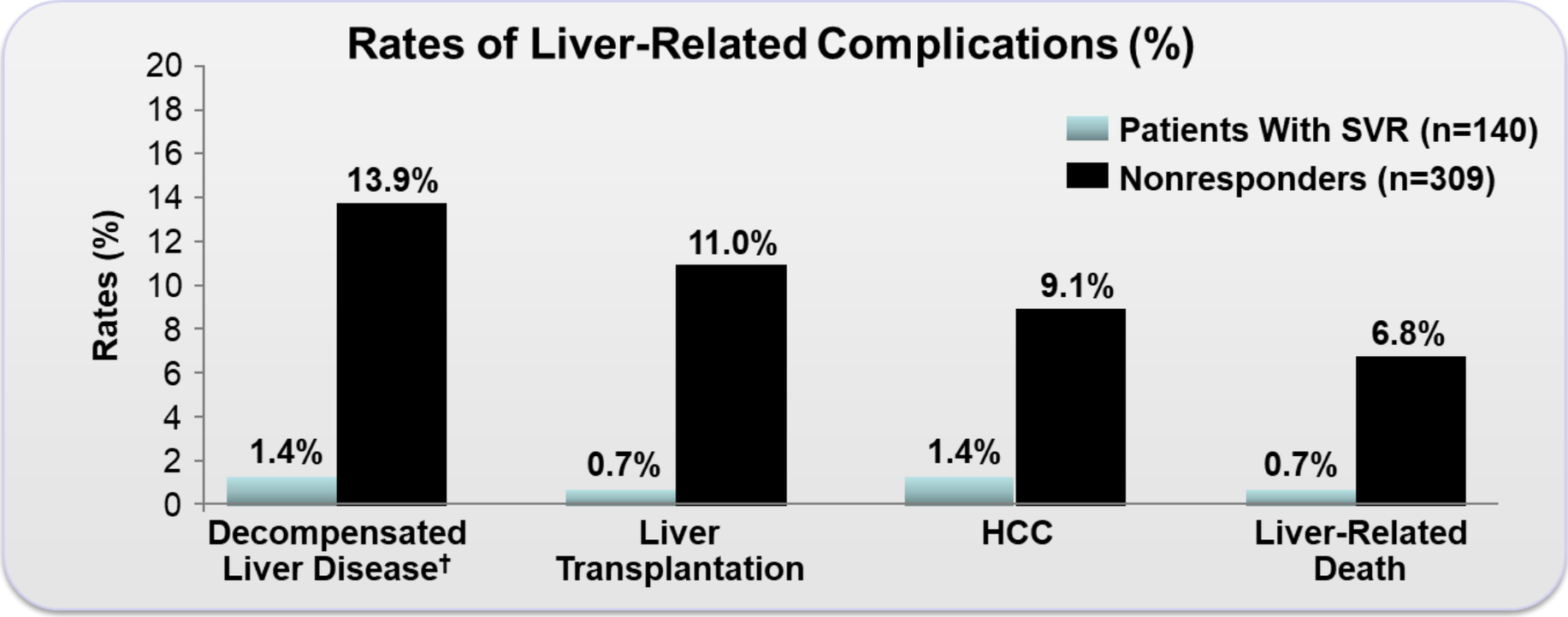
HEPATITIS C
BENEFITS OF THERAPY



- HALT-C Trial: multicenter study 1145 patients with advanced liver fibrosis treated with PEG-IFN/RBV
- Sustained viral response (SVR) significantly reduced HCV-associated complications and mortality

Morgan TR, et al. *Hepatology* 2010;52(3):833-44

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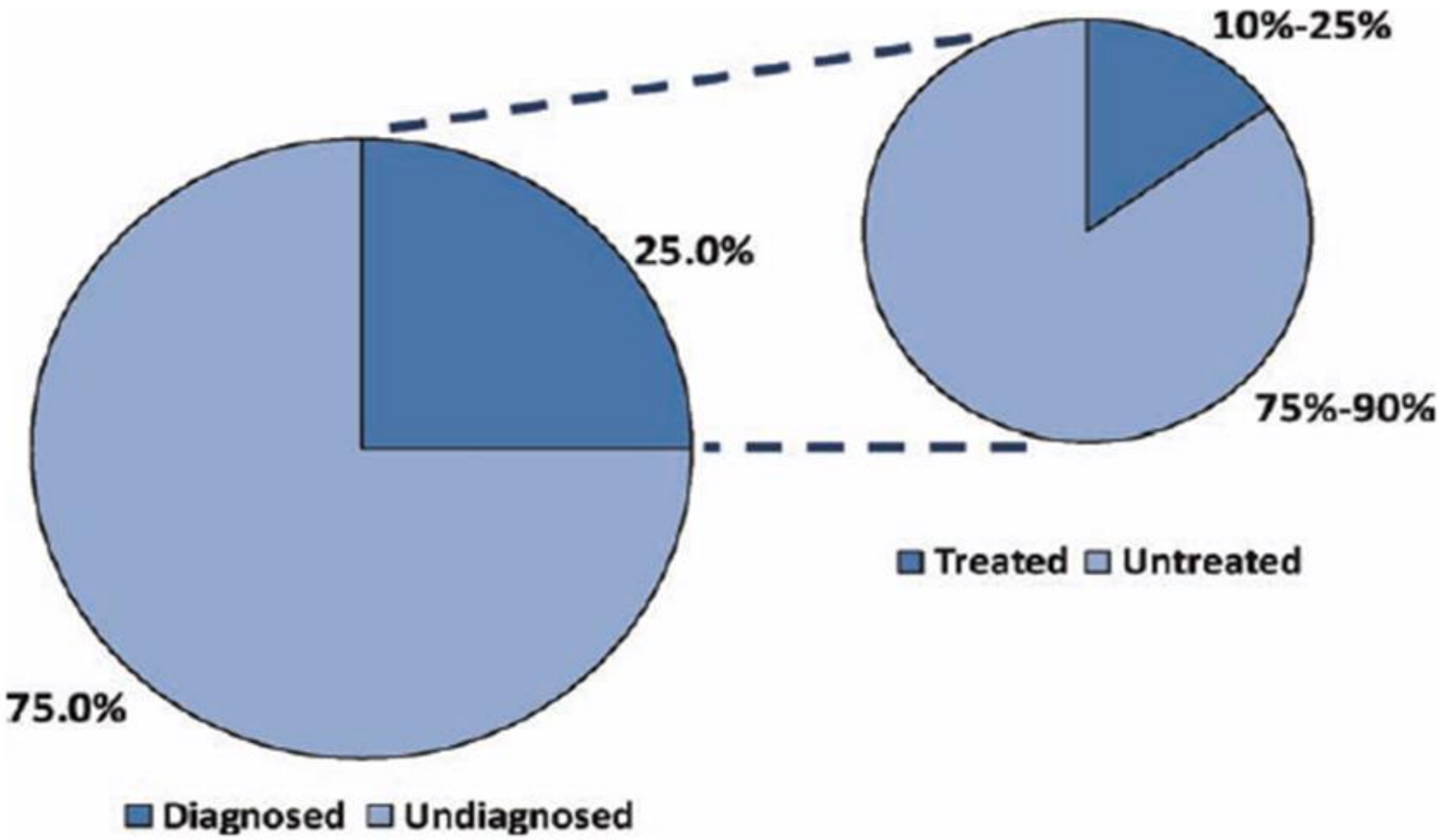
HEPATITIS C IN THE UNITED STATES
PROPORTION OF PERSONS DIAGNOSED AND TREATED



