Actualités de la prise en charge des hépatites virales B

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Institut national
de la santé et de la recherche médicale

Introduction

Clinical Practice Guidelines

JOURNAL OF HEPATOLOGY

EASL 2017 Clinical Practice Guidelines on the management of hepatitis B virus infection☆

European Association for the Study of the Liver*

This practice guidance was approved by the American Association for the Study of Liver Diseases on December 4, 2017.

View this article online at wileyonlinelibrary.com.
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Stratégie de l'OMS pour l'élimination des hépatites B/C

Interventions	Indicateurs	2015	2020	2030
Vaccination HVB	Taux de couverture	84%	90%	90%
Prévention transmission verticale	Taux de couverture de la 1ère dose à la naissance	39%	50%	90%
sécurité transfusionnelle	Dépistage au CNST	97%	95%	100%
sécurité injection	Taux des injections à risque	5%	0%	0%
Réduction des risques GR	Nb de seringue pour les UD/an	27	200	300
améliorer le Dic	% HVB diagnostiquées % HCV diagnostiquées	9% 20%	30% 30%	90% 90%
Traitement	HVB dic Tx HCV dic Tx	8% 7%	– –	80% 80%

WHO Global Hepatitis Report 2017. Available at: http://apps.who.int/iris/bitstream/handle/10665/255016/9789241565455-eng.pdf?sequence=1 (accessed November 2018)

Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013

Aparna Schweitzer, Johannes Horn, Rafael T Mikolajczyk, Gérard Krause, Jördis J Ott

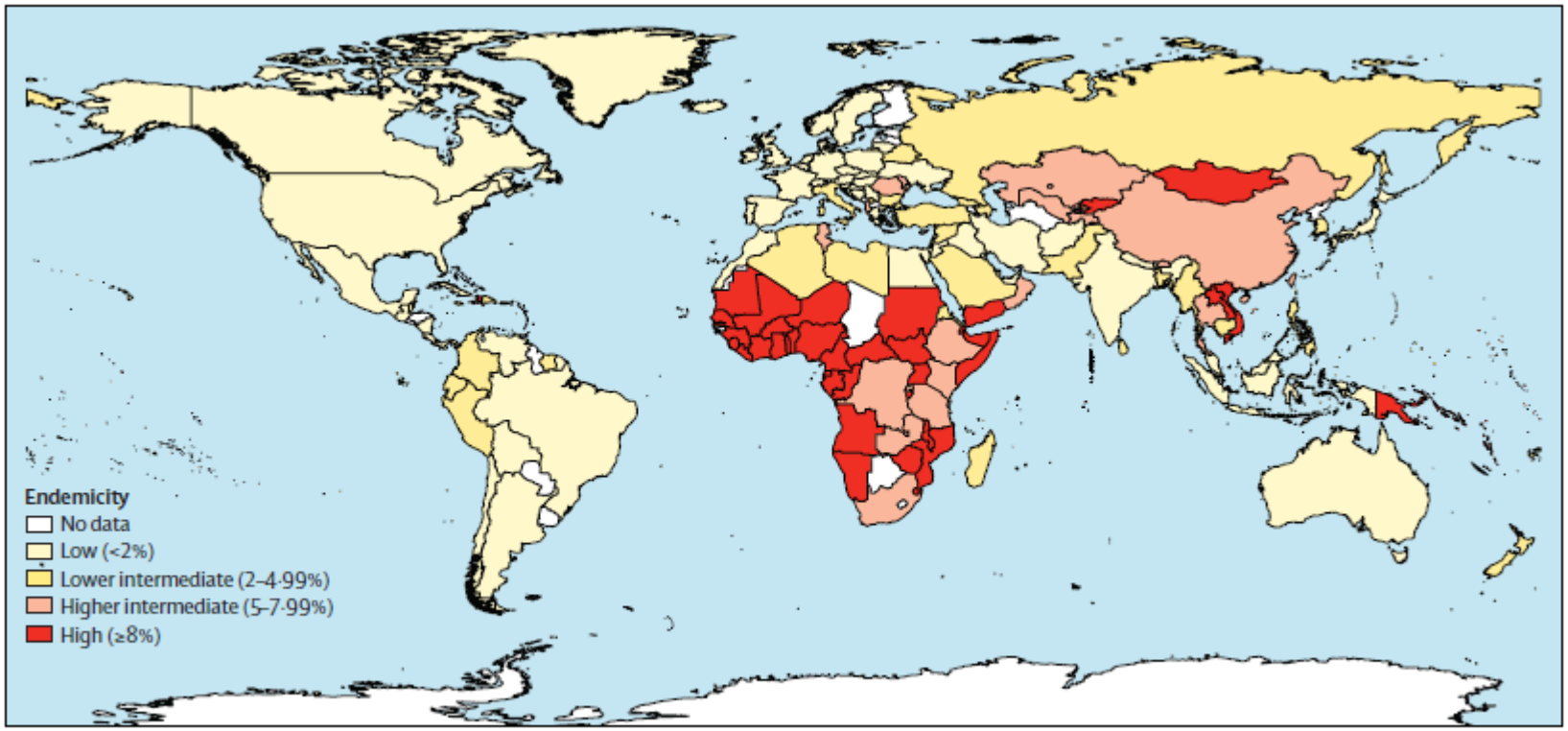


Figure 2: Global HBsAg endemicity (1957–2013)

www.thelancet.com Published online July 28, 2015 http://dx.doi.org/10.1016/S0140-6736(15)61412-X

Prévalence de l'antigène HBs dans la population générale

South-East Asia Region	2.0	1.5	4.0	39	29	77
Western Pacific Region	6.2	5.1	7.6	115	93	140
Total	**3.5**	**2.7**	**5.0**	**257**	**199**	**368**

Source: WHO, work conducted by the London School of Hygiene & Tropical Medicine (LSHTM). *See* Annex 2.

STATUS OF HEPATITIS C

INCIDENCE OF HCV INFECTION: TRANSMISSION PERSISTS

Several studies suggest that the incidence of HCV infection has decreased since the second half of the 20th century. First, most countries have age-specific prevalence of serological evidence of past or present infection, suggesting lower incidence in recent years *(34–37)*. Second, countries that conduct surveillance for acute hepatitis C reported decreases in the rates *(38)*. Third, countries that conducted more than one biomarker survey, such as Egypt, reported an evolution over time that suggests a decrease in incidence *(39)*. Fourth, injection safety improved, which reduced the incidence of injection-associated HCV infection *(40)*. However, estimates obtained from modelling suggest that worldwide, in 2015, there were still 1.75 million new HCV infections (global incidence rate: 23.7 per 100 000).

Unsafe health-care practices (including unsafe health-care injections) and injection drug use remain the leading modes of transmission. Areas with high rates of infection are located in the Eastern Mediterranean Region (62.5 per 100 000) and the European Region (61.8 per 100 000). In the Eastern Mediterranean Region, the most common cause of transmission of infection is unsafe health-care injections *(41, 42)*. In the European Region, injection drug use accounts for a substantial proportion of new infections (Table 3) *(43)*.

Even in areas of the world where the incidence was low in 2015, an increase in transmission may occur at any time and through various modes of transmission. In the United States of America, for instance, after many years of decrease, the incidence of HCV infection doubled between 2010 and 2014 *(44)*. The number of reported cases of acute hepatitis C among persons reporting

[a] Modelled estimate: 6.6 million, rounded. The WHO Regional Office for the Americas has worked with its Member States to generate estimates through country consultations and modelling. These national estimates were consolidated in 2016 into a regional estimate of 2.8 million people living with chronic HBV infection. The difference between these estimates is consistent with the different methods used. In addition, low-prevalence settings may lead to lower precision and greater uncertainty. WHO headquarters and regional offices will continue to engage in comparative modelling to further understand the source of these differences. Such analyses should allow more precise consensus estimates in the future. *See* Annex 2, and *(123)*.

La transmission du VHB est proportionnelle au taux d'ADN

Transmission horizontale

Personne infectée → Receveur

- Enfant à enfant
- Seringues
- Sexuelle
- Personnel soignant
- Transfusion
- Hémodialyse

Pas de facteur clair dans 20% des cas

Transmission verticale

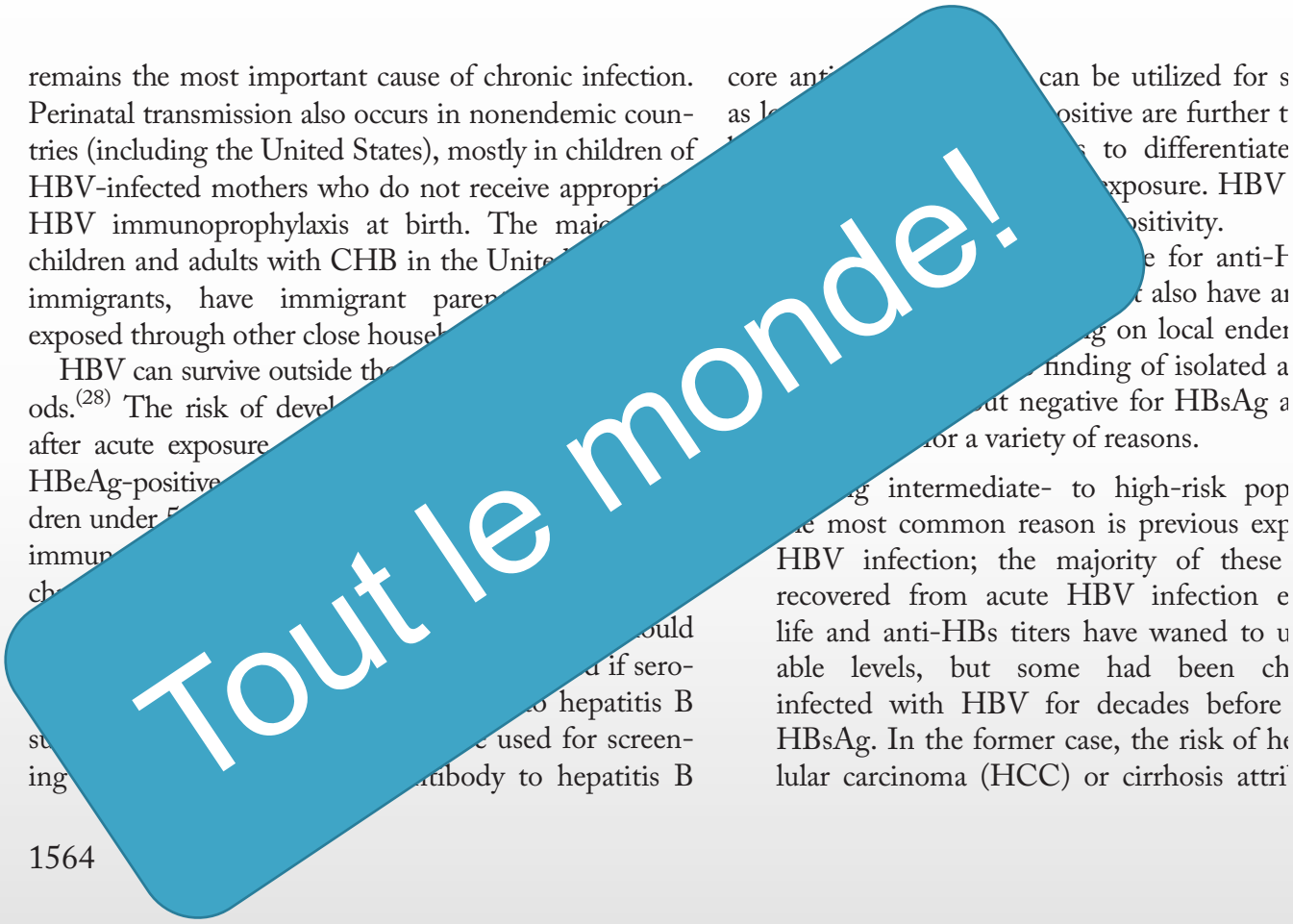
Mère
> 10^{6-8} UI/mL

↓ *Perinatal*

Enfant

CDC. Hepatitis B Information for the Public. Fact sheet. http://www.cdc.gov/hepatitis/B/PatientEduB.htm.
Lee WM. N Engl J Med. 1997;337:1733-1745. Lavanchy D. J Viral Hepat. 2004;11:97-107.

