B或C型肝炎性肝硬化的治療方針

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Outlines

- 如何診斷肝硬化
- 如何評估肝硬化的嚴重度
- 治療病毒性肝硬化的藥物
- 健保對於病毒性肝硬化的治療規定
- 治療病毒性肝硬化能改善Child-Pugh scores
- 治療病毒性肝硬化可以逆轉纖維化
- 治療病毒性肝硬化可以延長病人的存活
- 治療病毒性肝硬化可以降低肝癌的發生率
- 治療病毒性肝硬化可以降低肝癌的復發率

如何診斷肝硬化

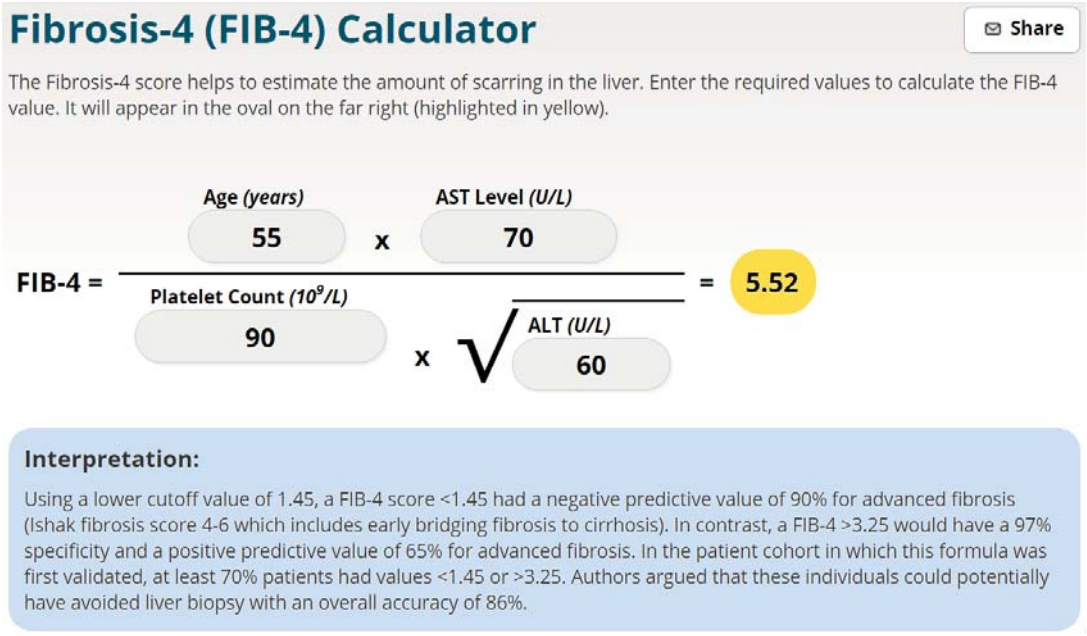
如何診斷肝硬化

- 超音波/CT/MRI
- 腹腔鏡
- 肝穿刺(切片)
- 抽血
- Fibroscan / ARFI

Histologic Scoring Systems for Fibrosis

Fibrosis	METAVIR	Ishak
None	0	0
Portal fibrosis (some)	1	1
Portal fibrosis (most)	1	2
Bridging fibrosis (occasional)	2	3
Bridging fibrosis (marked)	3	4
Incomplete cirrhosis	4	5
Cirrhosis	4	6

Fibrosis-4 (FIB-4)



健保 F3 之定義為：FIB-4 >= 3.25

AST to Platelet Ratio Index (APRI)

AST to Platelet Ratio Index (APRI) Calculator

Share

This is an **A**ST to **P**latelet **R**atio **I**ndex (APRI) calculator tool. Enter the required values to calculate the APRI value. The APRI Score will appear in the oval on the far right (highlighted in yellow). Most experts recommend using 40 IU/L as the value for the AST upper limit of normal when calculating an APRI value.

$$\text{APRI} = \frac{\dfrac{\text{AST Level } (IU/L)}{\text{AST (Upper Limit of Normal) } (IU/L)}}{\text{Platelet Count } (10^9/L)} \times 100 = 1.944$$

AST Level *(IU/L)*: 70

AST (Upper Limit of Normal) *(IU/L)*: 40

Platelet Count *(10^9/L)*: 90

Interpretation:

In a meta-analysis of 40 studies, investigators concluded that an APRI score greater than 1.0 had a sensitivity of 76% and specificity of 72% for predicting cirrhosis. In addition, they concluded that APRI score greater than 0.7 had a sensitivity of 77% and specificity of 72% for predicting significant hepatic fibrosis.[1]
For detection of cirrhosis, using an APRI cutoff score of 2.0 was more specific (91%) but less sensitive (46%). The lower the APRI score (less than 0.5), the greater the negative predictive value (and ability to rule out cirrhosis) and the higher the value (greater than 1.5) the greater the positive predictive value (and ability to rule in cirrhosis); midrange values are less helpful. The APRI alone is likely not sufficiently sensitive to rule out significant disease. Some evidence suggests that the use of multiple indices in combination (such as APRI plus FibroTest) or an algorithmic approach may result in higher diagnostic accuracy than using APRI alone.[2]

Vibration Controlled Transient Elastography (VCTE, Fibroscan)

Cut-off Value of Fibroscan

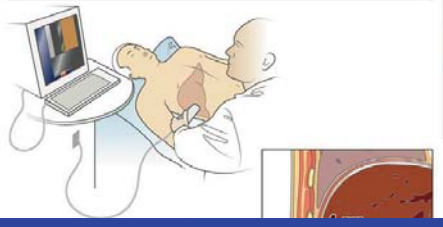
Chronic Hepatitis C
- Cutoffs to know:
  - 7.3 kPa suggests significant fibrosis
  - 12.5 kPa suggests cirrhosis

Chronic Hepatitis B
- Must know: HBV DNA
- Cutoffs to know:
  - 11.7 kPa suggests cirrhosis
  - If normal ALT: consider treating at 9.0 kPa

NAFLD
- Cutoff to know:
  - 10.3 kPa suggests cirrhosis
- Consider performing CAP assessment
- Consider XL probe for obese patients

Transient elastography: what the clinician needs to know

1. What is the underlying disease?
2. Other evidence of advanced liver disease? (e.g., perform a physical exam and check serological tests for fibrosis)
3. What can affect the test?
   a. Is the patient fasting?
   b. What is the body mass index?
   c. What is the burden of inflammation? (e.g., check ALT)
   d. Is the patient actively drinking alcohol?
   e. Is there evidence of cholestasis?



Alcoholic liver disease
- Must also know: drinking status
- Cutoffs to know:
  - 22.7 kPa suggests cirrhosis if drinking
  - 12.5 kPa suggests cirrhosis if abstinent

Biliary liver disease
- Must also know: alkaline phosphatases
- Cutoff to know:
  - 17.9 kPa suggests cirrhosis

Portal hypertension in cirrhotic patients
- Cutoffs to know:
  - 20.0 kPa suggests HVPG ≥ 10
  - 50.7 kPa suggests high risk of variceal bleeding

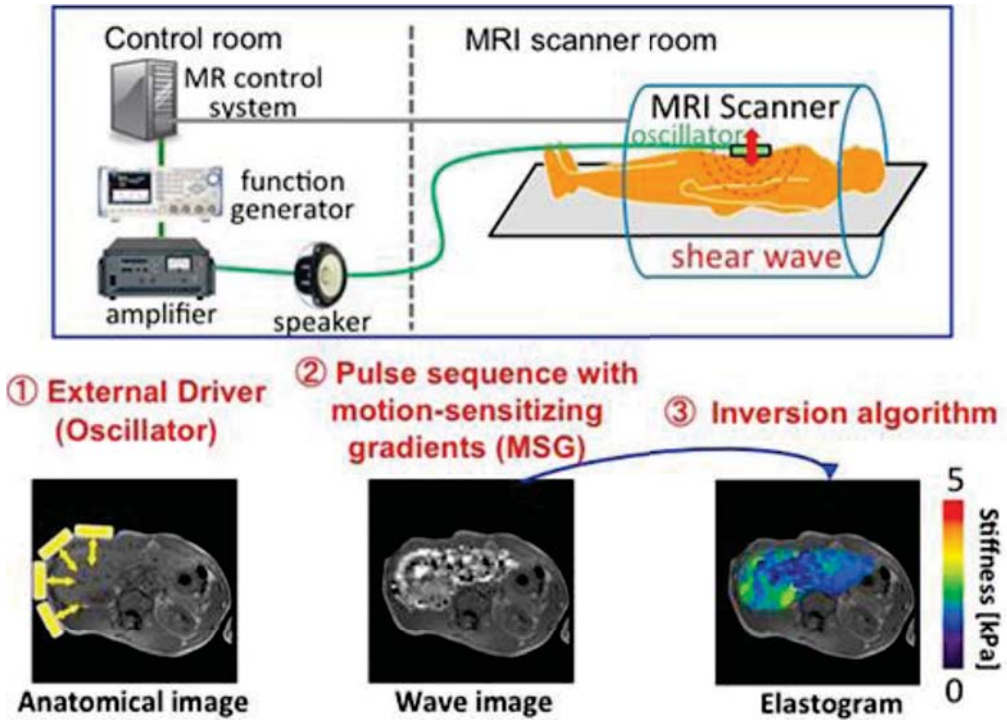
健保 F3 之定義為：Fibroscan ≧ 9.5Kpa

Tapper EB et al., CGH 2015;13:27–36

Acoustic Radiation Force Impulse (ARFI)