Clinical Liver Disease 2016; 8: 39-42

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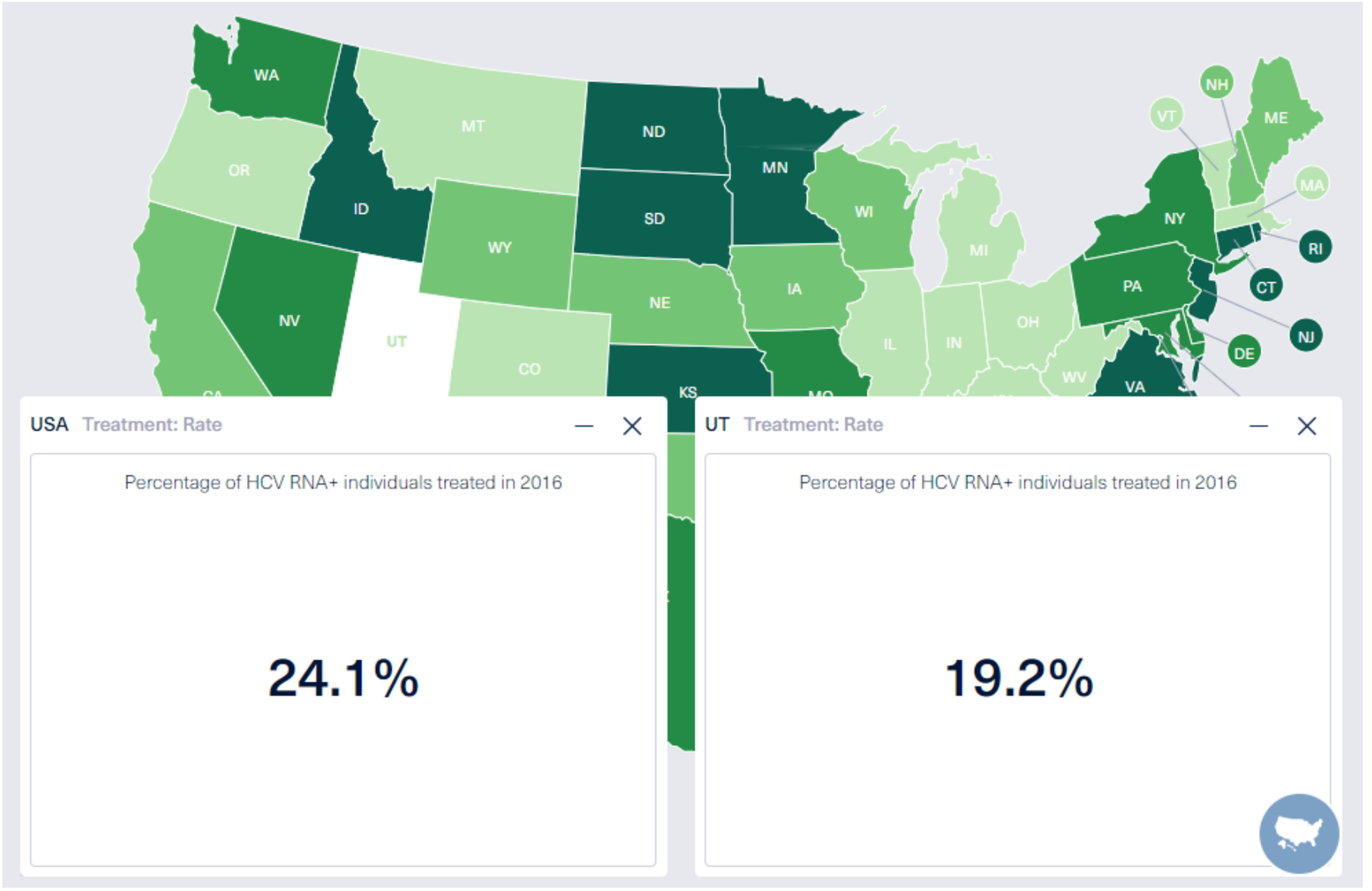
HEPATITIS C IN THE UNITED STATES
PROPORTION HCV RNA (+) PATIENTS STARTED DAA



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DIRECT ACTING ANTIVIRALS (DAA)

NS3/4A Protease Inhibitors (PI) **-previr**
- High potency
- Multi-genotypic coverage
- Intermediate to high barrier to resistance

NS5B Nucleoside Inhibitors (NI) **-buvir**
- Intermediate potency
- Pan-genotypic coverage
- High barrier to resistance

NS5A Inhibitors **-asvir**
- High potency
- Multi-genotypic coverage
- Low to intermediate barrier to resistance

NS5B Non-Nucleoside Inhibitors (NNI) **-buvir**
- Intermediate potency
- Limited-genotypic coverage
- Low barrier to resistance

Poordad F, Dieterich D. J Viral Hep 2012;19:449-464

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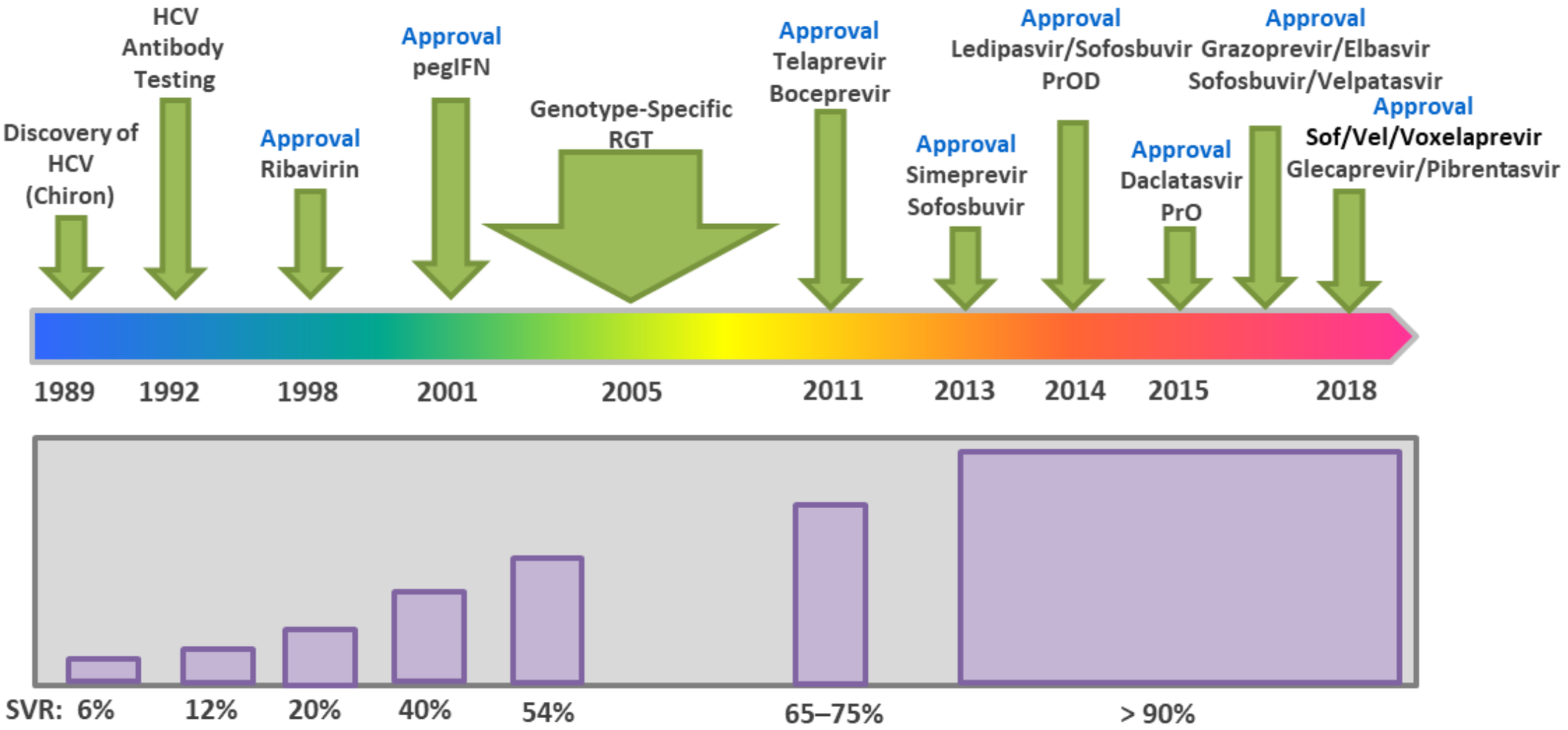
EVOLUTION OF ANTIVIRAL THERAPY