Qui dépister?



remains the most important cause of chronic infection. Perinatal transmission also occurs in nonendemic countries (including the United States), mostly in children of HBV-infected mothers who do not receive appropri HBV immunoprophylaxis at birth. The mai children and adults with CHB in the Unite immigrants, have immigrant paren exposed through other close housel

HBV can survive outside th ods.[28] The risk of devel after acute exposure HBeAg-positive dren under 5 immun ch

ould d if sero- o hepatitis B e used for screen- ntibody to hepatitis B

core ant can be utilized for s as l ositive are further t to differentiate xposure. HBV ositivity. e for anti-H t also have a g on local ende finding of isolated a ut negative for HBsAg a or a variety of reasons.

g intermediate- to high-risk pop e most common reason is previous exp HBV infection; the majority of these recovered from acute HBV infection e life and anti-HBs titers have waned to u able levels, but some had been ch infected with HBV for decades before HBsAg. In the former case, the risk of he lular carcinoma (HCC) or cirrhosis attri

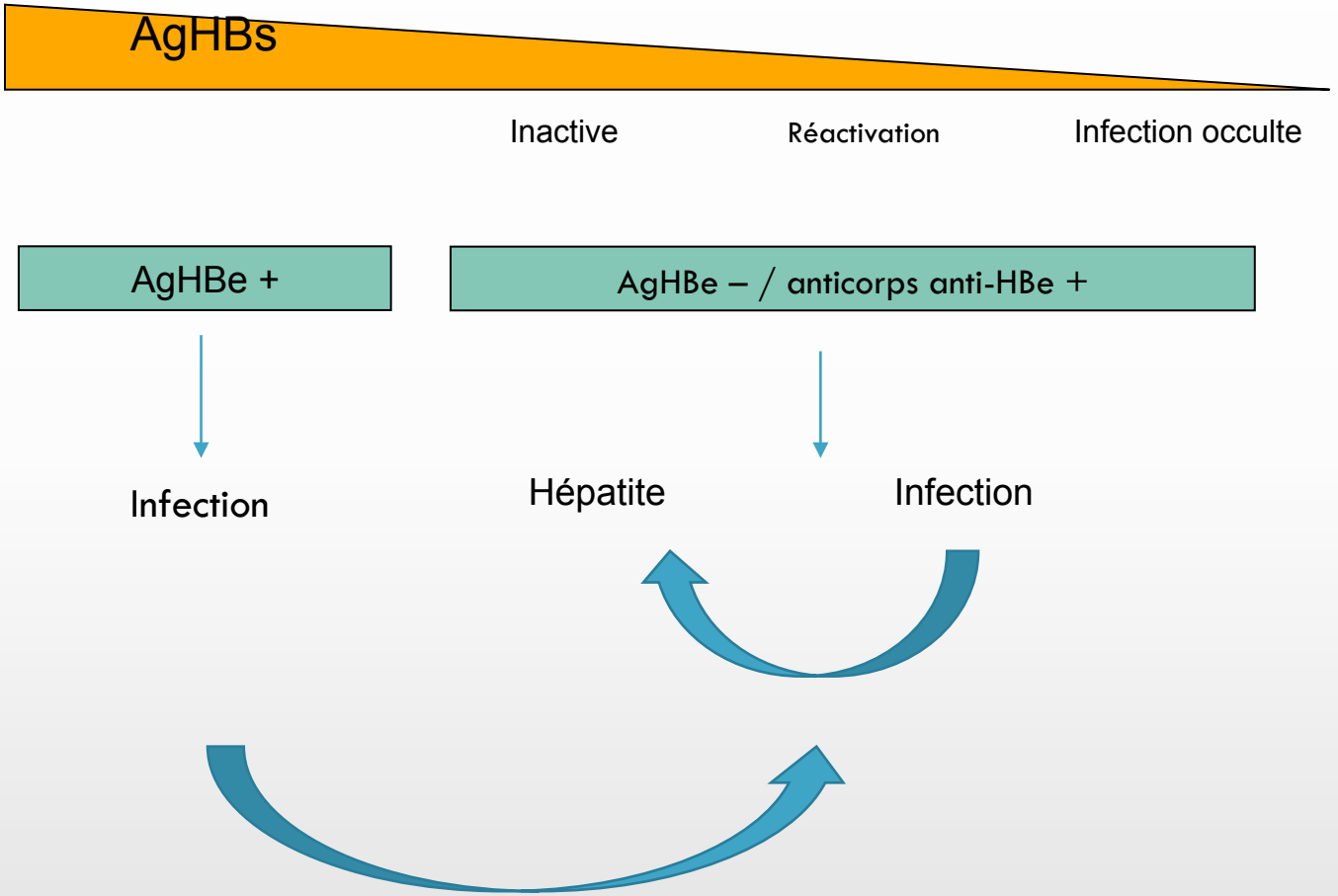
1564

Terrault NA et al. Hepatology 2018;67:1560-99

Nouvelles définitions

	AgHBe positif		AgHBe négatif	
	Infection	Hépatite	Infection	Hépatite
AgHBs	Elevé	Elevé/modéré	Bas	Modéré
ADN VHB (UI/ml)	> 10^7	10^4 - 10^7	< 2000	> 2000
ALAT	N	Elevée	N	Elevée
Fibrose	Absente	Modérée à sévère	Absente	Modérée à sévère

Histoire naturelle de l'hépatite B



Phase d'infection chronique AgHBe+

- ALAT normales
  - ✓ Femmes 19 UI/L
  - ✓ Hommes 30 UI/L
- Séroconversion HBe
  - ✓ 8% par an à la puberté
  - ✓ 12% par an chez l'adulte