健保 F3 之定義為：ARFI ≧ 1.81m/sec

MR Elastography



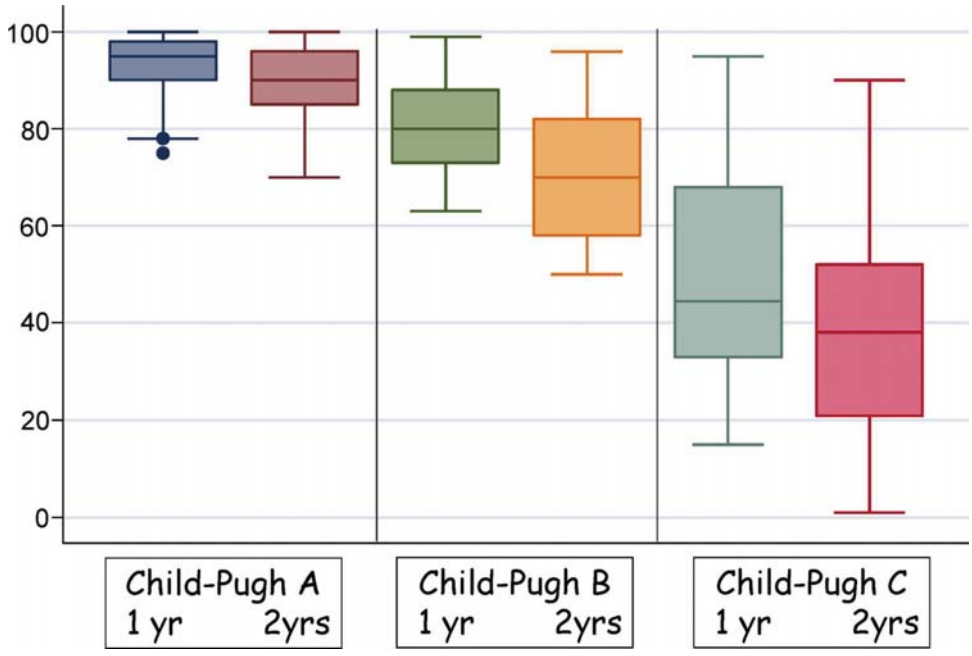
評估肝硬化的嚴重度

Child-Pugh classification

	1	2	3
Albumin (g/dL)	＞3.5	2.8-3.5	<2.8
Bilirubin (mg/dL)	<2	2-3	>3
Prothrombin time (seconds increased)	1-3	4-6	>6
Ascites	Nil	Mild	≧moderate
Encephalopathy	Nil	mild	≧moderate

A: 5-6, B: 7-9, C: 10-15

Two-year survival rates of LC



D'Amico G et al., JH 2006;44:217–231

MELD score

3.8 X $\log_e$(膽紅素[mg/dL])
✚ 11.2 X $\log_e$(INR,凝血酶原時間)
✚ 9.6 X $\log_e$(creatinine [mg/dL],肌酸酐,腎功能)
✚ 6.4 X (肝硬化的原因: 0 酒精性, 1 其他)

MELD Formula

The MELD score is calculated using the following formula:

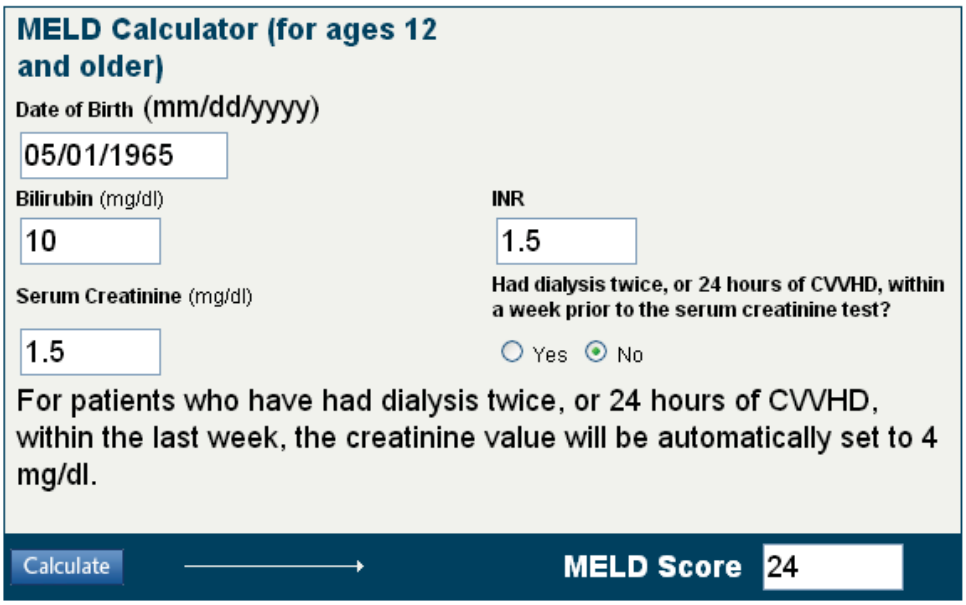
$$\text{MELD Score} = 0.957 \times \text{Log}_e(\text{creatinine mg/dL}) + 0.378 \times \text{Log}_e(\text{bilirubin mg/dL}) + 1.120 \times \text{Log}_e(\text{INR}) + 0.643^{*}$$

Multiply the score by 10 and round to the nearest whole number

HEPATOLOGY 2001;33:464-470

MELD Calculator

http://optn.transplant.hrsa.gov/resources/professionalResources.asp?index=9



Baveno IV staging of liver cirrhosis

Compensated

Stage 1: no varices, no ascites

Stage 2: varices, no ascites

Decompensated

Stage 3: ascites ± varices

Stage 4: variceal bleeding ± ascites

Franchis R. JH 2005;43:167–176

In Baveno IV, a session was devoted to predictive models in portal hypertension, during which classification stages of cirrhosis were proposed. Prospective validation of this classification is under way.
JH 2010;53:762–768

治療病毒性肝硬化的藥物

治療B型肝炎的藥物

- ✓ 長效型干擾素
- ✓ 干安能 (lamivudine, Zeffix)
- ✓ 干適能 (adefovir, Hepsera)
- ✓ 貝樂克 (entecavir, Baraclude)
- ✓ 喜必福 (telbivudine, Sebivo)
- ✓ 惠立妥 (tenofovir, Viread)
- ✓ 韋立得 (tenofovir alafenamide, Vemlidy)

治療C型肝炎的藥物

- 干擾素
- 口服抗病毒藥物
  (direct antirviral agent, DAA)

在台灣已經上市的C型肝炎口服藥

- 坦克干(Daklinza)+速威干(Sunvepra)
- 維建樂(Viekirax) +易奇瑞(Exviera)
- 夏奉寧(Harvoni)
- 索華迪(Sovaldi)
- 賀肝樂(Zepatier)
- 艾百樂(Maviret)

健保對於病毒性肝硬化的治療規定

全民健康保險加強慢性B、C型肝炎治療試辦計畫

HBsAg(+)

↓

肝硬化病患

(1) HBsAg (+)且血清HBV DNA≧2,000IU/mL

+

(2) 肝組織切片（Metavir F4或Ishak F5以上)

or

超音波/電腦斷層/核磁共振診斷為肝硬化併食道或胃靜脈曲張

or

超音波/電腦斷層/核磁共振診斷為肝硬化併脾腫大

可長期使用

Zeffix(100mg) / Sebivo(600mg)

Baraclude(0.5mg) / Viread (300mg)

治療C型肝炎的口服抗病毒藥物

限使用於Anti-HCV 陽性超過六個月、HCV RNA 為陽性之成人病患，且需符合下列條件：

經由肝組織切片或肝臟纖維化掃描或FIB-4 證實，等同METAVIR system 纖維化大於或等於F3；或超音波診斷為肝硬化併食道或胃靜脈曲張，或超音波診斷為肝硬化併脾腫大