Courtesy: Hugo E. Vargas, MD, Mayo Clinic

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FACTORS THAT IMPACT DAA SELECTION AND TREATMENT DURATION

- Viral genotype
- Presence/absence liver cirrhosis
  - Compensated vs. decompensated
- Prior treatment experience
  - NS5A resistance, RAVs in prior non-responders
- Renal function (CKD stage 4 or 5)
- Drug-drug interactions
- Solid organ transplant status

Clinical Liver Disease 2016; 8: 39-42

www.hcvguidelines.org

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CONCLUSIONS

- **5 million** persons living with HBV or HCV in United States
- **65-75%** infected individuals not aware of their status
- **HBV is "treatable", although not yet "curable", long-term viral suppression is common**
- New antiviral and immune modulatory therapies in development
- **HCV is curable**
- Newer DAA regimens:
  - Treat all genotypes (pangenotypic)
  - Shorter duration treatment (8-12 weeks in most cases)

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NON-ALCOHOLIC FATTY LIVER DISEASE

- Initial histologic description by Dr. J. Ludwig and colleagues at Mayo Clinic in 1980
- Histopathologic injury similar to that seen in alcohol liver disease among patients without significant alcohol intake = "nonalcoholic fatty liver disease" (NAFLD)

Mayo Clin Proc 1980;55:434

JAMA 2015;313:2263

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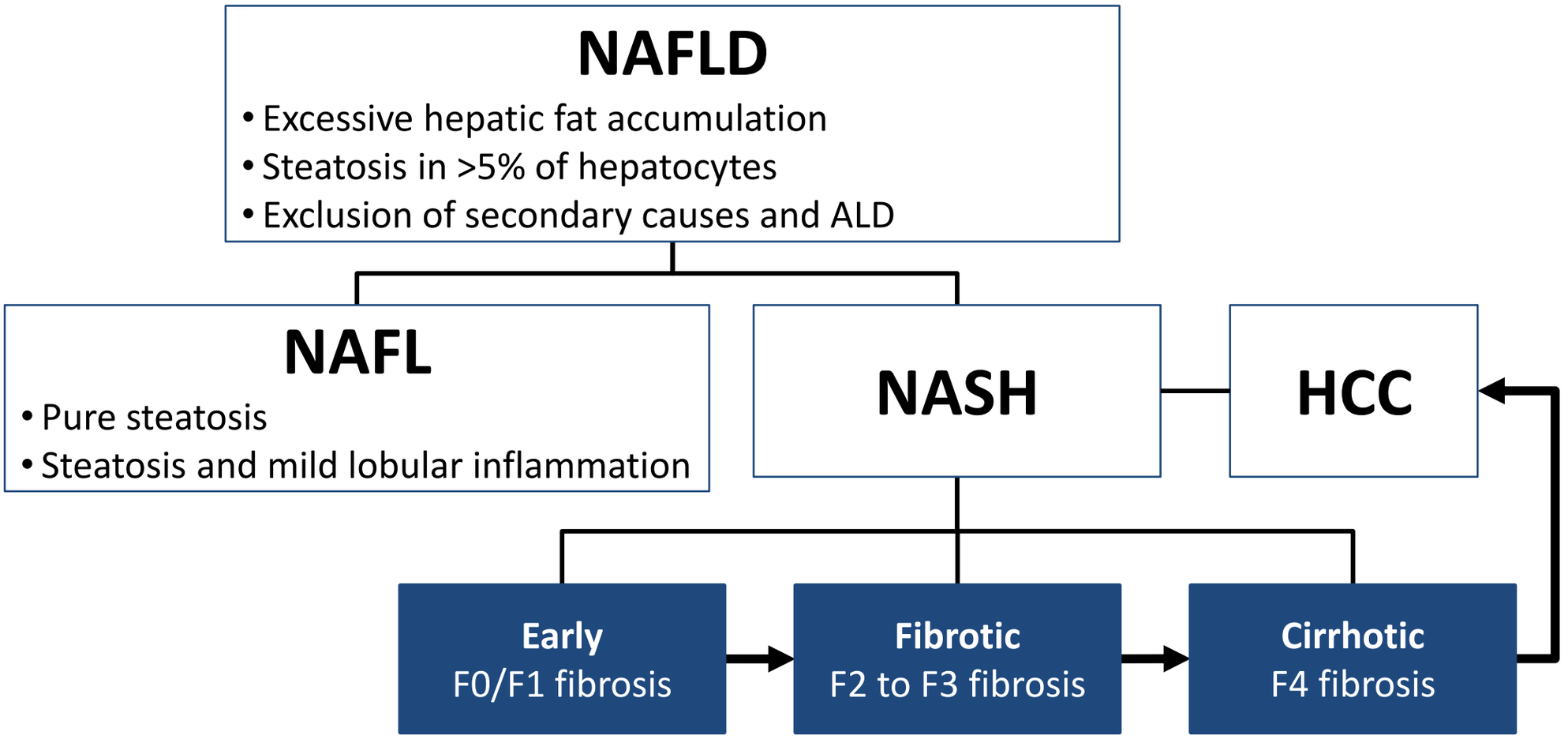
DEFINITIONS



Definitive diagnosis of NASH requires a liver biopsy

EASL CPG NAFLD. J Hepatology 2016

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NAFLD EPIDEMIOLOGY
GENERAL POPULATION

- **NAFLD most common cause liver disease worldwide**
- Global prevalence 25% adult population
  - Middle East 32%
  - South America 30%
  - United States 30%
  - Africa 14%
- NASH among NAFLD patients: 25-30%
- NASH prevalence 1.5%-6.5% adults worldwide

Hepatology 2016;64:73

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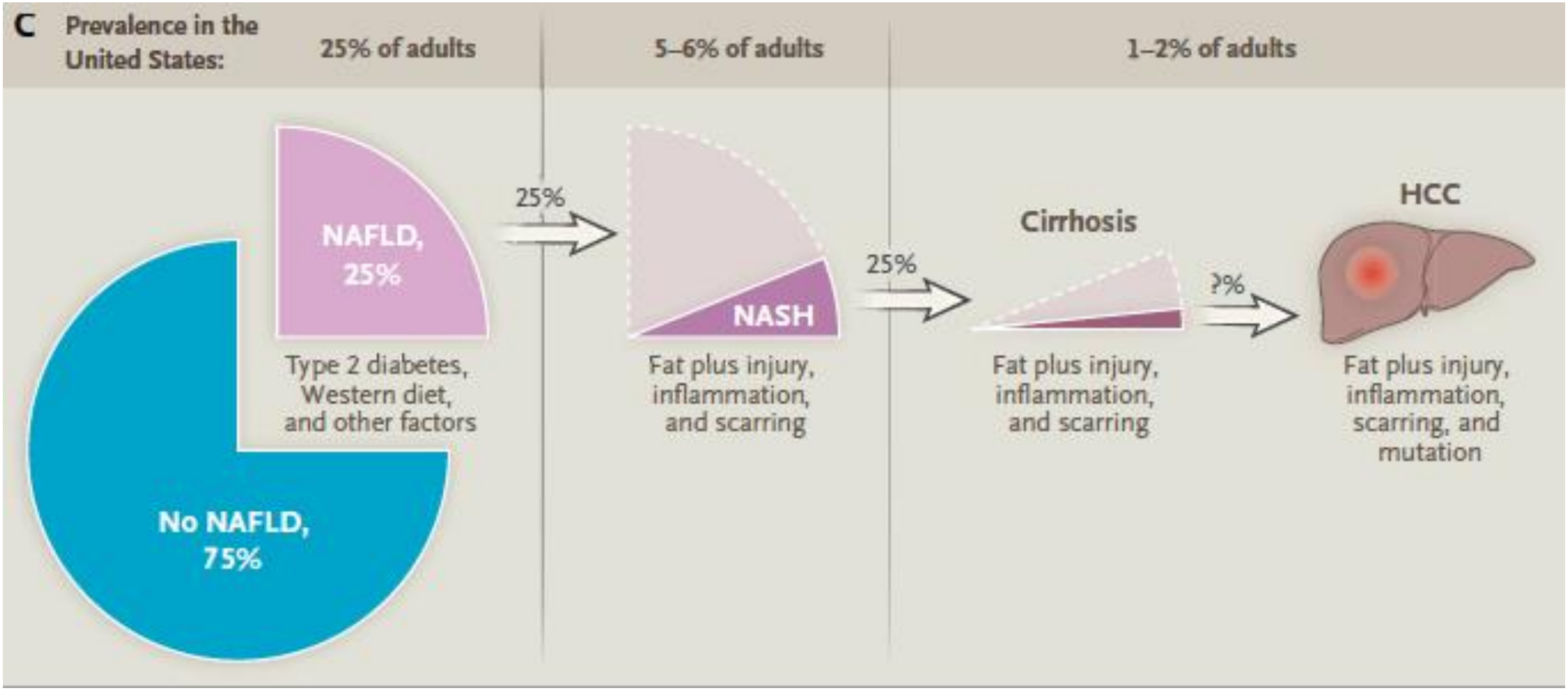
NAFLD PREVALENCE UNITED STATES