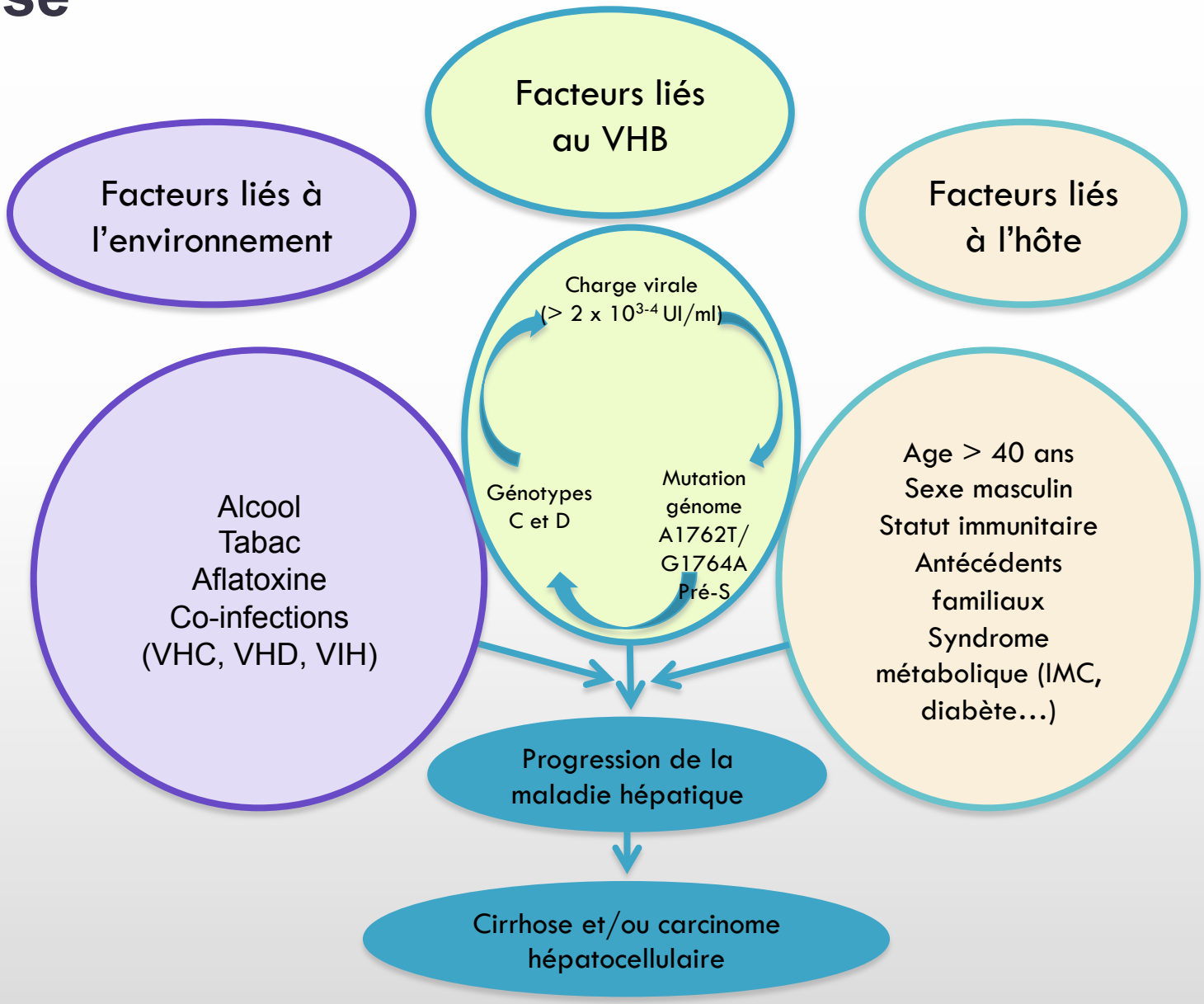
Après séroconversion HBe

AASLD
AMERICAN ASSOCIATION FOR THE STUDY OF LIVER DISEASES

Hépatite	Infection
10 à 30%	70 à 90%

10% à 20%

Facteurs influençant la progression de la fibrose



Liaw & Sollano. Liver Int 2006

Facteurs de risque de progression de la fibrose

Facteurs liés à l'hôte

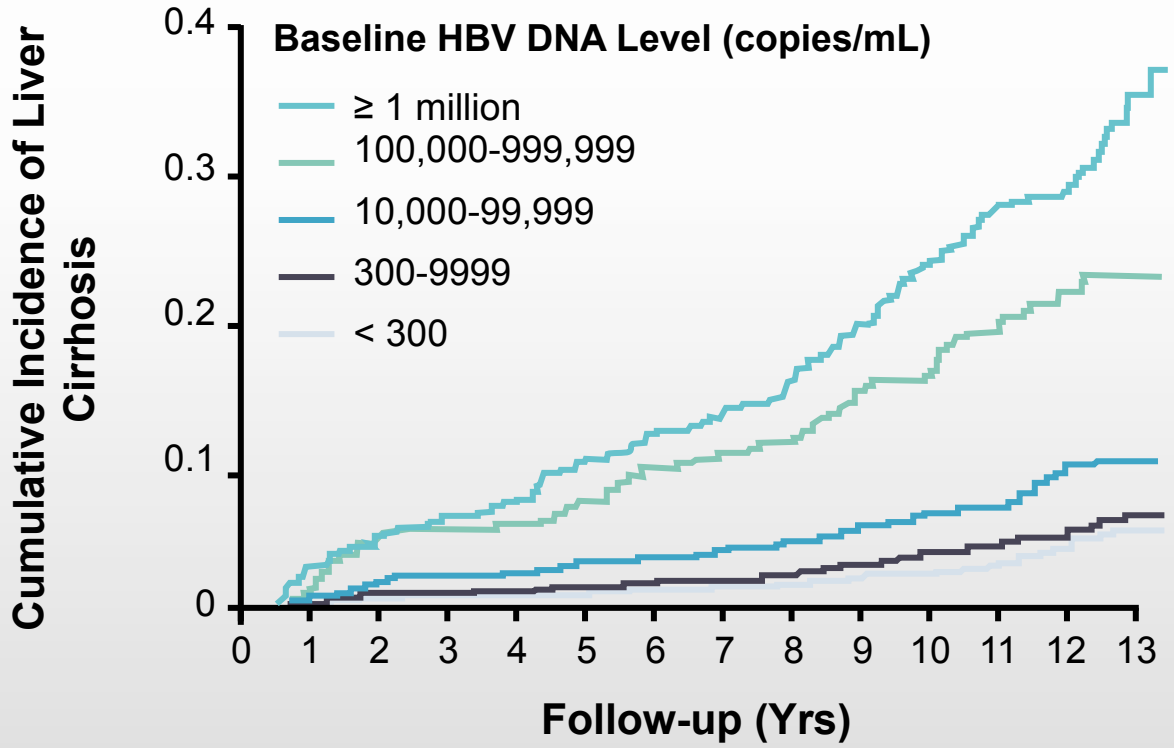
- Hommes
- Age
- Syndrome métabolique
- Alcool
- Coinfections

Facteurs liés au virus

- Taux d'ADN VHB
- AgHBe positif
- Génotype VHB?
  - C > B > A/D

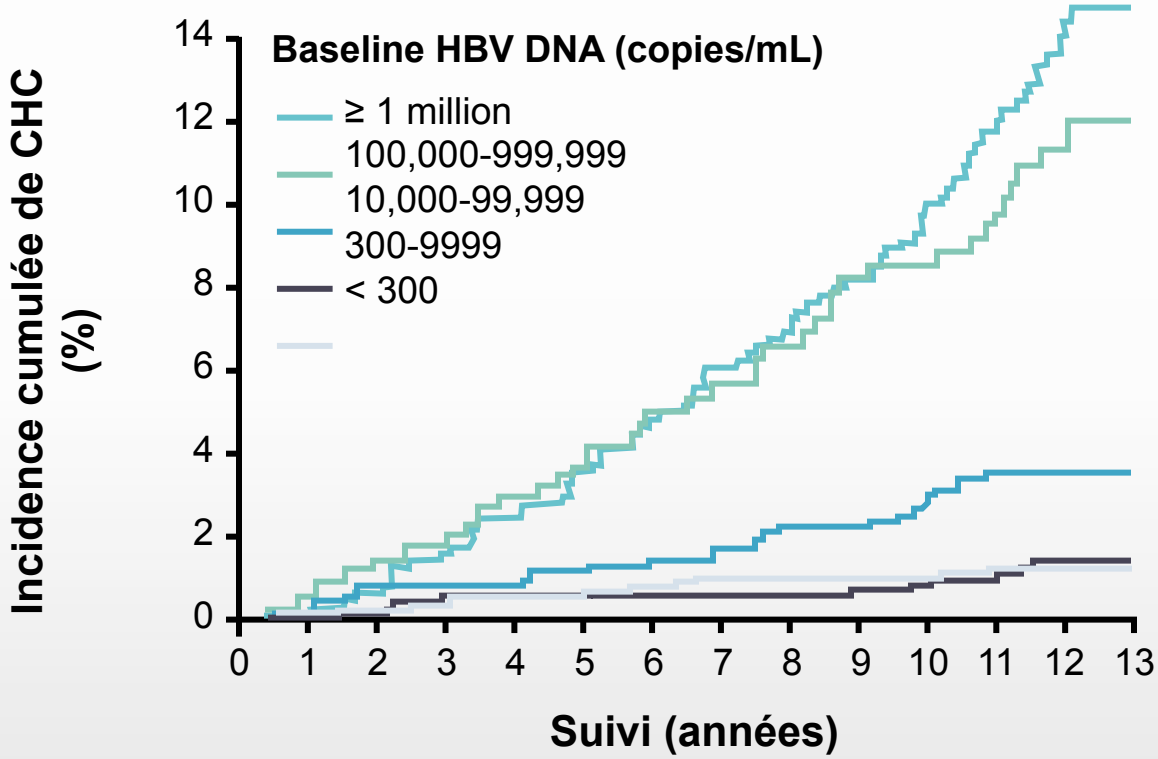
REVEAL. Le risque de progression vers la cirrhose augmente avec la charge virale

- Long-term (mean follow-up: 11.4 yrs) cohort study to determine risk of cirrhosis and HCC in untreated, HBsAg-positive individuals in Taiwan (N = 3582)



Iloeje UH, et al. Gastroenterology. 2006;130:678-686.

Le risque de survenue d'un CHC augmente avec la charge virale (n=3653)



L'incidence du CHC augmente avec la charge virale ($P < .001$)

Le CHC peut survenir même en l'absence de cirrhose

Chen CJ, et al. JAMA. 2006;295:65-73.

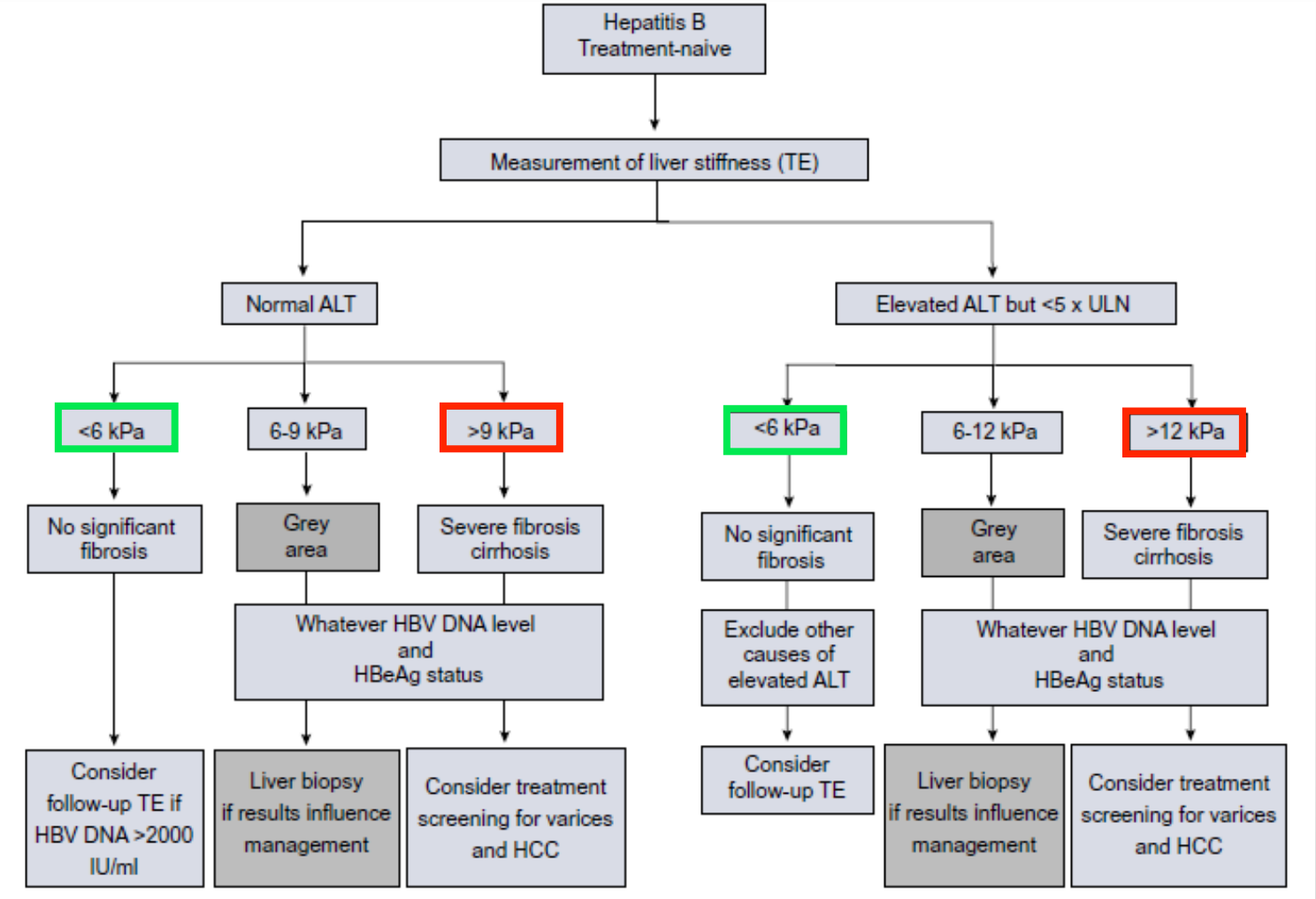
REVEAL. Facteurs de risque de CHC

Facteurs	Adjusted HR	IC 95%	*P*
Hommes	2.1	1.3-3.3	.03
Age (par année)	1.09	1.07-1.11	< .001
HBeAg positive	2.6	1.6-4.2	< .001
Cirrhose	9.1	5.9-13.9	< .001
HBV DNA (copies/mL)			
▪ < 300	1.0	Ref	< .001*
▪ 300-9999	1.1	0.5-2.3	.86
▪ 10,000-99,999	2.3	1.1-4.9	.02
▪ 100,000-999,999	6.6	3.3-13.1	< .001
▪ ≥ 1,000,000	6.1	2.9-12.7	< .001