-> 2019起，解除纖維化的設限條件

不同基因型的規定略有差異，所使用的藥物也不同。

治療病毒性肝硬化能改善Child-Pugh scores MELD scores

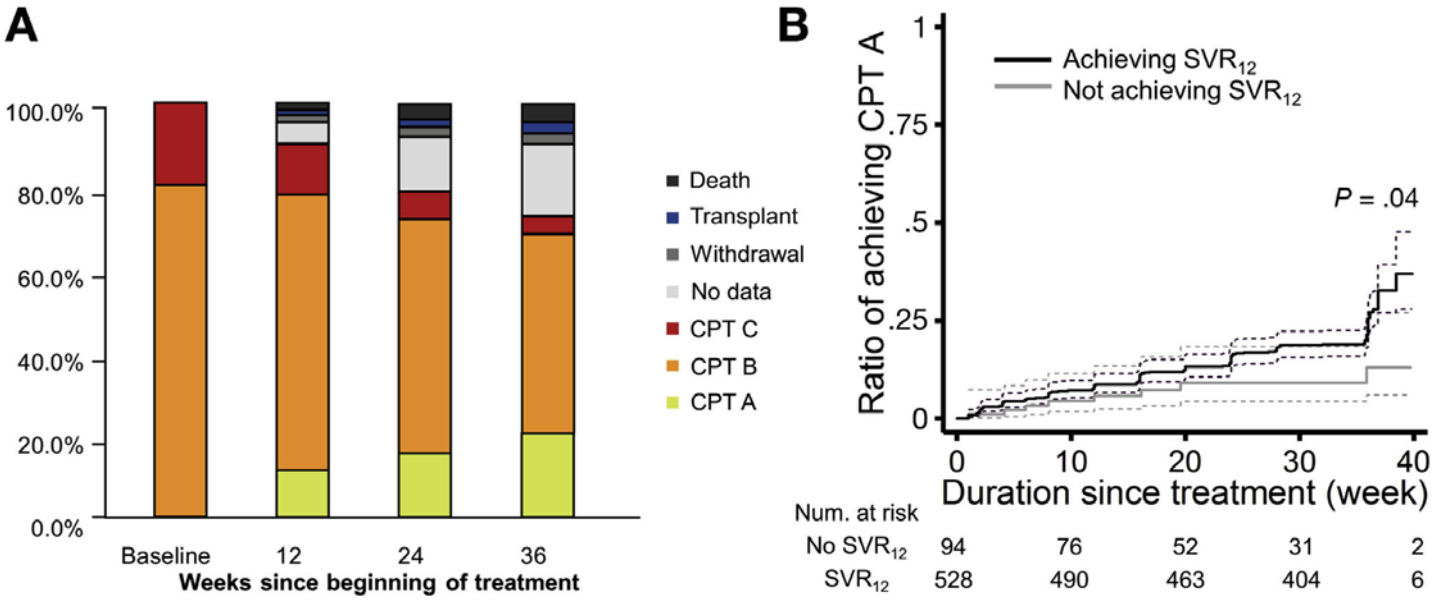
ETV-048: Improvement in MELD/CTP Scores

Parameter	Wk 24		Wk 48	
	ETV	ADV	ETV	ADV
Mean **MELD** score change from BL (SE)	-2.0 (0.45)	-0.9 (0.46)	**-2.6** (0.62)	-1.7 (0.50)
CTP score improvement or no worsening,* n/N (%)	66/100 (66)	65/91 (71)	61/100 (61)	61/91 (67)
CTP score ≥ 2 point reduction,* n/N (%)	32/100 (32)	22/91 (24)	35/100 (**35**)	25/91 (27)
CTP class improvement,† n/N (%)	25/93 (27)	22/81 (27)	35/93 (38)	29/81 (36)

*Noncompleter = failure.
†CTP class C/B to A only.

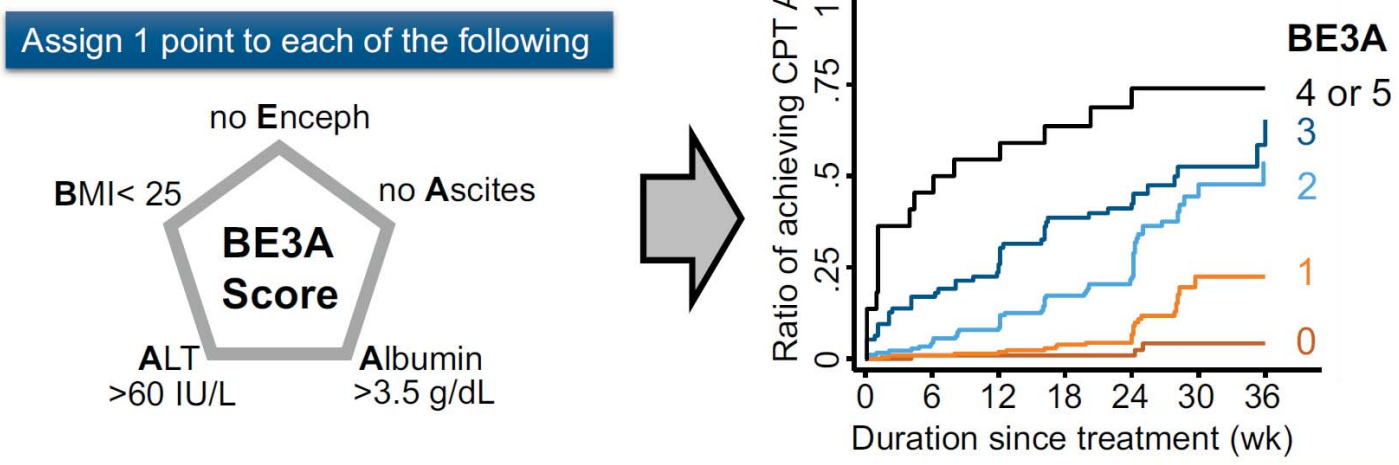
Liaw YF, et al. Hepatology. 2011;54:91-100.

DAA Improves Child Score in Decompensated Liver Cirrhosis



El-Sherif O et al, Gastroenterology 2018;154:2111–2121

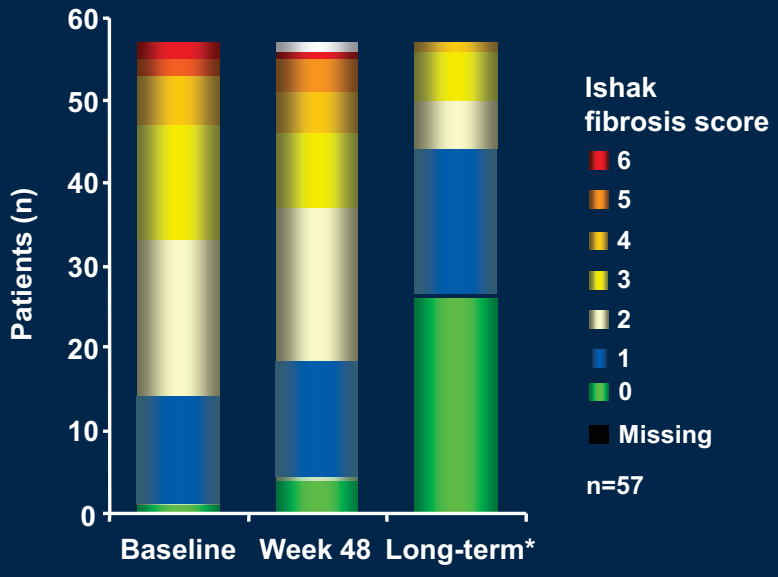
DAA Improves Child Score in Decompensated Liver Cirrhosis



El-Sherif O et al, Gastroenterology 2018;154:2111–2121

治療病毒性肝硬化
可以逆轉纖維化

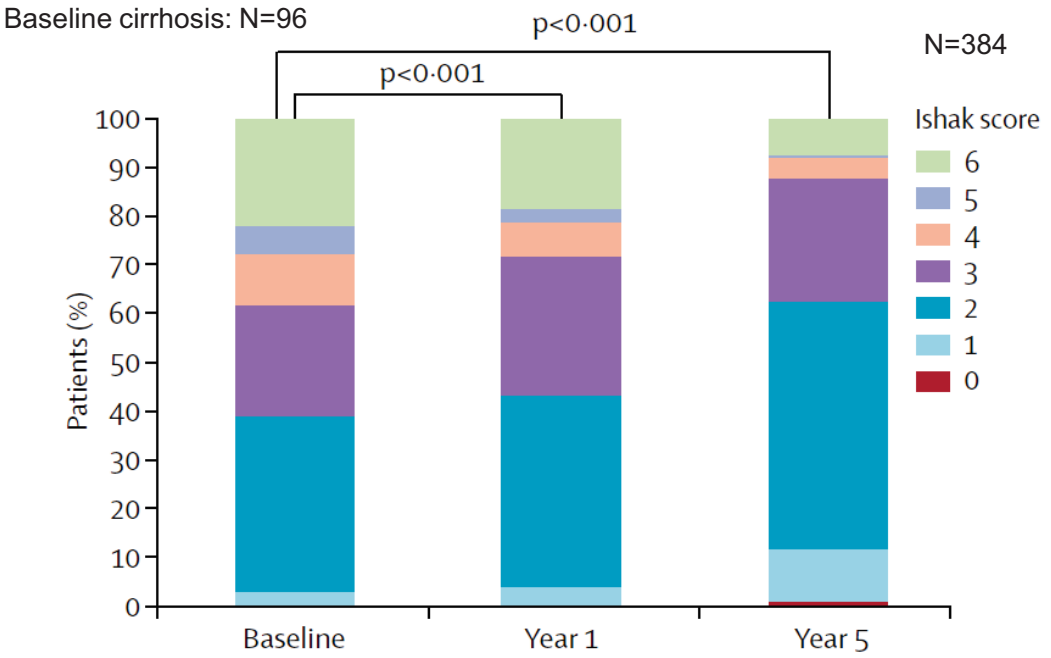
ETV Long term treatment Distribution of Ishak fibrosis scores at baseline, Year 1 and Years 3–7



* Median time of long-term biopsy: 280 weeks (range: 144–316 weeks).