N Engl J Med 2017;377:2063

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NAFLD EPIDEMIOLOGY
HIGH RISK POPULATION

- **Obesity (BMI >30 kg/m^2)**
  - NAFLD affects 50% obese persons
  - NAFLD affects 95% undergoing bariatric surgery
- Type 2 DM 25%-60%
- Dyslipidemia 70%
- Hypertension 40%
- Metabolic syndrome 40%

Hepatology 2016;64:73

Hepatology 2018;67:328

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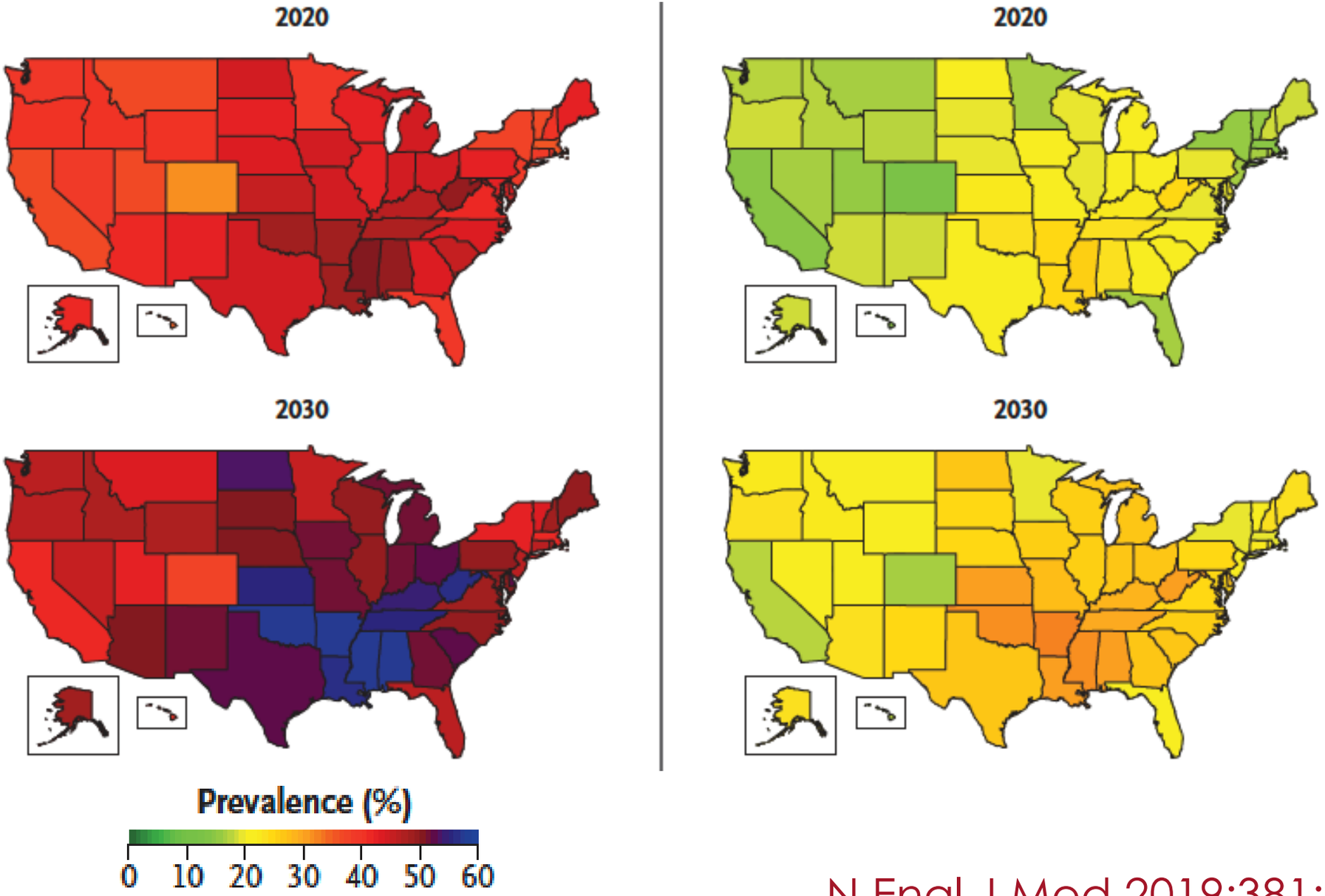
PROJECTED OBESITY IN ADULTS IN THE US

BMI >30 kg/m^2

BMI >35 kg/m^2



N Engl J Med 2019;381:2440

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NAFLD
ECONOMIC BURDEN

- United States: 64 million persons NAFLD → annual medical cost $100 billion ($1,600/person)

- NASH with fibrosis: 3 to 4 million persons → annual cost $10 - $15 billion

Hepatology 2016;64:1577

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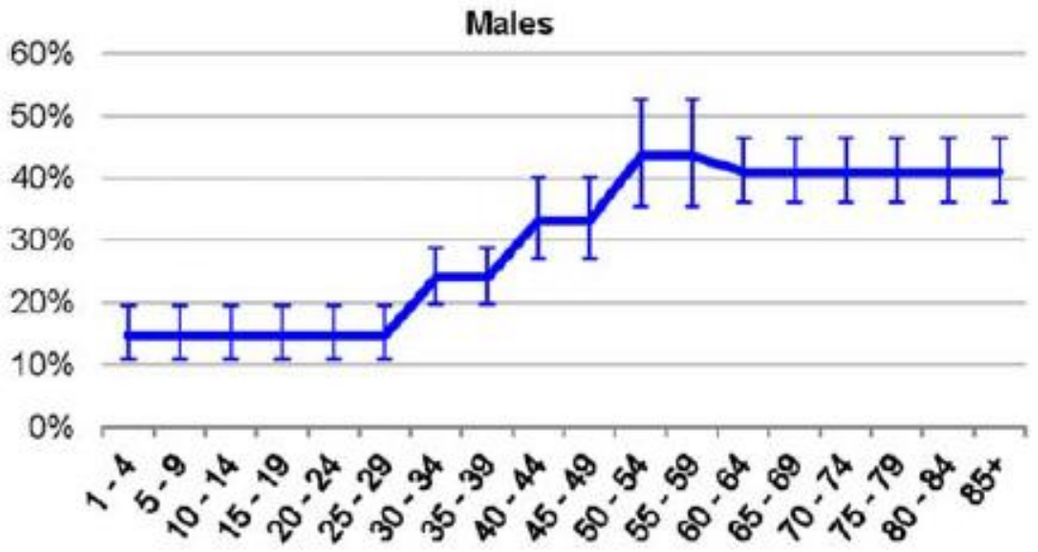
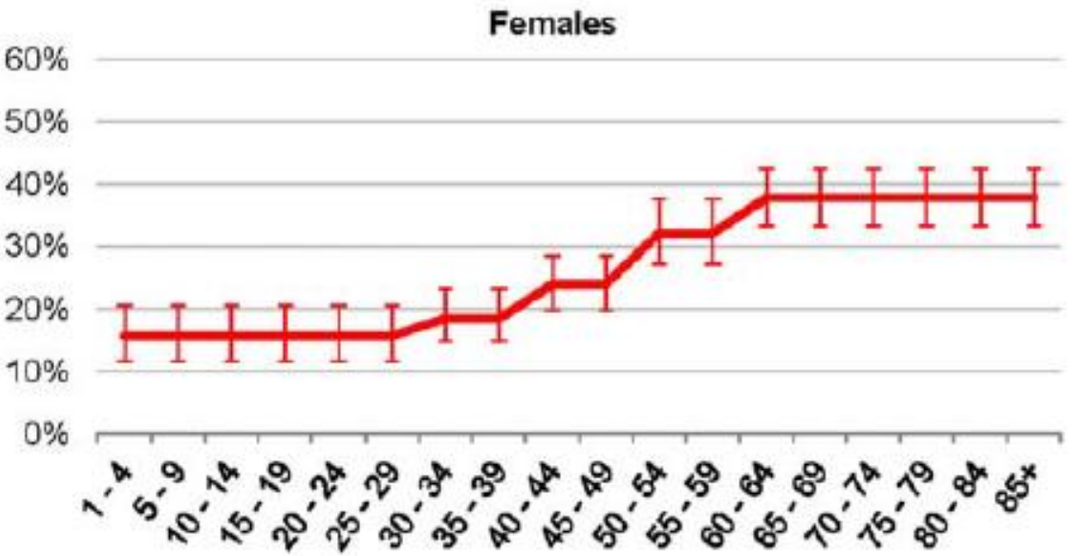
UNITED STATES PREVALENCE NAFLD ADJUSTED BY AGE AND SEX