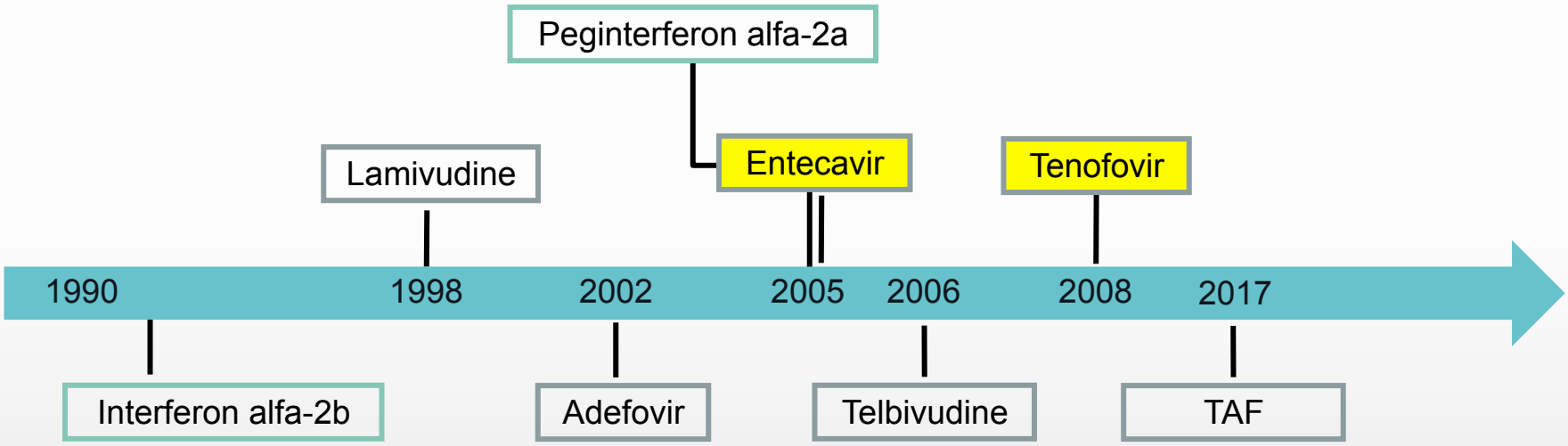
**P* value for trend.

Chen CJ, et al. JAMA. 2006;295:65-73.

L'hépatite B est une maladie hépatique. N'oublions pas d'évaluer la fibrose hépatique



Traitements de l’hépatite B



Quel traitement?

- L'administration à long terme d'un analogue à forte barrière de résistance est le traitement de choix, quelque soit la sévérité de la maladie hépatique
- Les régimes thérapeutiques à privilégier sont **ETV, TDF et TAF** en monothérapie
- **LAM, ADV et TBV ne sont pas recommandés**

Quelle place pour l'interféron?

- PegIFNa peut être considéré comme une option dans le traitement initial des patients avec hépatite chronique minime.
- Durée recommandée de PegIFNa : 48 semaines
- NUC + PegIFNa n'est pas recommandé

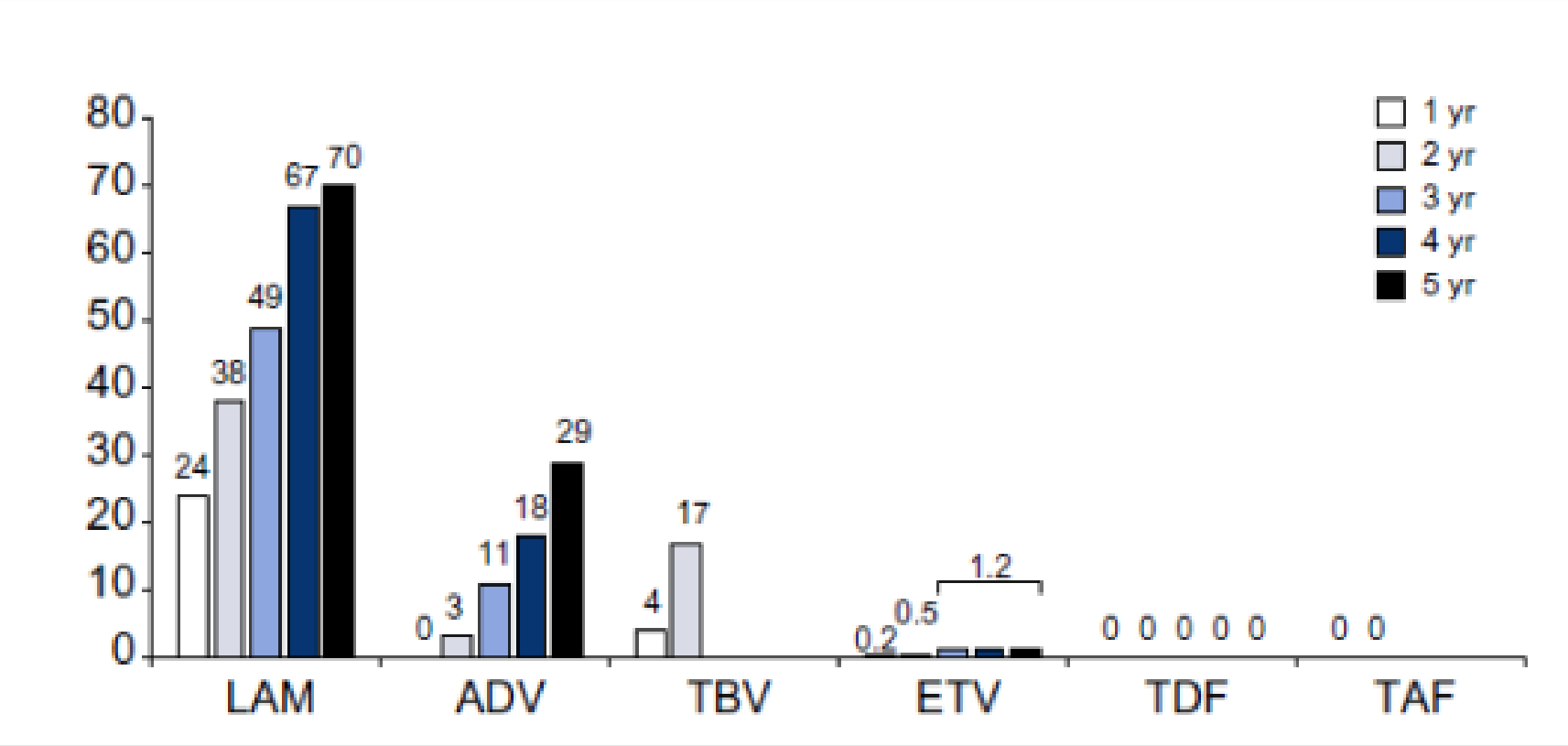
Efficacité des anti-viraux

	Entecavir[1,2]	Tenofovir[3]	PEG-IFN α-2a[4,5]
HBeAg positive	n = 354	n = 176	n = 271
HBV DNA undetectable	67%	76%	25%[a]
HBeAg seroconversion	21%	21%	27%
ALT normalisation	68%	68%	39%
HBsAg loss	2%	3.2%	2.9%[b]
HBeAg negative	n = 325	n = 250	n = 177
HBV DNA undetectable	90%	93%	63%[a]
ALT normalisation	78%	76%	38%
HBsAg loss	0.3%	0%	0.6%[b]

Results at 48 weeks
[a] HBV DNA < 400 copies/mL; [b] At 72 weeks

1. Chang T-T, et al. N Engl J Med 2006;354:1001–10.
2. Lai C-L, et al. N Engl J Med 2006;354:1011–20.
3. Marcellin P, et al. N Engl J Med 2008;359:2442–55.
4. Lau GKK, et al. N Engl J Med 2005;352:2682–95.
5. Marcellin P, et al. N Engl J Med 2004;351:1206–17.

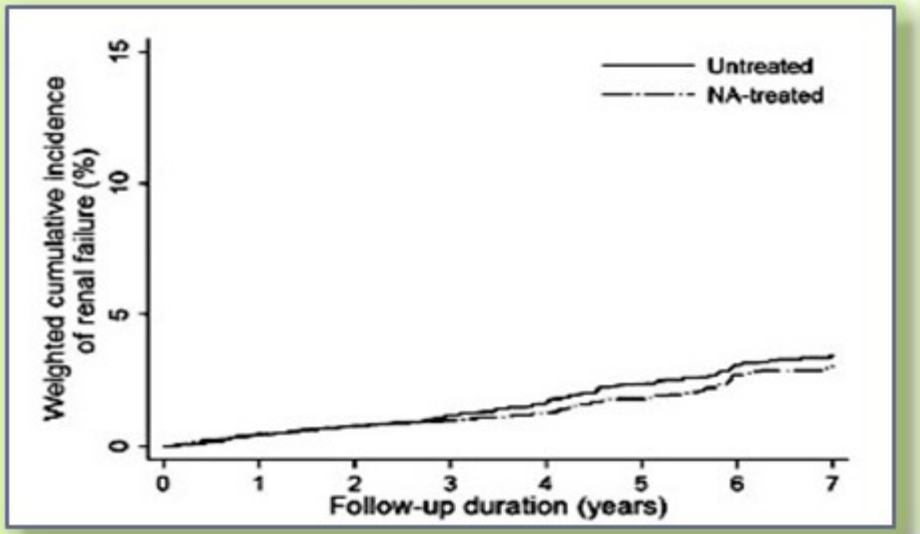
Incidence cumulée des résistances



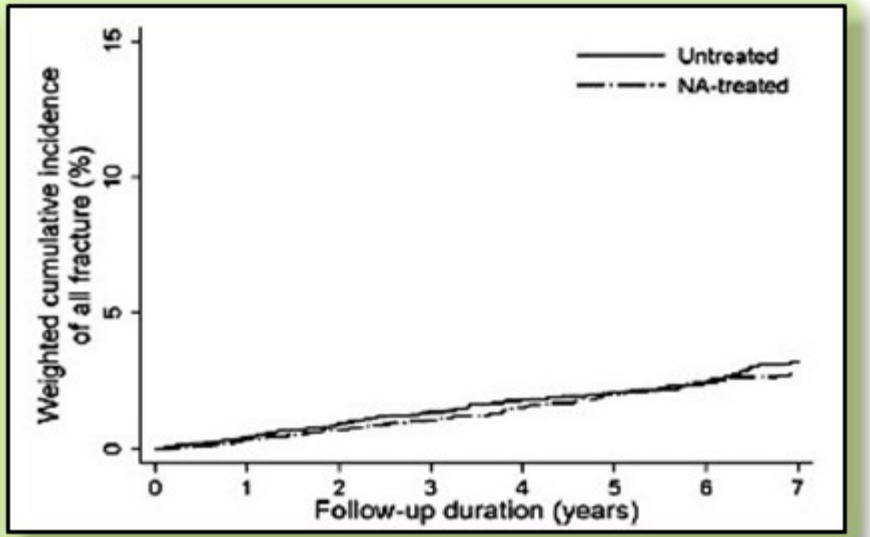
Le traitement au long cours par analogues est sans risque