Chang TT et al., HEPATOLOGY 2010;52:886-893

Improvement of hepatic fibrosis after 5-year TDF



Marcellin P et al., Lancet 2013; 381: 468–75

Comparison of Liver Fibrosis Stage in patients of CHC reaching SVR

	Fibrosis stage[a]				
	Post-treatment				
Pretreatment	F0	F1	F2	F3	F4
F0	1	2	0	0	0
F1	14	16	7	0	0
F2	7	23	12	2	0
F3	0	5	12	7	4
F4	0	1	2	6	5
Total (n/N) (%) (95% CI)					

Fibrosis improved in 56%, stable in 32%, Deteriorated in 12%

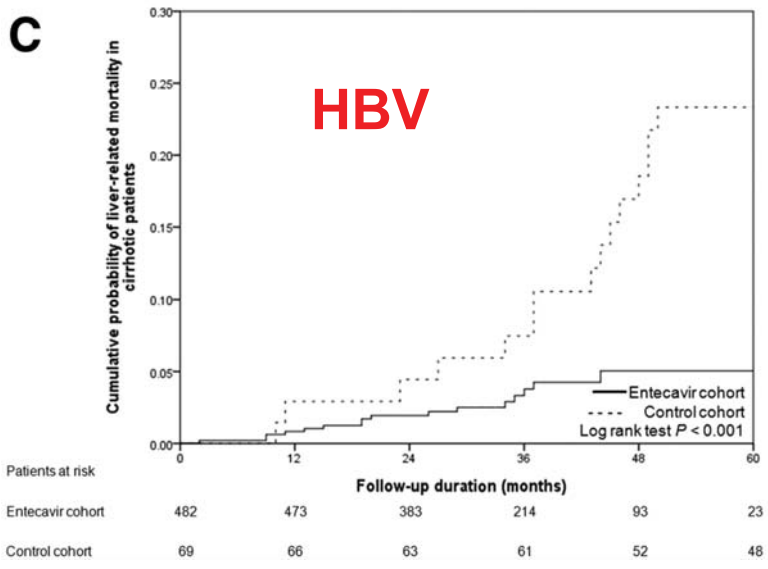
Regression of cirrhosis in 9/14 patients

Maylin S. et al., GASTROENTEROLOGY 2008;135:821–829

治療病毒性肝硬化
可以延長病人的存活

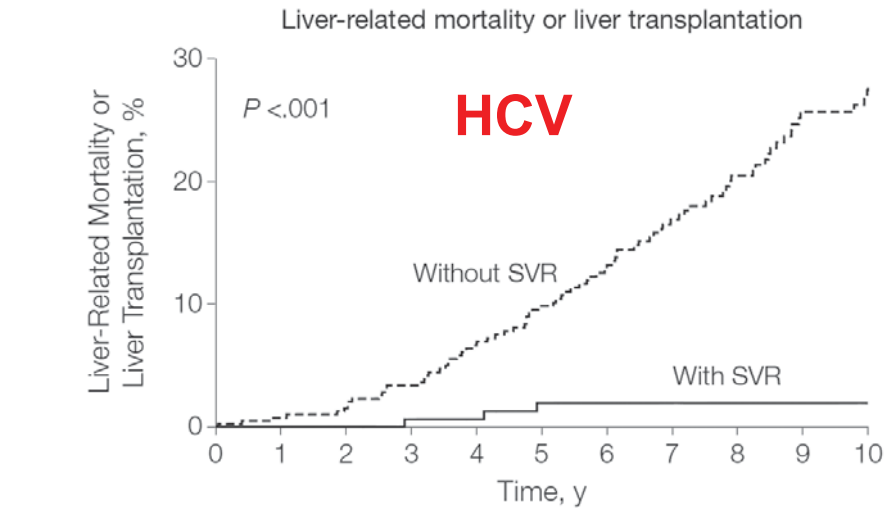
Cumulative probability of liver-related mortality in cirrhotic patients

Liver-related mortality: death related to cirrhosis complications and/or HCC



Wong GL et al. HEPATOLOGY 2013;58:1537-1547

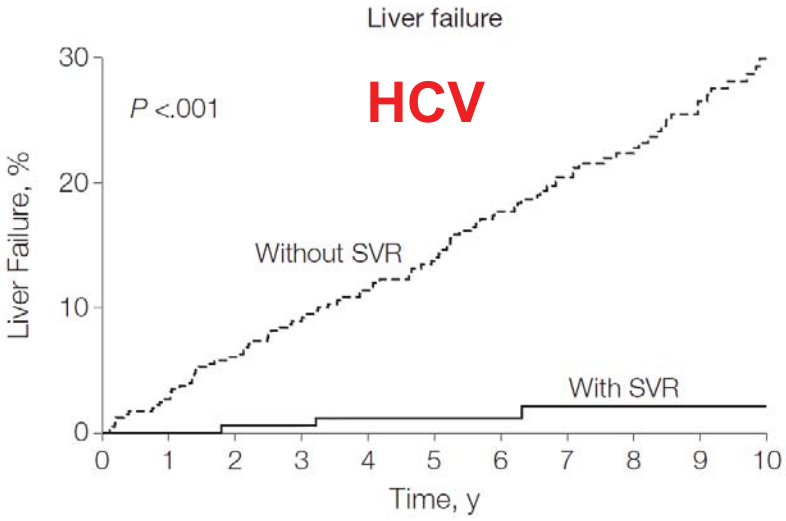
SVR and Liver-related mortality



No. at risk											
Without SVR	405	392	380	358	334	305	277	229	187	146	119
With SVR	192	181	168	162	155	144	125	88	56	40	28

Van der Meer AJ et al., JAMA 2012-;308:2584-2593

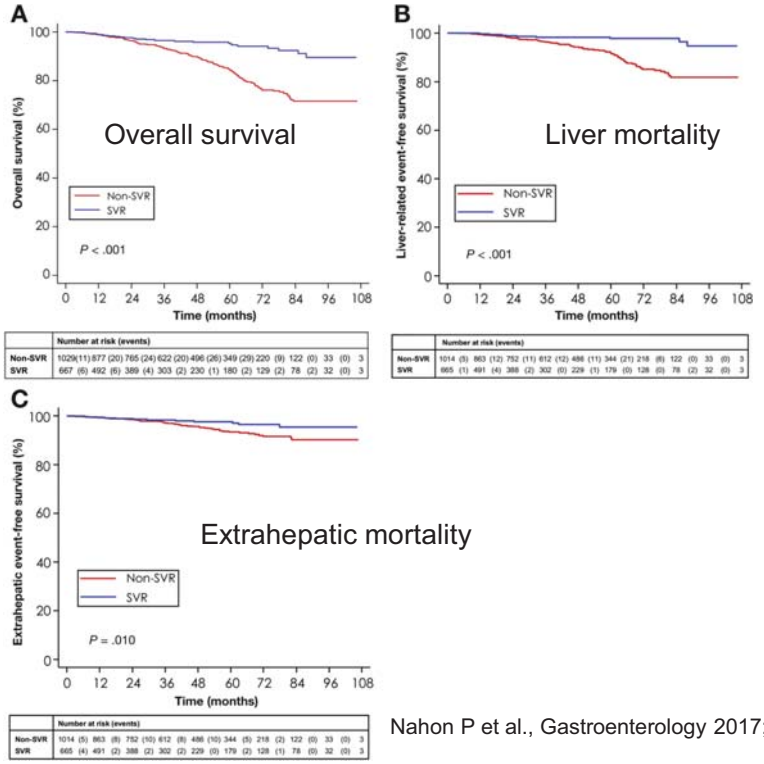
SVR and Liver failure



No. at risk											
Without SVR	405	384	361	337	314	288	259	216	184	143	113
With SVR	192	180	166	160	152	141	123	88	56	40	28

Van der Meer AJ et al., JAMA 2012-;308:2584-2593

SVR improves survival in HCV-LC

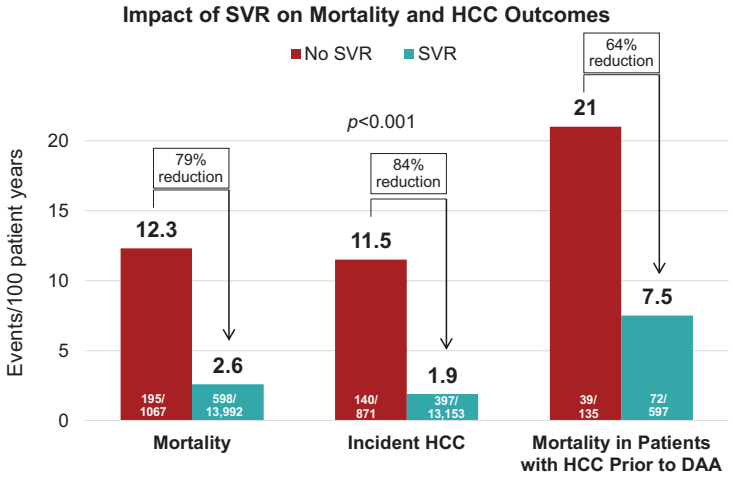


Nahon P et al., Gastroenterology 2017;152:142–156

Impact of SVR with DAAs On Mortality in Patients With Advanced Liver Disease

Veterans Affairs HCV Clinical Case Registry

All-cause mortality rates and incident HCC rates in 15,059 HCV-infected Veterans with advanced chronic liver disease (FIB-4 >3.25) from the HCV registry through Sept 2016.



Patients achieving SVR after DAA treatment had significantly lower all-cause mortality and lower incident HCC rates than those who did not achieve SVR.