Hepatology 2018;67:123

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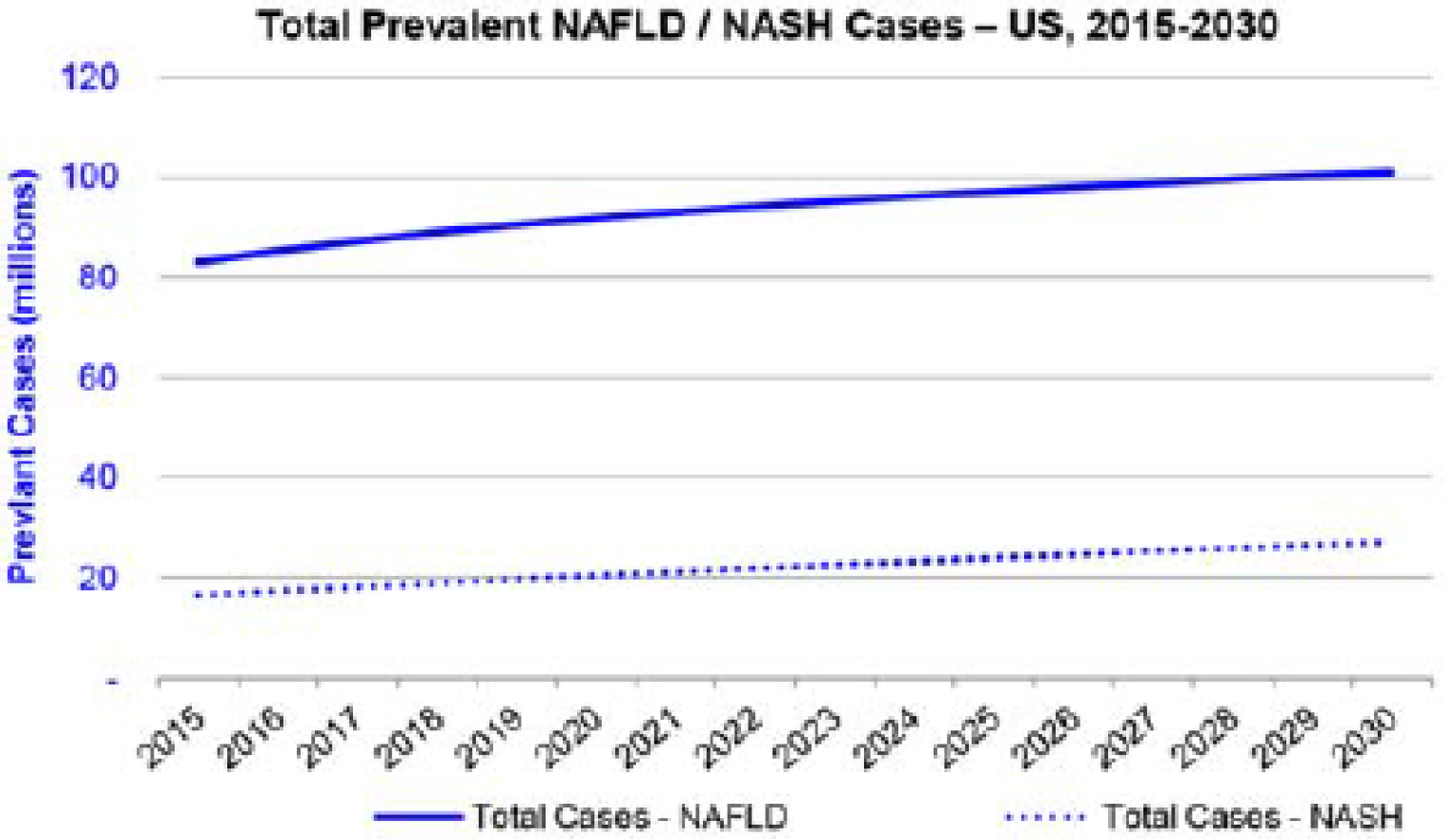
NAFLD CURRENT AND FUTURE BURDEN OF DISEASE IN THE UNITED STATES