Etude de cohorte de 53500 sujets (7046 traités) – suivi moyen 4,9 ans

Cumulative Incidence of Renal Failure



Cumulative Incidence of All Fractures



Wong GL et al. Hepatology 2015;62:684-93

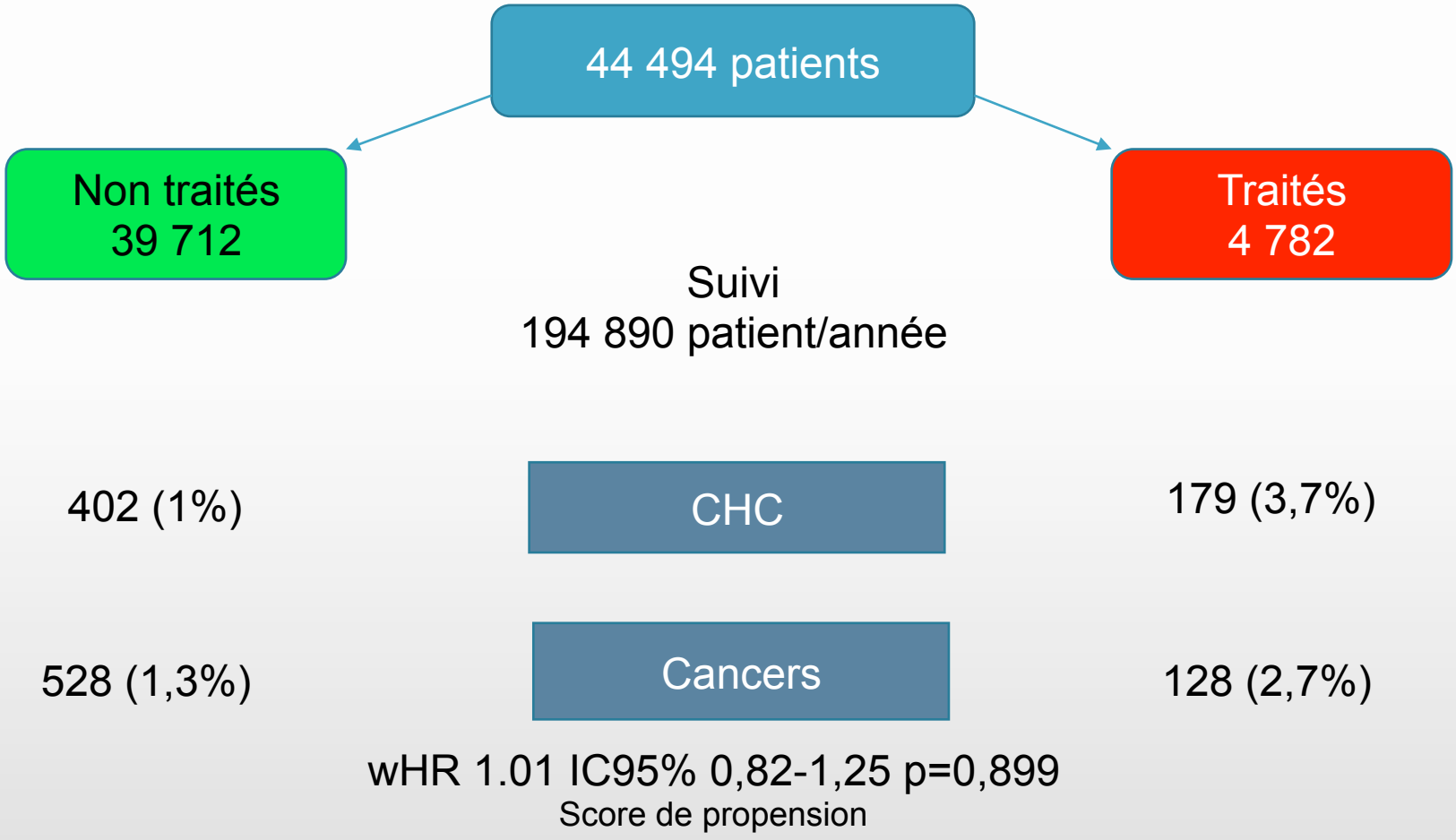
Suivi des patients traités par NUC

also reported chronic tubular damage and decline of e
bone mineral density in TDF treated patients.[70,79–87] T
it seems appropriate for now to monitor all CHB patient
with TDF therapy for adverse renal effects with serum c
(eGFR) and serum phosphate levels. Moreover, CHB pa
high renal risk undergoing any NA therapy should be m
with serum creatinine (eGFR) levels. The frequency of re
itoring can be every 3 months during the first year a
6 months thereafter, if no deterioration. Closer renal m

Journal of H

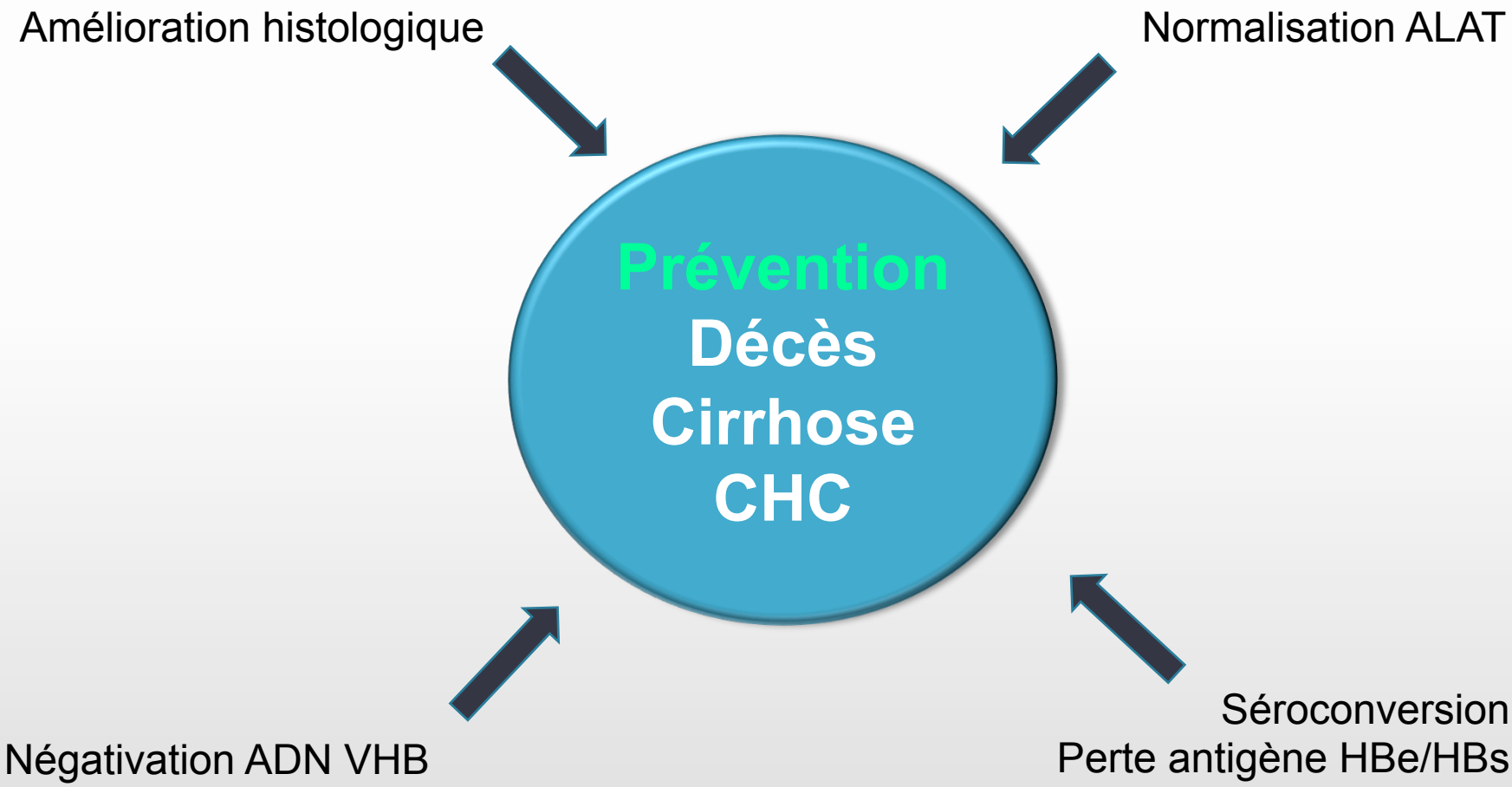
Please cite this article in press as: European Association for th
atitis B virus infection. J Hepatol (2017), http://dx.doi.org/10.