Backus, et al. Hepatology. 2018

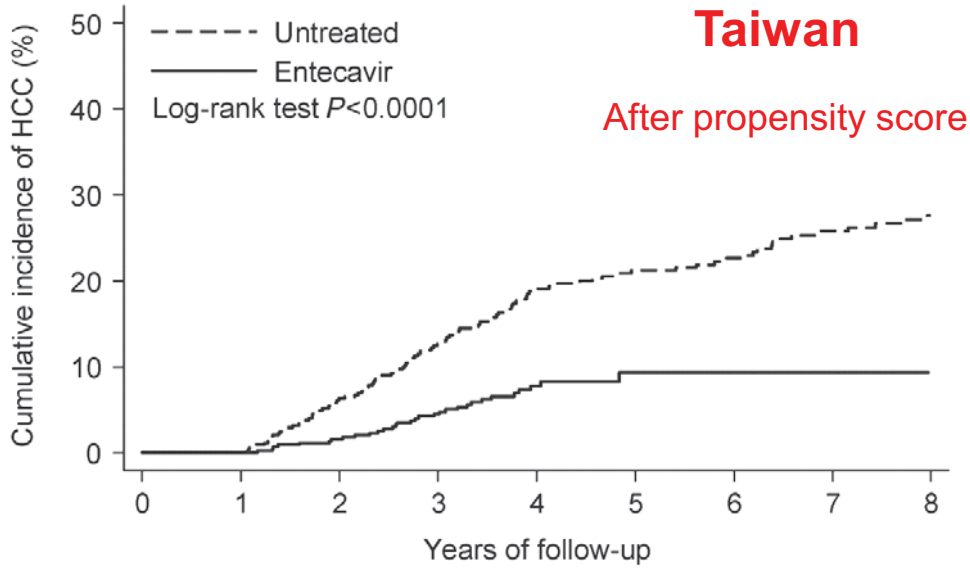
Benefit of DAA

LEVEL OF EVIDENCE	DAA BENEFITS
Low-Moderate Level 	**Cirrhosis:** Liver-related mortality All-cause mortality Transplant delisting MELD score reduction Fibrosis regression* **Mild liver disease:** All-cause mortality **Extra-hepatic manifestations:** Diabetes Cardiovascular disease QoL PRO Lymphoma

Ioannou GN & Feld JJ. Gastroenterology 2019

治療病毒性肝硬化
可以降低肝癌的發生率

Four-year ETV therapy reduces HCC



Taiwan

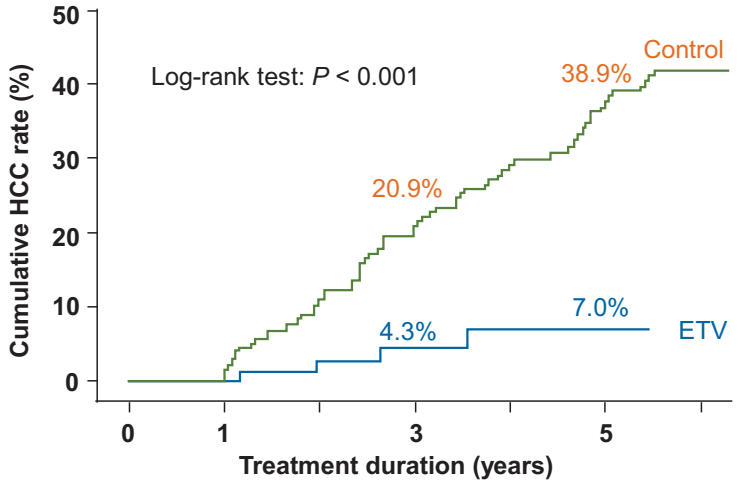
After propensity score

Number at risk									
Untreated	450	450	414	351	284	243	211	172	143
Entecavir	450	450	443	363	206	69	37	15	1

Su TH et al., Liver International 2016; 36: 1755–1764

Reduction in HCC incidence with ETV in cirrhotic patients

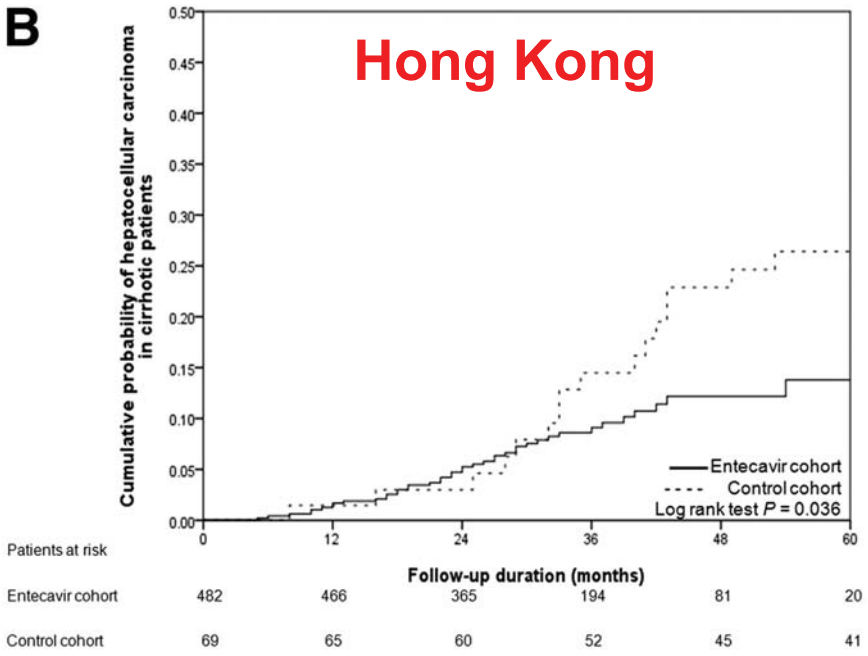
Japan



No. at risk						
ETV	79	79	72	53	35	17
Control	85	85	76	65	54	47

Hosaka T et al. Hepatology 2013;58:98-107

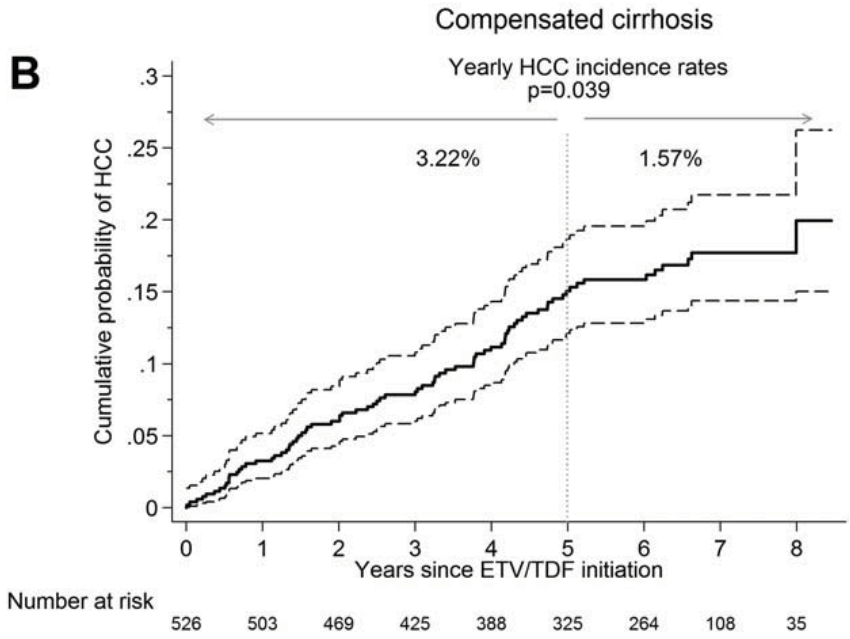
Cumulative probability of HCC in cirrhotic patients



Wong GL et al. HEPATOLOGY 2013;58:1537-1547

HCC risk decreases beyond year 5 of ETV/TDF therapy in Caucasian chronic hepatitis B patients

Europe

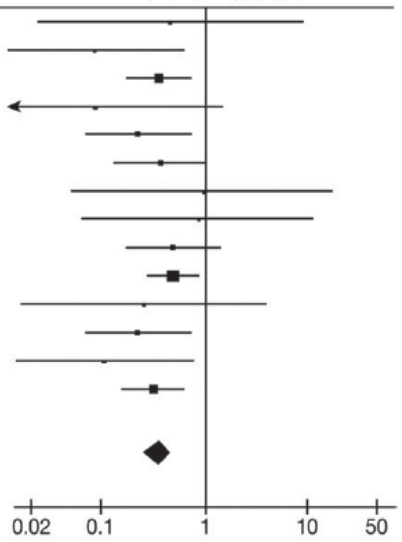


Papatheodoridis GV et al., HEPATOLOGY 2017;66:1444-1453

HCC in HCV-related liver cirrhosis (SVR vs no SVR)

Study or subgroup	SVR Events	SVR Total	NSVR Events	NSVR Total	Weight	Risk ratio M-H, random, 95% CI
Azzaroli 2004	0	21	2	50	1.0%	0.46 [0.02, 9.27]
Braks 2007	1	37	24	76	2.3%	0.09 [0.01, 0.61]
Bruno 2007 (1)	7	124	122	759	16.0%	0.35 [0.17, 0.73]
Floreani 2008 (2)	0	40	5	38	1.1%	0.09 [0.00, 1.51]
Hasegawa 2007 (3)	3	48	16	57	6.3%	0.22 [0.07, 0.72]
Hung 2006	5	73	11	59	8.7%	0.37 [0.14, 1.00]
Nishiguchi 1995	0	7	2	38	1.0%	0.97 [0.05, 18.43]
Okanoue 1999	0	2	7	38	1.3%	0.87 [0.06, 11.79]
Shioda 1999	4	204	18	448	7.6%	0.49 [0.17, 1.42]
Shiratori 2005	11	64	73	207	26.9%	0.49 [0.28, 0.86]
Tanaka 1998	0	8	10	47	1.2%	0.25 [0.02, 3.96]
Veldt 2008	3	142	32	337	6.4%	0.22 [0.07, 0.71]
Yoshida 1999 (4)	1	53	30	168	2.2%	0.11 [0.01, 0.76]
Yu 2006	9	85	27	80	18.3%	0.31 [0.16, 0.63]
Total (95% CI)		**908**		**2402**		**0.35 [0.26, 0.46]**