Hepatology 2018;67:123

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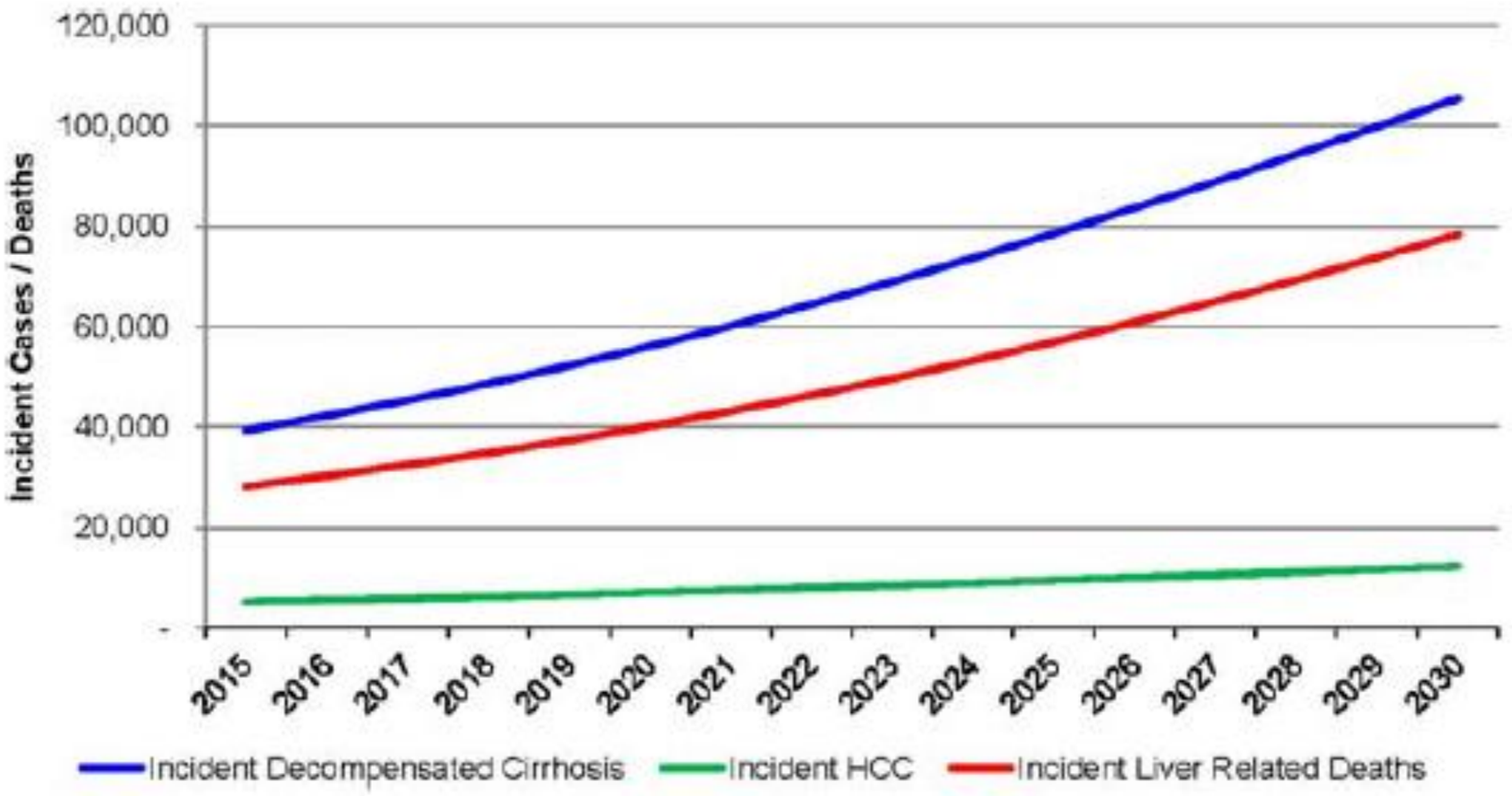
NAFLD CURRENT AND FUTURE BURDEN OF DISEASE IN THE UNITED STATES



Hepatology 2018;67:123

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NAFLD PROGNOSIS

- Strong association between NAFLD and cardio-metabolic complications resulting increased cardiovascular morbidity and mortality
- Main causes of mortality: **cardiovascular disease**, **non-liver malignancies** and **liver-related mortality** with **increased risk** of **HCC**
- **Stage of liver fibrosis <u>most</u> important predictor cardiovascular and liver-related complications and death**

Hepatology 2012;55:2005

Clin Liver Dis 2018;22:11

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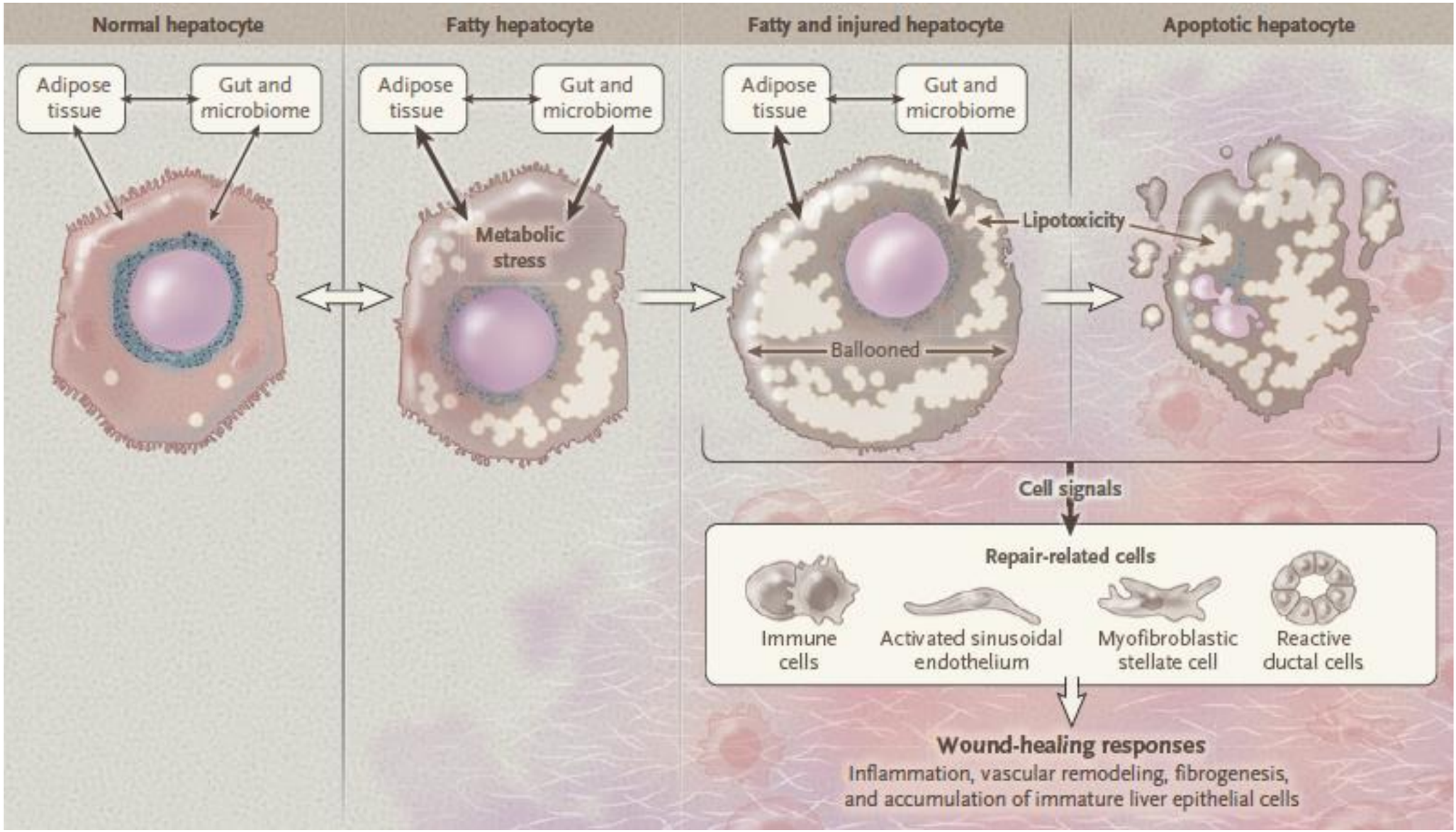
NASH PATHOGENESIS



N Engl J Med 2017;377:2063

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PATHOPHYSIOLOGY
GENETICS

- Specific SNP influence development NAFLD & NASH
  - **Patatin-like phospholipase 3 (*PNPLA3*) or adiponutrin single nucleotide polymorphism confers increased risk NASH and liver fibrosis and is more prevalent among Hispanics**

Hepatology 2011;53:1883-1894

N Engl J Med 2010;362:1082-1089

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DIAGNOSIS
CLINICAL QUESTIONS

- Does this patient have NAFLD or another liver disease?
- If NAFLD, does the patient have NASH?
- If NASH, does the patient have advanced fibrosis?