Le traitement au long cours n'augmente pas le risque de cancer



Wong G et al. Aliment Pharmacol Ther 2017;45:1213-24

Buts du traitement de l'hépatite B



Effet sur la maladie hépatique

La stéatose est un facteur associé à la persistance de la cytolyse chez les patients traités par NUC

Persistance d'une cytolyse à 5 ans : 18% des cas

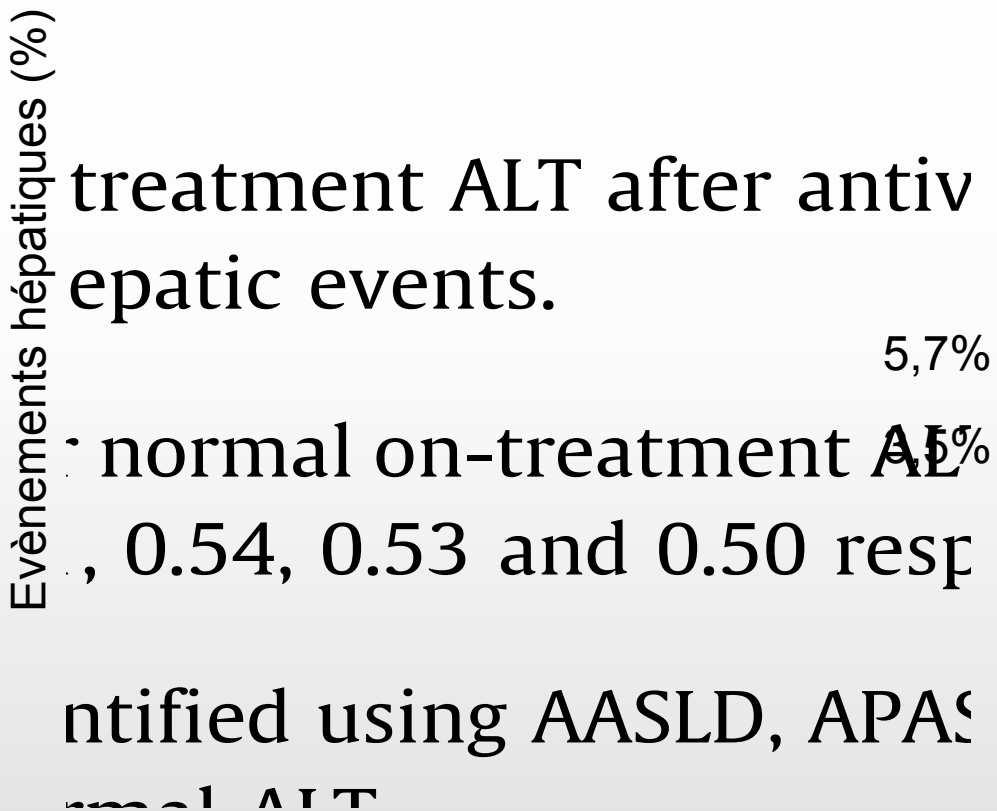
Baseline HBeAg positive vs negative	3.497	1.653–6.576
Baseline steatosis ≥5% vs <5%	2.234	1.031–4.852
Year 5 steatosis ≥5% vs <5%	3.762	1.560–7.375
Age ≤40 vs >40 y	2.099	1.014–4.342

[a] Factors included in the forward model selection (selection entry = 0
significant were as follows: baseline BMI, diabetes, baseline HBV
baseline HBsAg level, baseline Knodell necroinflammatory score, HB
<69 IU/mL at year 5, and change in Knodell necroinflammatory sco

Jacobson IM et al. Clin Gastroenterol Hepatol 2017;15:1087-94

471 patients traités pendant 5 ans par Tenofovir
PBH : 467 à baseline et 339 à 5 ans

Le taux d'ALAT normal (< 30 UI/L) est associé à un moindre risque de complications hépatiques