Risk ratio M-H, random, 95% CI

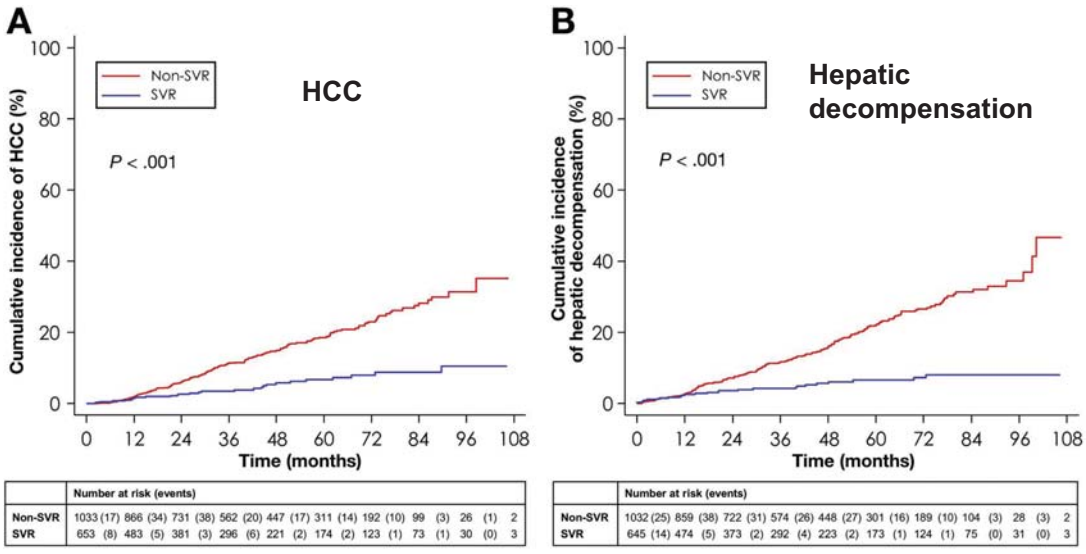


Heterogeneity: $Chi^2 = 8.67$, df = 13 ($P = .80$)
Test for overall effect: Z = 7.06 ($P < .00001$)

(1) NSVR patients were retreated with SVR in 38 leaving 759 patients with NSVR
(2) 9 patients with NSVR lost to follow-up
(3) 18 patients continued to receive treatment and could not be evaluated for response
(4) 9 patients with NSVR lost to follow-up

Singal et al. CGH 2010;8:192–199

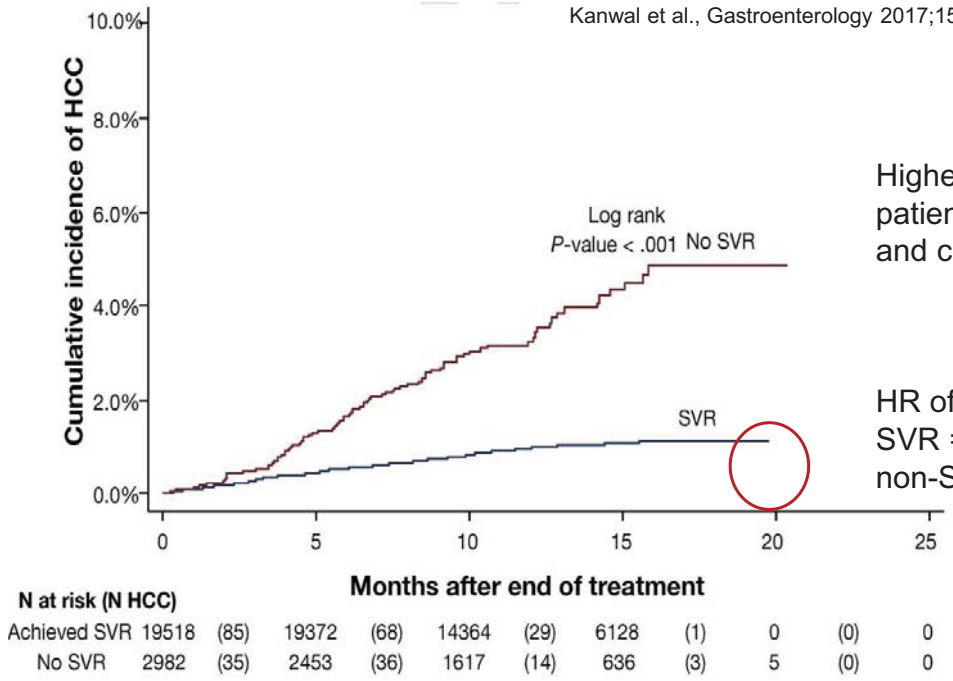
SVR decreases incidence of HCC and hepatic decompensation in HCV-LC



Nahon P et al., Gastroenterology 2017;152:142–156

The incidence of HCC is Reduced in HCV patients After SVR by DAA

Kanwal et al., Gastroenterology 2017;153:996–1005 Veterans Affairs

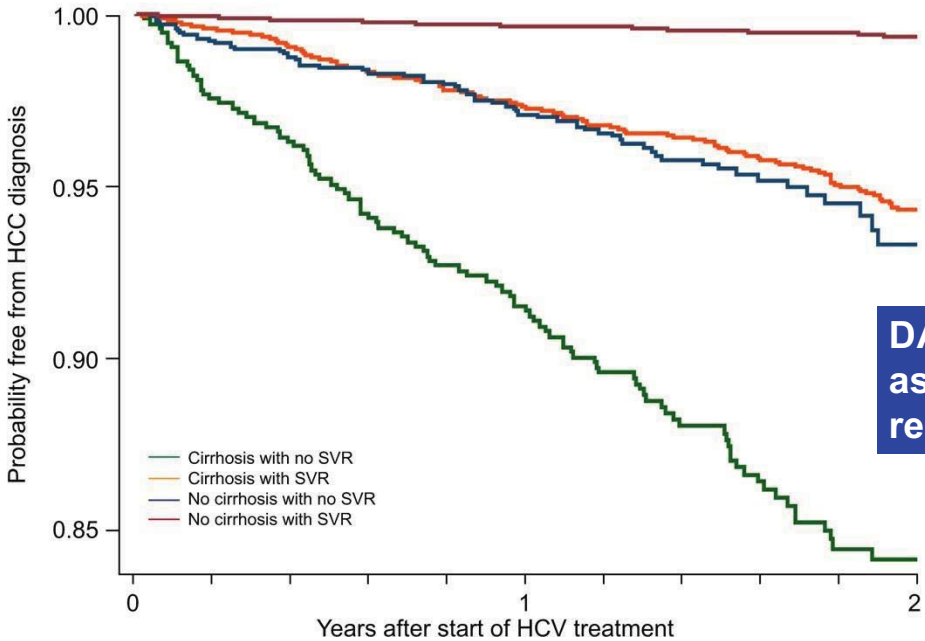


N at risk (N HCC)										
Achieved SVR	19518	(85)	19372	(68)	14364	(29)	6128	(1)	0	(0)	0
No SVR	2982	(35)	2453	(36)	1617	(14)	636	(3)	5	(0)	0

Higher HCC rate in older patients, African Americans, and cirrhotics

HR of developing HCC after SVR = 0.90, versus 3.45 in non-SVR

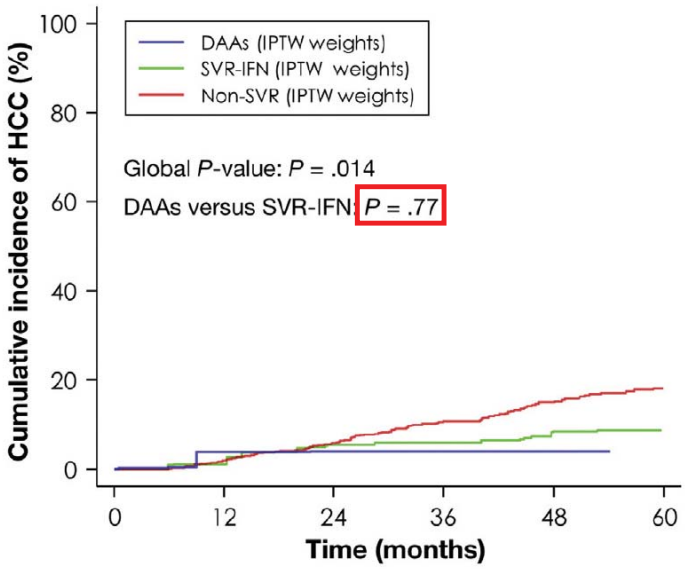
Kaplan-Meier curves of survival free of HCC by cirrhosis and SVR status after DAA-only antiviral treatment:
SVR is associated with a reduction in HCC risk both among patients with cirrhosis and those without cirrhosis.



DAA-induced SVR is associated with a 71% reduction in HCC risk.