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NAFLD
IMAGING DIAGNOSIS

Comparison of ultrasound, computed tomography, and MRI for detection and evaluation of hepatic steatosis

Imaging Modality	Strengths	Limitations
US	• Widely accessible • Inexpensive	• Suboptimal sensitivity/ specificity for mild steatosis • Operator dependent • Qualitative
CT	• High sensitivity for moderate to severe steatosis	• Suboptimal sensitivity/ specificity for mild steatosis • Radiation risks
MRI	• Best sensitivity and sensitivity for steatosis (even mild steatosis) • May be superior to biopsy specimen in terms of estimating total hepatic fat • Can be used to follow patients longitudinally with treatment	• Expensive • Limited availability as a screening tool

Clinics Liver Dis 2018;22:93

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NAFLD/NASH DIAGNOSTIC EVALUATION

Least invasive	Fibrosis	NASH
	Fibroscan	
	NAFLD Fibrosis store/BARD Fibrotest/ELF score	CK18 NASHTest/ FibroMax
		Contrast USS
Most invasive	Liver biopsy	Liver biopsy

Aliment Pharmacol Ther 2011;33:525-540

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NAFLD
DIAGNOSTIC IMAGING FIBROSIS

- Technological advances have allowed development of non-invasive assessment of **liver elasticity as surrogate of fibrosis**
  - Transient elastography (FibroScan®)
  - MR elastography
  - Acoustic radiation force impulse (ARFI)

Clinics Liver Dis 2018;22:73

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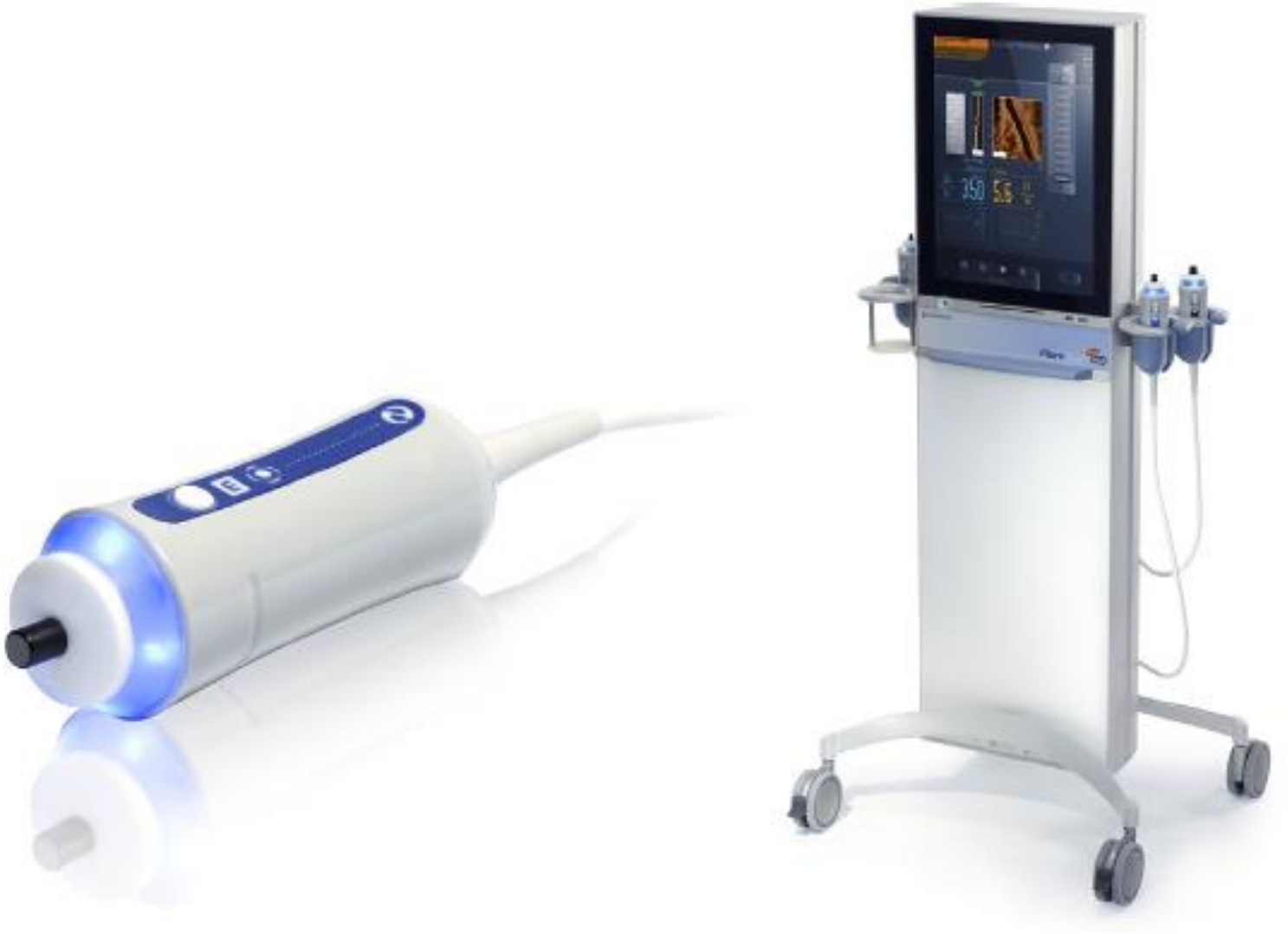
VIBRATION CONTROLLED TRANSIENT ELASTOGRAPHY (FIBROSCAN®)



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VIBRATION CONTROLLED TRANSIENT ELASTOGRAPHY (FIBROSCAN®)

Table 4
Fibrosis stages and proposed transient elastography cutoffs

Fibrosis Stage	Cutoff (kPa)	Sensitivity (%)	Specificity (%)
F ≥2	5.35–7.4	87.5	78.4
F ≥3	8.0–12.85	93.7	91.1
F ≥4	10.3–17.5	96.2	92.2

Data from Hashemi SA, Alavian SM, Gholami-Fesharaki M. Assessment of transient elastography (FibroScan) for diagnosis of fibrosis in non-alcoholic fatty liver disease: a systematic review and meta-analysis. Caspian J Intern Med 2016;7(4):242–52.

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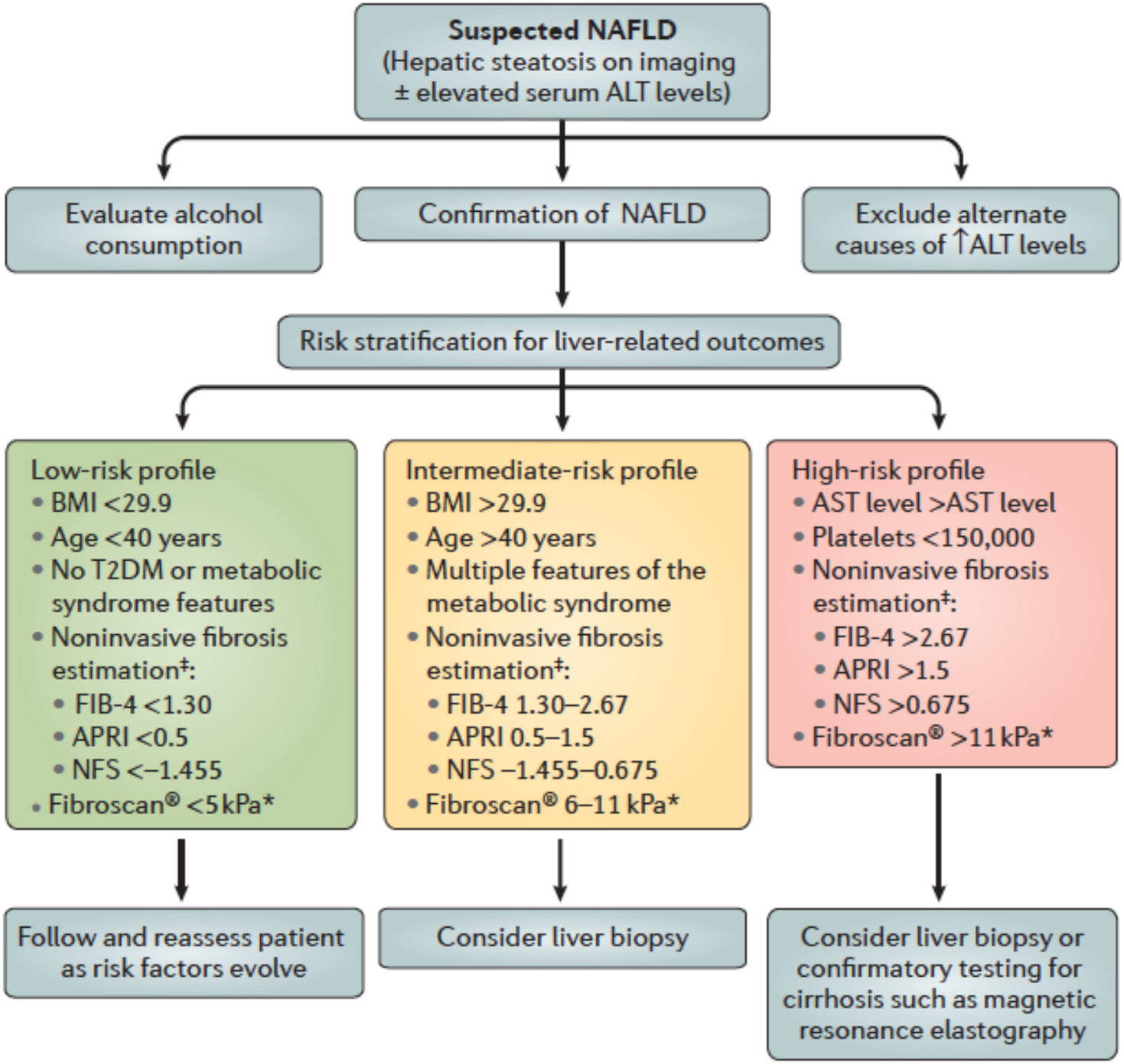