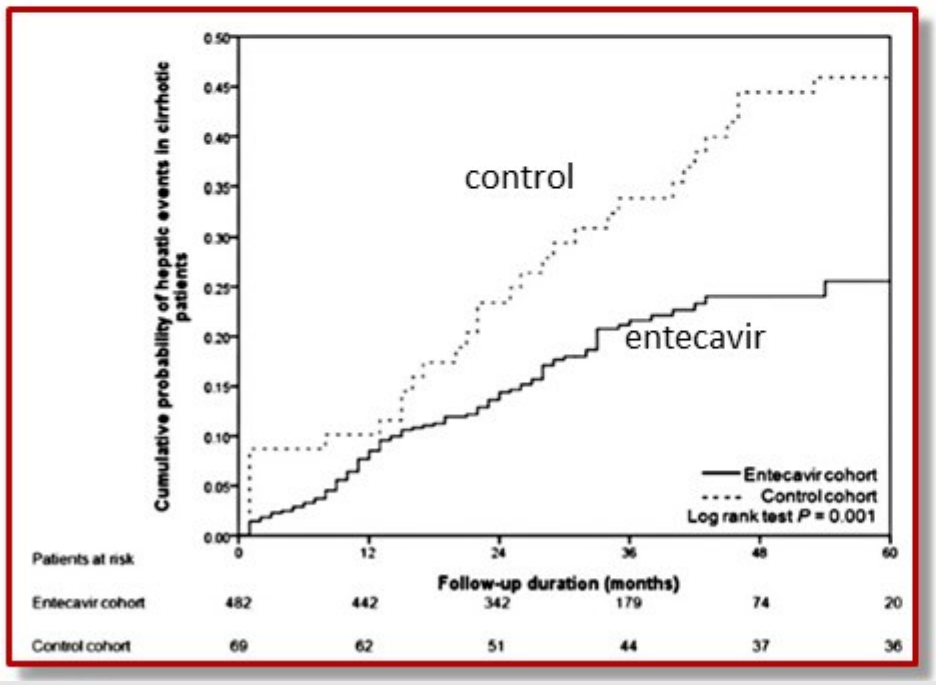


Comparaison des patients avec ALAT < 30 vs > 30 UI/L après un an de traitement

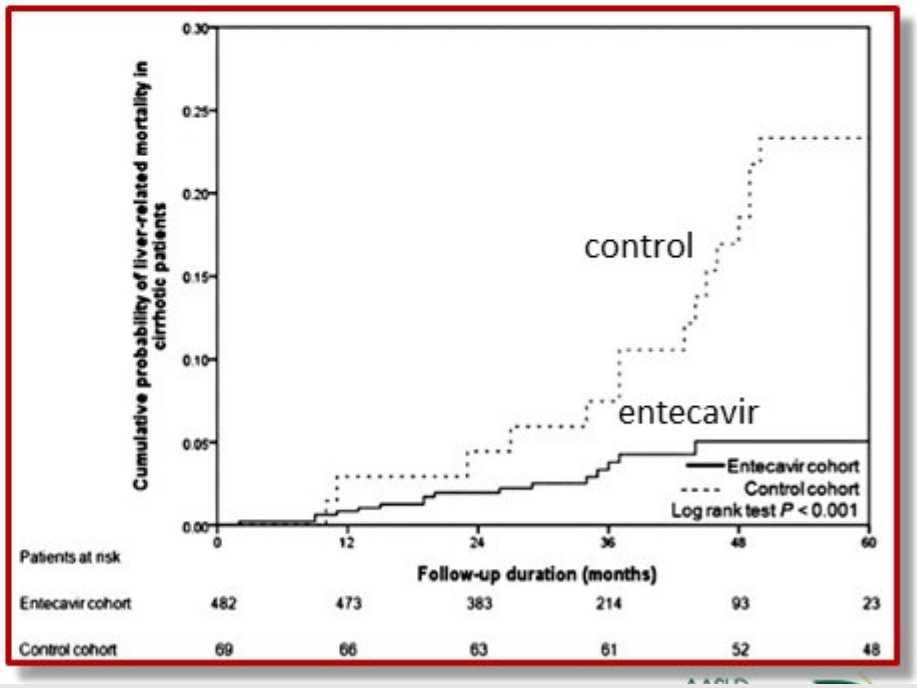
Wong G et al. J Hepatol 2018;69:793-802

Le traitement par entecavir diminue l'incidence des complications de la cirrhose

HEPATIC DECOMPENSATION



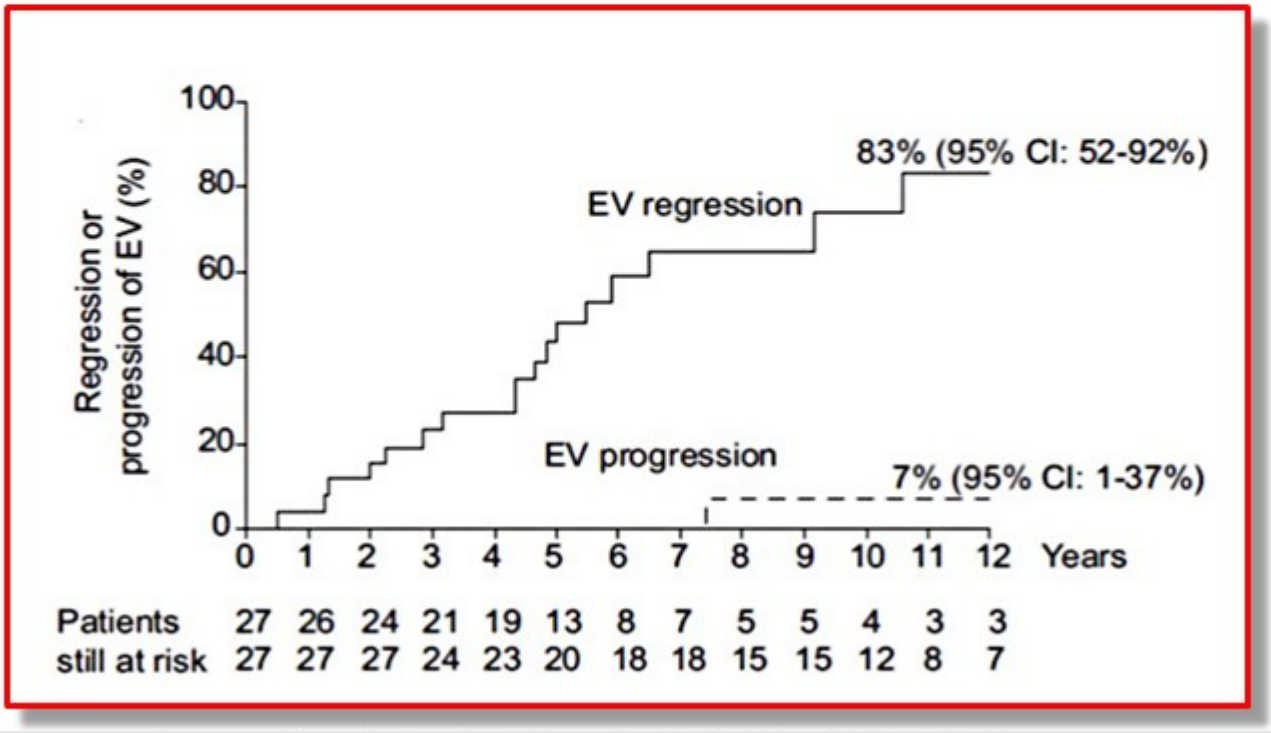
LIVER-RELATED MORTALITY



Wong GL et al. Hepatology 2013;58:1537-47

Le traitement améliore l'hypertension portale

107 e-Ag negative HBV Child's-Pugh A cirrhosis; 12 years follow up



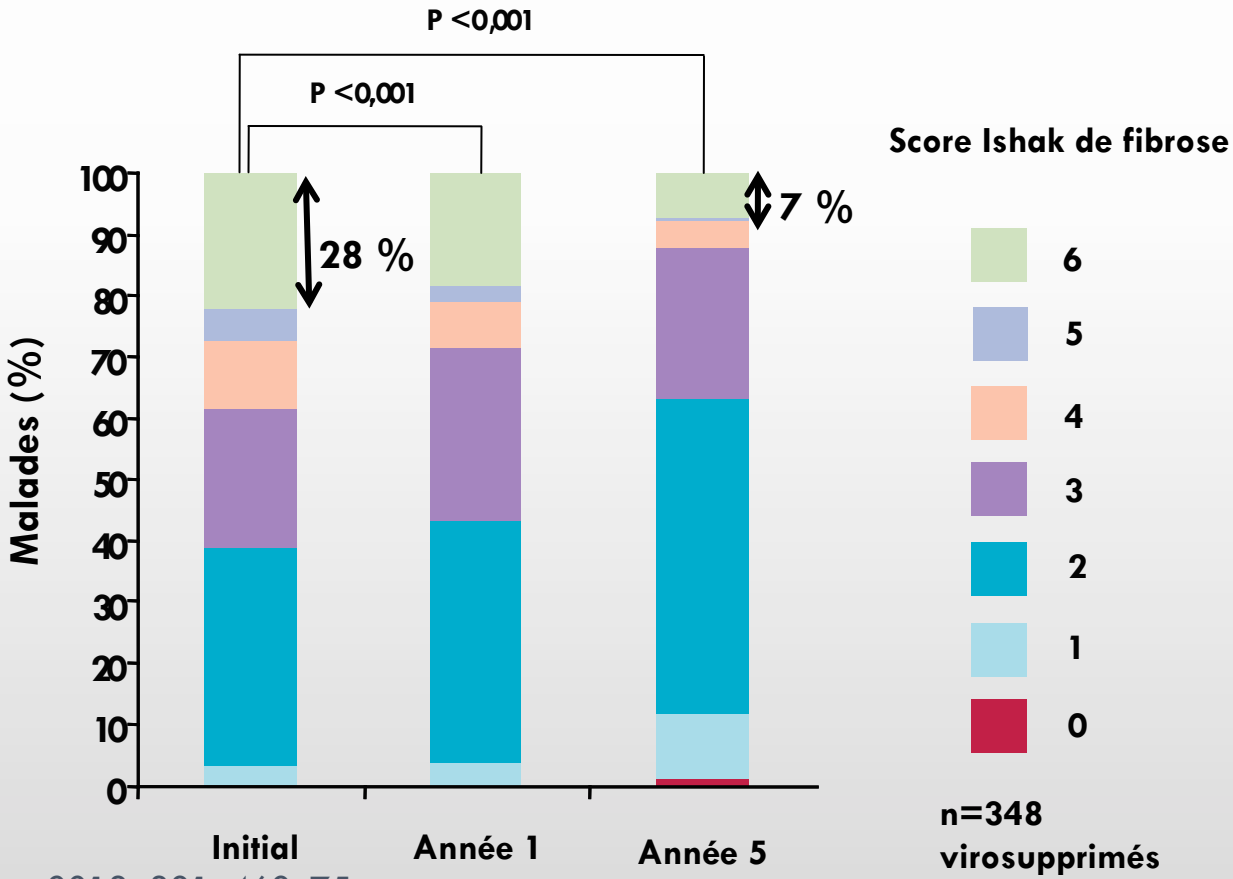
Lampertico P et al. J Hepatol 2015;63:1118-25

Effet sur la fibrose

Ténofovir et régression de la fibrose (Ishak≥ 5)

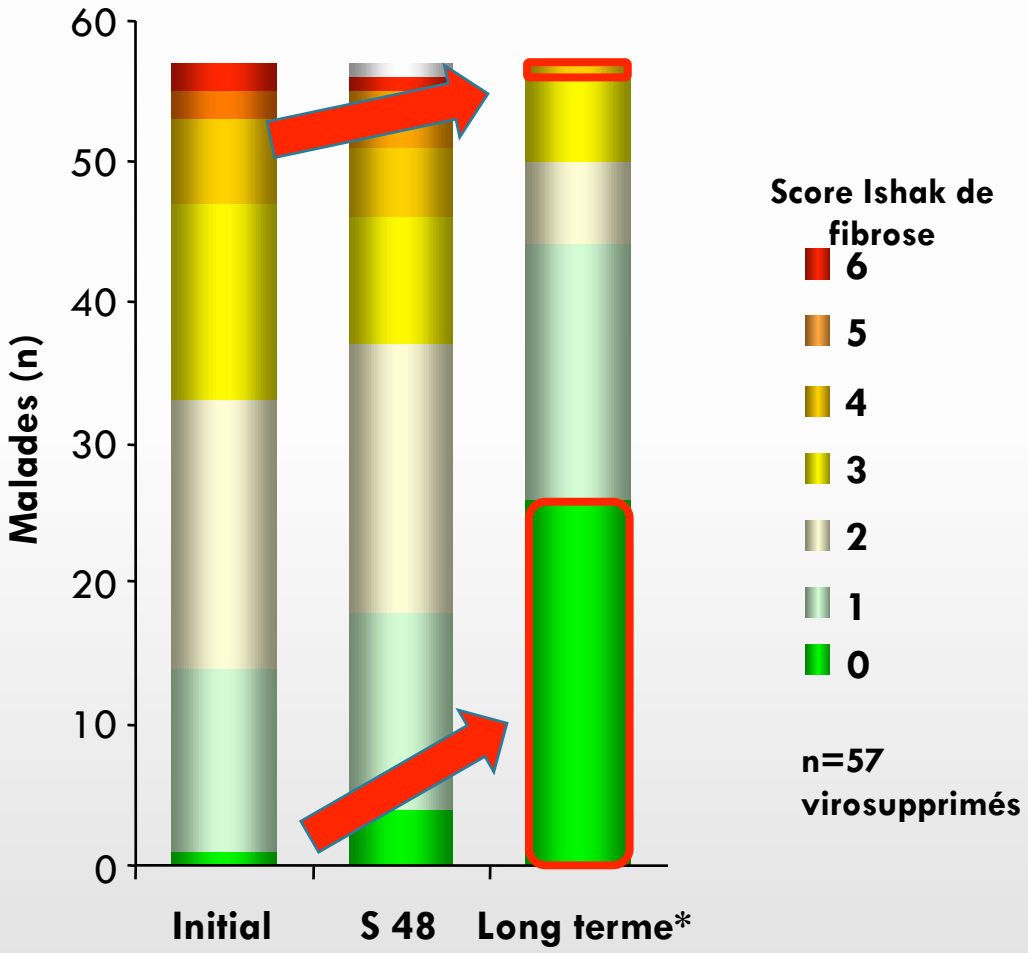
28% avec cirrhose à la phase initiale (96/348)

7% après 5 ans de traitement (25/348)



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

Entécavir et régression de la fibrose



* Temps médian de la biopsie à long terme : 6 ans (extrémités : 3–7 ans)

Chang TT et al, Hepatology 2010; 52: 866-93

Effet sur le risque de CHC

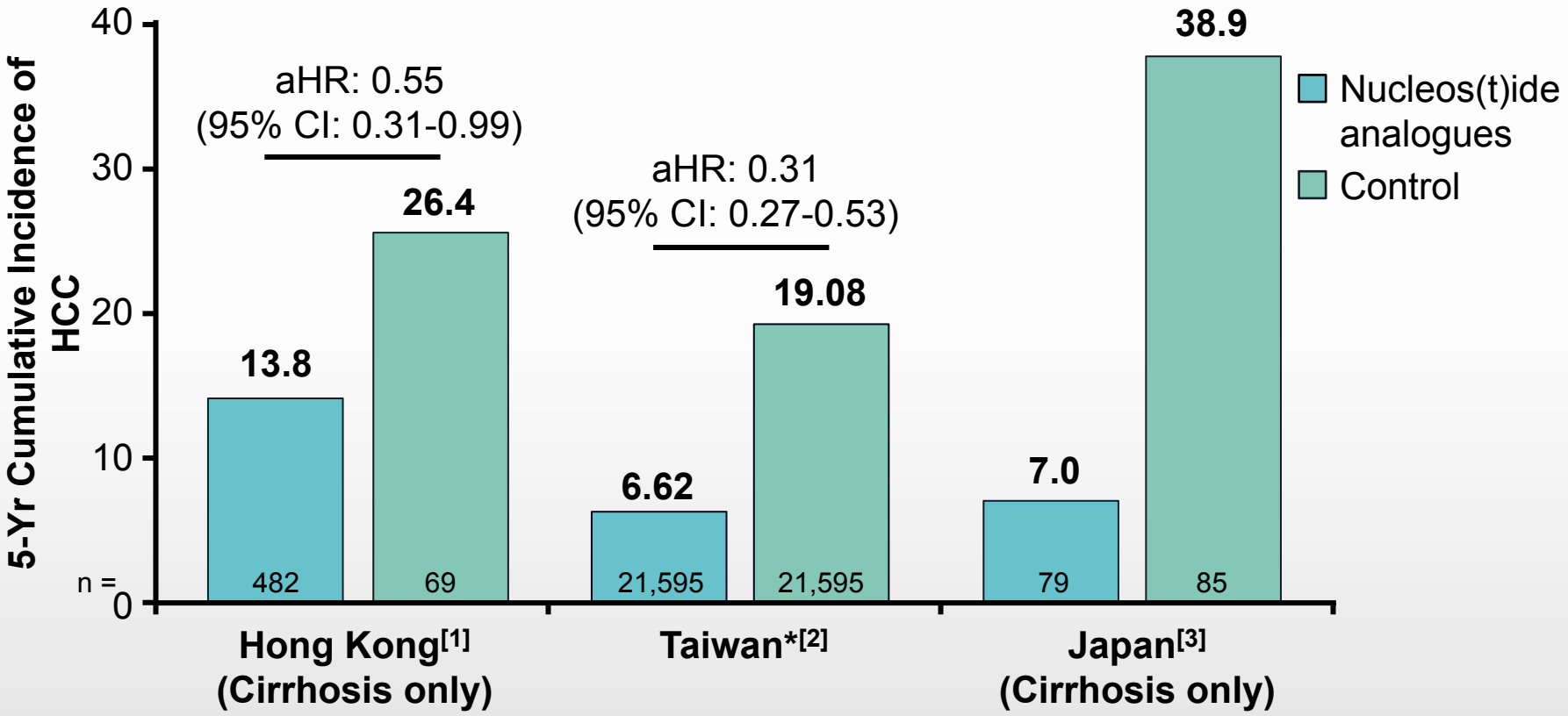
Le traitement antiviral B réduit le risque de CHC

Méta-analyse de 12 études « IFN » et 4 études « analogues »

	Nombre de CHC		RR	p
	IFN	PBO		
Population totale	4,6%	9%	0,66	0,006
Cirrhotique	11,6%	21,5%	0,51	0,001
	Analogues	PBO		
Population totale	2,8%	13,0%	0,22	0,0008
Cirrhotique	3,9%	22,4%	0,17	0,02
Non cirrhotique	1,8%	8%	0,21	0,0001
Sans résistance	2,9%	7,6%	0,36	0,009
AgHBe +	2,0%	9,9%	0,20	< 0,0001