Ioannou GN et al, JH 2018;68: 25–32

Incidence of HCC of DAA treatment using IPTCW



Groups	Number at risk (events)					
DAAs (IPTW weights)	956	698	384	92	53	43
SVR-IFN (IPTW weights)	1076	1043	965	849	771	532
Non-SVR (IPTW weights)	1029	892	760	613	478	357

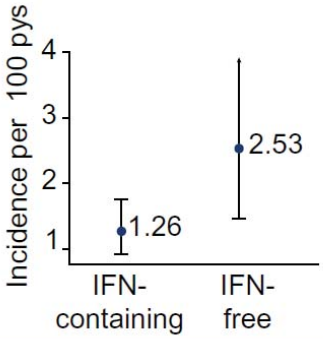
ANRS CO12 CirVir Group
compensated biopsy-proven HCV-associated cirrhosis recruited from 2006 through 2012 at 35 centers in France

IPTCW: inverse probability of treatment and censoring

Nahon et al, Gastroenterology 2018;155:1436–1450

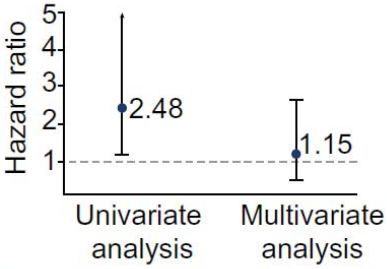
The risk of hepatocellular carcinoma in cirrhotic patients with hepatitis and sustained viral response: role of the treatment regimen

Crude incidence of HCC occurrence by regimen



Characteristics	IFN-containing patients	IFN-free patients
Mean age	48.1 yr	52.1 yr
% decompensated	9.5	30.4
% treatment experienced	27.6	52.2
% thrombo-cytopenic	22.1	39.3

Association between IFN-free *vs.* IFN-containing therapy and HCC occurrence



1. The crude incidence of HCC occurrence for IFN-free patients is twice as high as for IFN-containing patients

→

2. But IFN-free patients are more likely to be thrombocytopenic, treatment experienced, decompensated, and older

→

3. Once these differences are accounted for, the association between IFN-free therapy and HCC occurrence disappears

Innes H et al, JH 2018;68:646–654

Hepatocellular carcinoma risk following direct-acting antiviral HCV therapy: A systematic review, meta-analyses, and meta-regression

Waziry et al., JH 2017;67:1204–1212

Reem Waziry[1,*], Behzad Hajarizadeh[1], Jason Grebely[1], Janaki Amin[2], Matthew Law[1], Mark Danta[3], Jacob George[4], Gregory J. Dore[1]

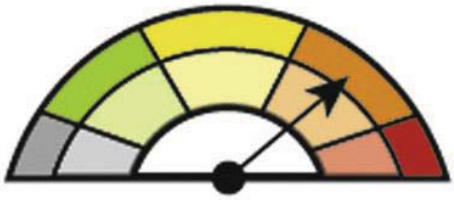
[1]*The Kirby Institute, UNSW Sydney, Sydney, Australia;* [2]*Faculty of Medicine and Health Sciences, Macquarie University, Sydney, Australia;* [3]*St Vincent's Clinical School, UNSW Sydney, Australia;* [4]*Storr Liver Centre, Westmead Millennium Institute and Westmead Hospital, University of Sydney, Sydney, Australia*

Table 3. Meta-regression analysis of factors associated with occurrence of hepatocellular carcinoma following HCV cure (Observations = 26).

Variable	Univariate analysis			Multivariate analysis†		
	RR	95% CI	*p* value	aRR	95% CI	*p* value
Treatment						
IFN	1.00	–	–	1.00	–	–
DAA	2.77	1.46–5.25	<0.01	0.68	0.18–2.55	0.56
Average follow-up, years	0.88	0.80–0.97	0.01	0.75	0.56–0.99	0.04
Average age	1.11	1.03–1.18	<0.01	1.06	0.99–1.14	0.12
Genotype 1	1.01	0.99–1.03	0.14	–	–	–

All numbers were rounded to two decimal places.
aRR, adjusted rate ratio; CI, confidence interval; DAA, direct-acting antiviral; IFN, interferon; RR, Rate Ratio.
† Five studies were excluded from the adjusted analysis due to incomplete data on age.

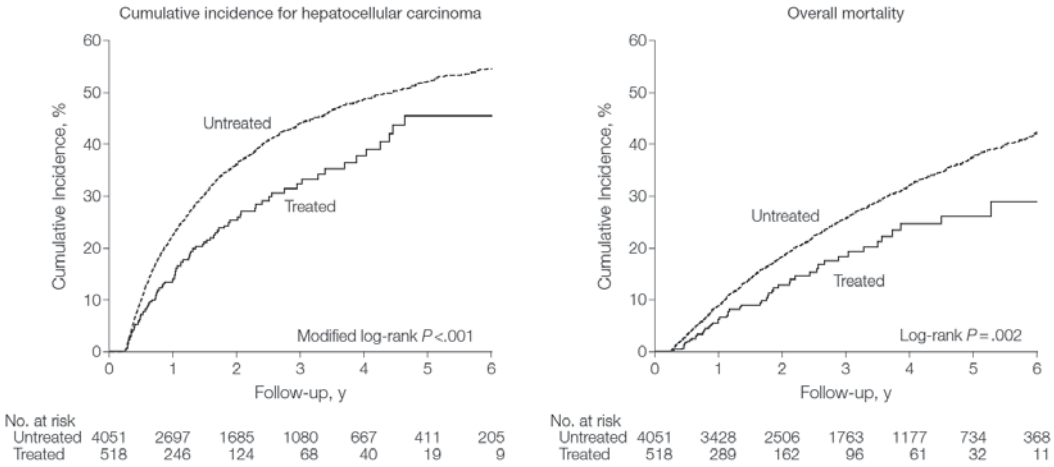
Benefit of DAA

LEVEL OF EVIDENCE	DAA BENEFITS
Moderate-High Level 	**Cirrhosis:** HCC incidence HVPG reduction **Extra-hepatic manifestations:** Cryoglobulinemia

Ioannou GN & Feld JJ. Gastroenterology 2019

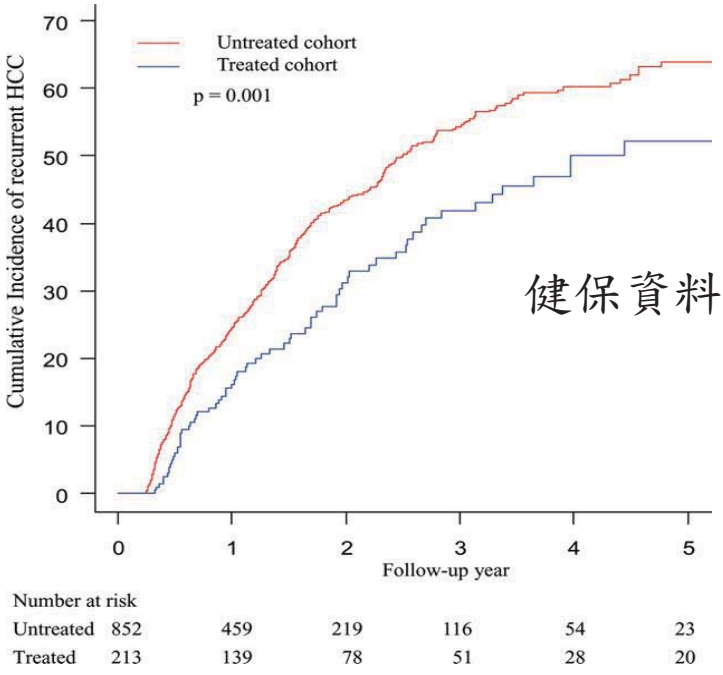
治療病毒性肝硬化
可以降低肝癌的復發率

使用核苷(酸)類似物可以降低術後肝癌的復發



Wu CY et al. JAMA 2012;308(18):1906-1913

Recurrence of resected HCC in chronic hepatitis C



健保資料庫

Number at risk						
Untreated	852	459	219	116	54	23
Treated	213	139	78	51	28	20

Yao-Chun Hsu et al. HEPATOLOGY 2013;58:150-157

Research Article JH 2016;65:727–733

EASL | JOURNAL OF HEPATOLOGY

Early occurrence and recurrence of hepatocellular carcinoma in HCV-related cirrhosis treated with direct-acting antivirals

Fabio Conti[1,†], Federica
Paolo Caraceni[3], F
Gabriel

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of Medical and Surgical Sciences (DIMEC),
e di Faenza, Italy

Research Article JH 20

EASL | JOURNAL OF HEPATOLOGY

Unexpected high rate of early tumor recurrence in patients with HCV-related HCC undergoing interferon-free therapy☆

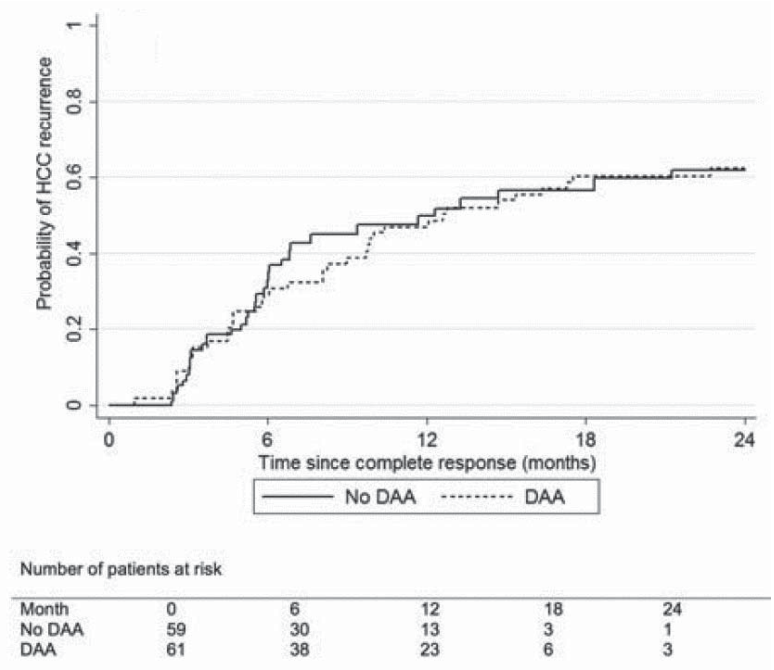
María Reig[1,†], Zoe Mari
Sabela Lens[2], Alba Díaz
Jos