Nat Rev GI Hepatol 2016;13:196

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WHAT I WANT TO KNOW FROM A LIVER BIOPSY?

- Does the patient have NAFL or NASH?
- Is there an alternative diagnosis?
- How much fibrosis is there (F0-F4)?

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NAFLD HISTOLOGY SCORING SYSTEMS

NAS (NASH CRN)		SAF (FLIP consortium)	
Steatosis	0–3	Steatosis	S0–S3
Ballooning	0–2	Activity	A0–A4
		Ballooning (0–2)	
Inflammation	0–3	Inflammation (0–2)	
		Fibrosis	F0–F4
NAS	0–8	SAF	$S_{0–4}$ $A_{0–4}$ $F_{0–4}$

Gastroenterology 2016

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NASH AND LIFESTYLE MODIFICATIONS

Table 1. Lifestyle Modifications to Mitigate Nonalcoholic Steatohepatitis.*

Lose 7% of body weight if overweight or obese
Limit consumption of fructose-enriched beverages
Limit consumption of alcohol (≤1 drink/day for women and ≤2 drinks/day for men)
Drink two or more cups of caffeinated coffee daily

N Engl J Med 2017;377:2063

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CURRENT MANAGEMENT NAFLD/NASH

- Weight loss 7-10% and moderate caloric restriction coupled with regular exercise are most beneficial
- Pioglitazone or vitamin E in biopsy-proven NASH
- Bariatric surgery in selected patients can result in histologic improvement

Aliment Pharmacol Ther 2017;45:494

JAMA 2015;313:2263

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BARIATRIC SURGERY AND NAFLD/NASH

- NAFLD present 24%-98% obese patients
- Meta-analysis 15 studies evaluating histologic changes after bariatric surgery:
  - 92% improved/resolved steatosis
  - 82% improved/resolved NASH
  - 66% improved/resolved fibrosis

Hepatic Medicine: Evidence & Research 2014:6 103-112

Clin Gastroenterol Hepatol 2008;6:1396-1402

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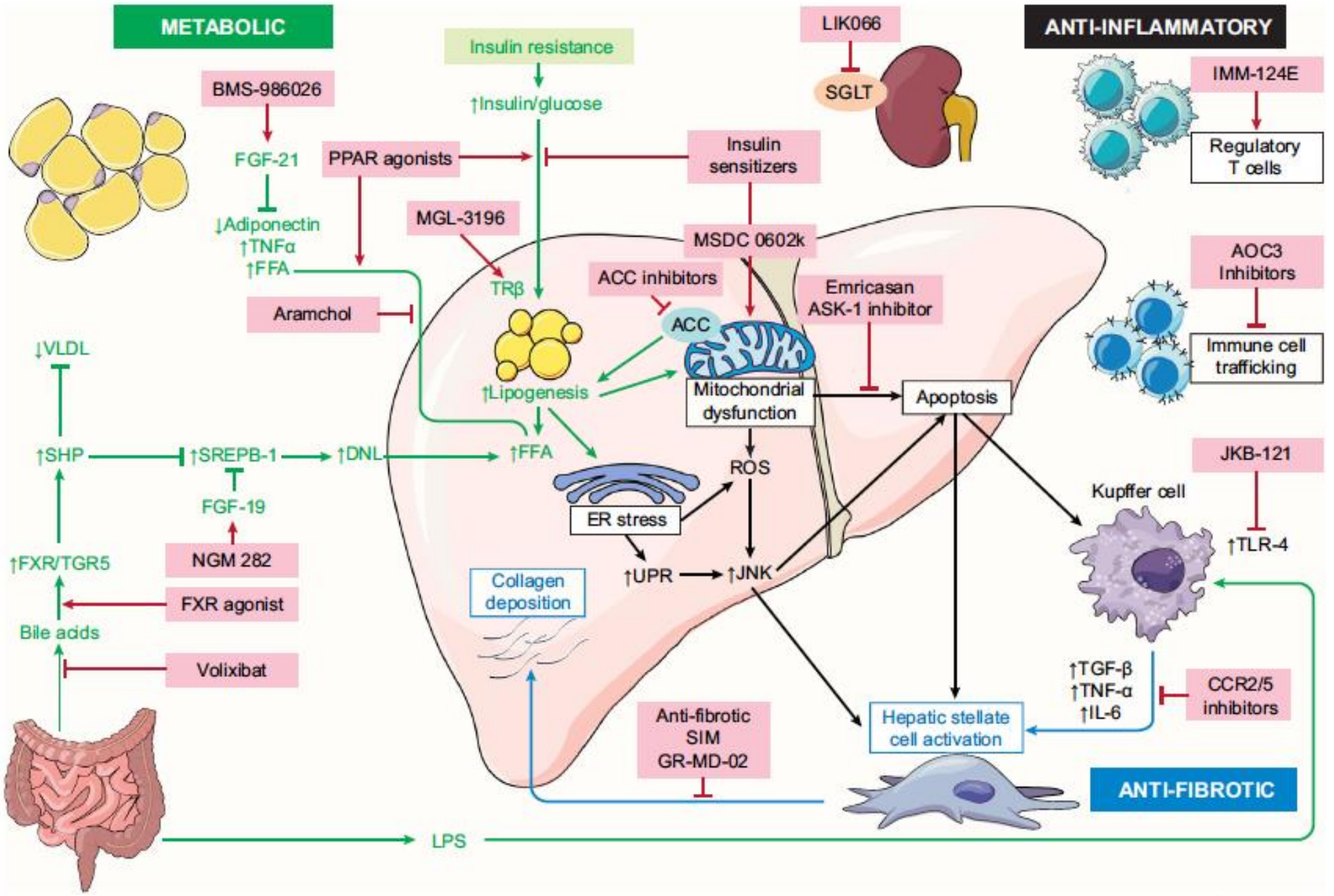
MECHANISM OF ACTION TREATMENT NAFLD/NASH



J Hepatology 2018

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NAFLD TREATMENT HISTOLOGIC OUTCOMES

Drug	Steatosis	Reduction NAS	NASH resolution	Fibrosis
Pioglitazone	✓	✓	✓	✓
Vitamin E	✓	✓	✗	✗
Obeticholic acid	✓	✓	✗	✓
Elafibranor	✓	✗	✓	✗
Liraglutide	✓	✗	✓	✗
Cenicriviroc	✗	✗	✗	✓

Aliment Pharmacol Ther 2017;45:494

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CONCLUSIONS

- NAFLD growing public health problem closely tied to obesity epidemic
- NAFLD associated with increased cardiovascular morbidity and mortality
- Liver biopsy currently best tool to stage NAFLD and select patients with NASH and fibrosis at increased risk for poor clinical outcomes

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CONCLUSIONS

- Lifestyle changes are mainstay of therapy
- Bariatric surgery in select patients improves histology
- Currently limited pharmacologic intervention, although multiple pharmaceuticals are in clinical trials

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