[1]Barcelona Clinic Liver Cancer (BCLC) G
Biomédica en Red de Enfermedades He
Barcelona, CIBERehd, Barcelona, Spain;
Hepatología, Clínica Universidad de Na
Barcelona, IDIBAPS, Universi
University of Barcel

airaegui[4], Andrea Ribeiro[1],
ía Varela[7], Bruno Sangro[4],
uix[1,*,‡]

iversity of Barcelona, Centro de Investigación
r Unit, Hospital Clínic, IDIBAPS, University of
BERehd, IDIPHIM, Madrid, Spain; [4]Unidad de
nt of Pathology, BCLC Group, Hospital Clínic
Group, Hospital Clinic Barcelona,
le Asturias, Oviedo, Spain

DAAs Do Not Increase the Risk of HCC Recurrence After Local-Regional Therapy



- Retrospective study
- 149 LT candidates
- inverse probability of treatment weights (IPTW)

Huang AC et al, HEPATOLOGY 2018; 68:449-461

Hepatocellular carcinoma risk following direct-acting antiviral HCV therapy: A systematic review, meta-analyses, and meta-regression

Waziry et al., JH 2017;67:1204–1212

Reem Waziry[1,*], Behzad Hajarizadeh[1], Jason Grebely[1], Janaki Amin[2], Matthew Law[1], Mark Danta[3], Jacob George[4], Gregory J. Dore[1]

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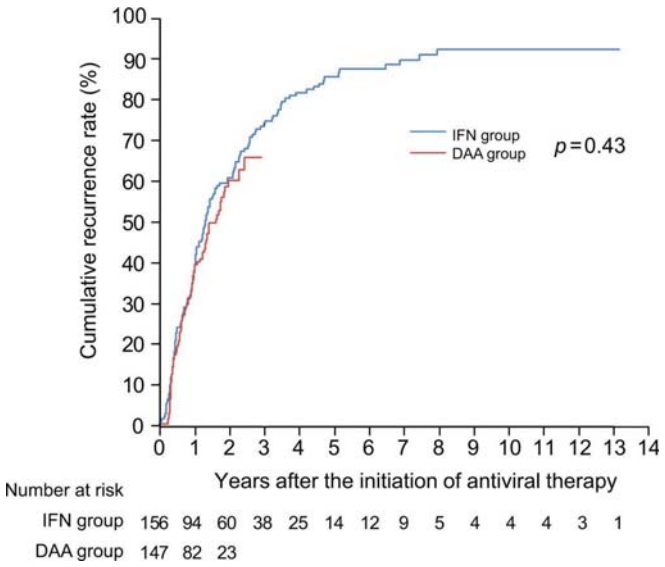
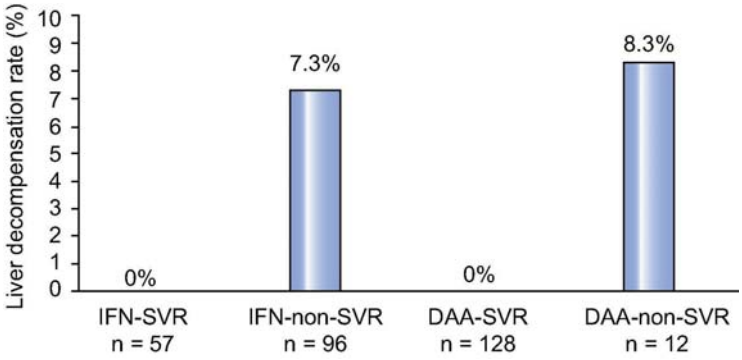
Table 4. Meta-regression analysis of factors associated with recurrence of hepatocellular carcinoma following HCV cure (Observations = 17).

Variable	Univariate analysis			Multivariate analysis		
	RR	95% CI	*p* value	aRR	95% CI	*p* value
Treatment						
IFN	1.00	–	–	1.00	–	–
DAA	1.36	0.49–3.76	0.53	0.62	0.11–3.45	0.56
Average follow-up, years	0.86	0.70–1.05	0.15	0.79	0.55–1.15	0.19
Average age	1.11	0.96–1.28	0.12	1.11	0.96–1.27	0.14
Genotype 1	1.01	0.97–1.05	0.49	–	–	–

All numbers were rounded to two decimal places.
aRR, adjusted rate ratio; CI, confidence interval; DAA, direct-acting antiviral; IFN, interferon; RR, Rate Ratio.

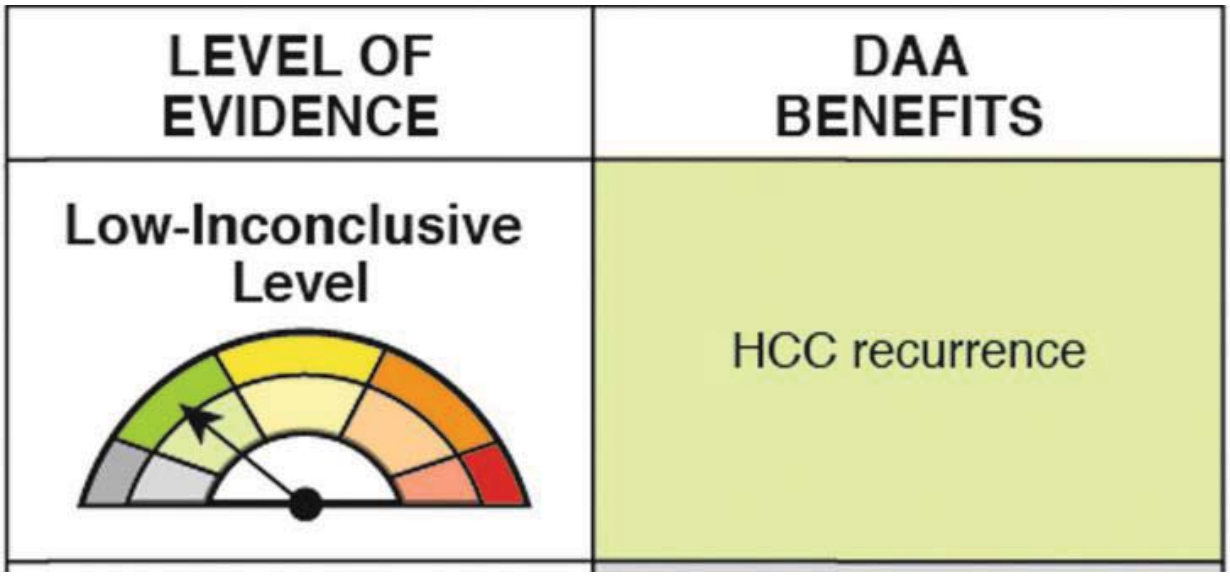
HCC recurrence rates did not differ between patients who received IFN-based therapy and DAA therapy





Nishibatake Kinoshita M et al, J Hepatol 2019;70:78-86

Benefit of DAA



Ioannou GN & Feld JJ. Gastroenterology 2019

除了治療時間較長外，用DAA治療 compensated liver cirrhosis = non-cirrhosis

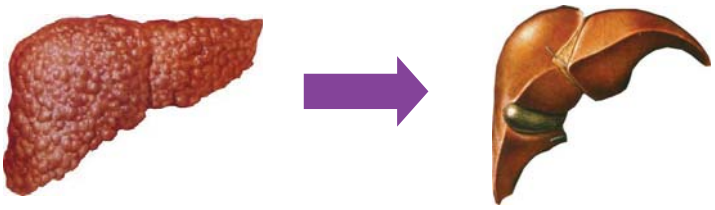
2018 AASLD guideline

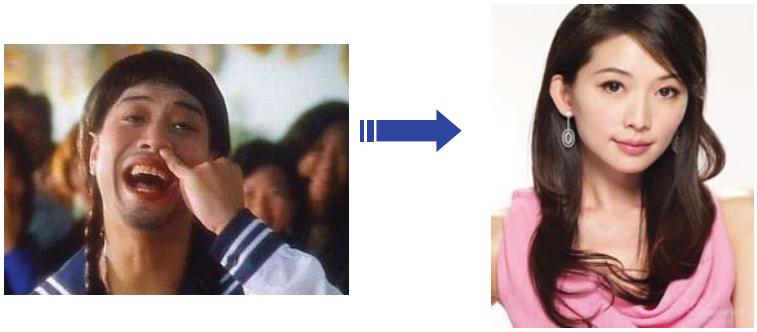
Regimens not recommended for:

Patients With Decompensated Cirrhosis (Moderate or Severe Hepatic Impairment; Child-Turcotte-Pugh Class B or C)

NOT RECOMMENDED	RATING
Paritaprevir-based regimens	III, B
Simeprevir-based regimens	III, B
Elbasvir/grazoprevir-based regimens	III, C
Glecaprevir/pibrentasvir	III, C
Sofosbuvir/velpatasvir/voxilaprevir	III, C

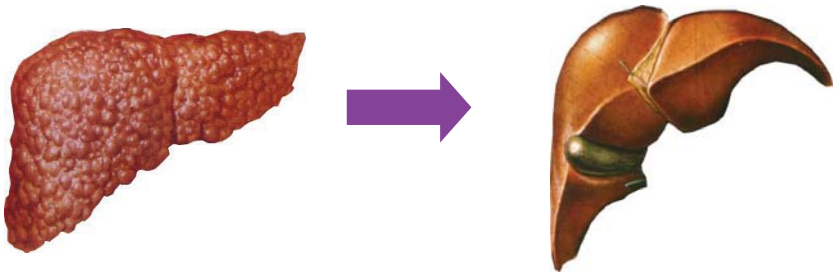
逆轉肝硬化 (in the past)





Mission impossible

逆轉肝硬化 (now)



肝硬化是
可逆的

結論

- 病毒性肝硬化是可逆的
- 治療病毒性肝硬化可延長病人的存活
- 治療病毒性肝硬化可降低肝癌的發生率
- 需要積極治療
- 與移植中心合作

您與您的病人，可以雙贏

Thanks