Sung JJY, et al. Aliment Pharmacol Ther 2008; 28: 1067-77

Le traitement antiviral B diminue le risque de CHC

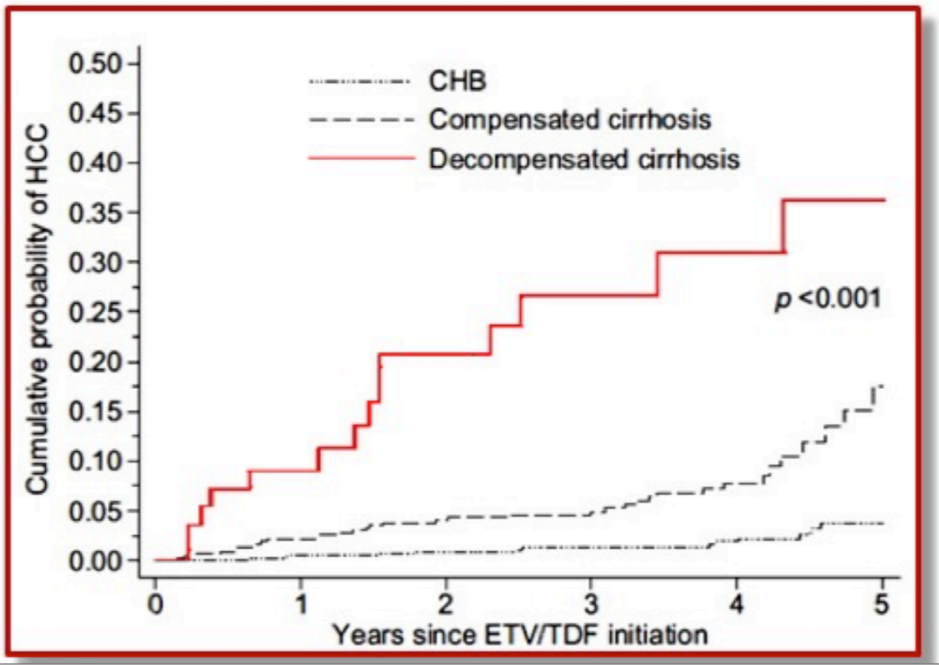


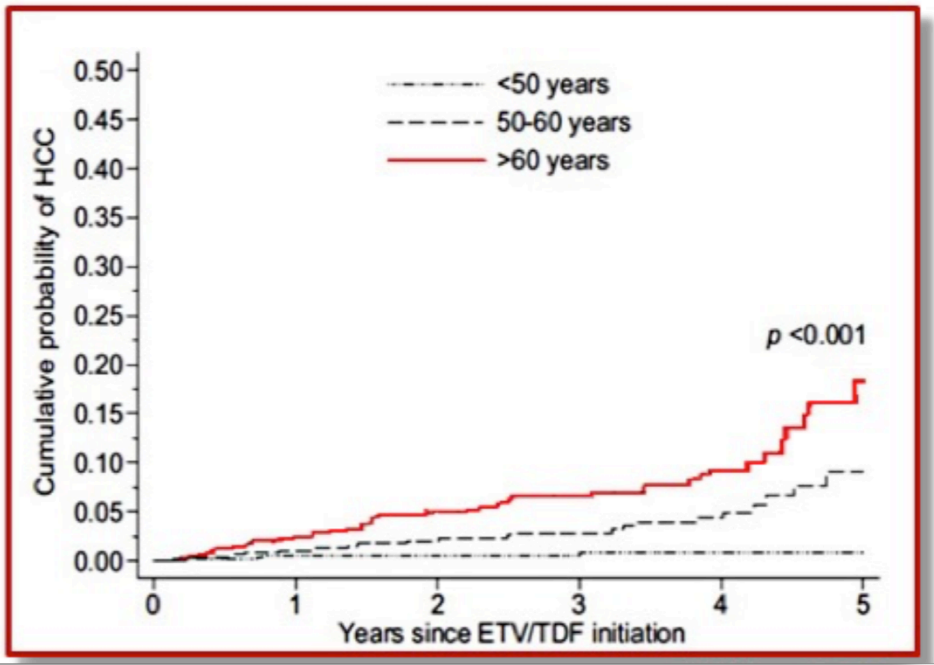
*Incidence rates include cirrhotic pts (13.6% of pts had cirrhosis at baseline) and noncirrhotic pts.

1. Wong GL, et al. Hepatology. 2013;5:1537-1547.
2. Wu CY, et al. Gastroenterology. 2014;147:143-151.
3. Hosaka T, et al. Hepatology. 2013;58:98-107.

Chez le patient cirrhotique, le traitement du VHB n'annule pas le risque de CHC

1666 Caucasian chronic HBV patients treated for a mean of 39 months with entecavir or tenofovir





Papatheodoridis GV et al. J Hepatol 2015;62:363-70

C’est surtout à très long terme que le traitement diminue le risque de CHC

sis, the association of liver stiffness <12 kPa at
with lower HCC risk beyond year 5 did not m
statistical significance after adjustment for age a
telets at year 5 (HR, 0.302; 95% CI, 0.073-1.25
0.099) (Table 2).

HCC RISK SCORES FOR PREDICTION FOR HCC WITHI 5-10 YEARS OF THERAPY

Among 1,171 patients with evaluable PA
score before the onset of ETV/TDF, the mear
dard deviation) value of the score was 13.3
whereas PAGE-B score was low (<10) i
(21.4%), intermediate (10-17) in 582 (49.7%

Papatheodoridis GV et al. Hepatology 2017;66:1444-53

N=1951

Le traitement diminue le risque de CHC à long terme surtout chez les patients cirrhotiques

the patients with baseline cirrhosis, liver sti
kPa at year 5 was associated with lower ris
after year 5 (HR, 0.245; 95% CI, 0.061-0
0.047). No other patient characteristic was f
associated with HCC risk during years 5-10

In multivariable analysis of the baseline fa
older age and lower platelet counts, but no
were found to be independent predictors
development beyond year 5. On the othe
multivariable analysis of factors at year 5,
lower platelet counts, and liver stiffness ≥1
independently associated with a higher risk
after year 5. Among the patients with basel
sis, the association of liver stiffness <12 kP

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Among 1,171 patients with evaluable
score before the onset of ETV/TDF, the m
dard deviation) value of the score was
whereas PAGE-B score was low (<10

Facteurs de risque de CHC après 5 ans

Age > 50 ans, plaquettes basses, élasticité ≥ 12 kPa après 5 ans

Papatheodoridis GV et al. Hepatology 2017;66:1444-53

Le risque de CHC diminue si la charge virale est indétectable sous entecavir (vs < 2000 UI/ml)

	HR (9
Age (/year)	1.00 (0.
Male (versus female)	1.16 (0.
Obesity (yes versus no)	0.86 (0.
HBeAg$^+$ (yes versus no)	0.91 (0.
HBeAg seroconversion (yes versus no)†	1.05 (0.
HBV DNA (/$\log_{10}$IU/L)	0.86 (0.
Cirrhosis (yes versus no)	3.32 (2.
Virological response	
MVR	Refe
LLV	1.69 (1.

*Multivariable model included age, sex, HBeAg, HBV
†Among HBeAg positive patients (n = 483).
Abbreviations: CI, confidence interval; HR, hazard ratio

340

(MVR) rather than the virological response at a single time point. Notably, CVR, defined by a decrease in HBV DNA to an undetectable level (<12 IU/mL), was observed in almost all patients (97%) in our cohort. However, during long-term follow-up, only 498 of 875 patients (57%) showed persistently undetectable HBV DNA levels (MVR), while the remaining patients experienced intermittent or transient episodes of detectable HBV DNA with levels <2,000 IU/mL (LLV). This suggests that patients receiving potent NUC therapy need regular HBV DNA monitoring to verify that they are not experiencing LLV because a single CVR does not guarantee MVR.

We then evaluated factors associated with LLV. HBeAg status, HBV DNA levels, presence of

P=0.44

It is possible that adherence to drug or t
ment of drug-associated mutations is asso
LLV. Adherence to the drug regimen is a
factor in maintaining the virological respon
377 patients with LLV, there was no docu
tory of stopping the drug. However, becaus
rospective design of this study, detailed info
adherence was lacking. Among patients
drug-resistance mutations were documente
minor proportion. However, drug-resista
was not systematically performed in patie
LLV. Therefore, there remains a possibil
development of resistance-associated m
associated with LLV. In this study, body
was higher in those with LLV than MVR

P=0.001

875 patients (51% cirrhose)
Suivi médian 4,5 ans
85 CHC (9,7%)

Kim JH et a; Hepatology 2017;66:335-43

Peut-on prédire le risque de CHC sous NUC?

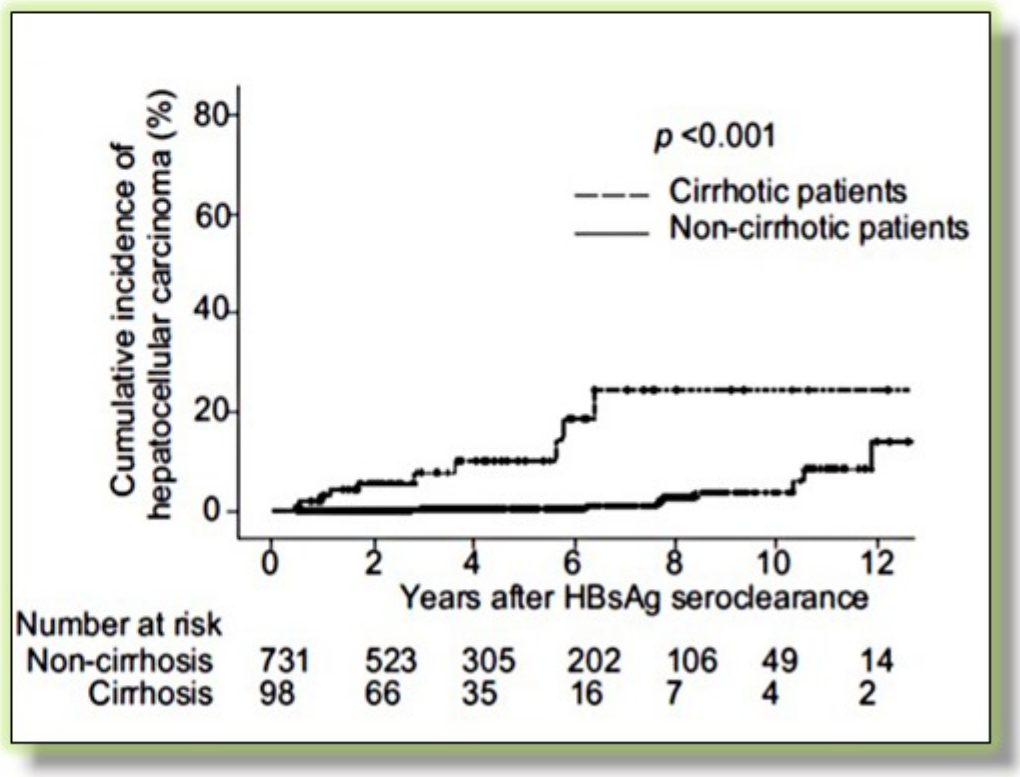
http://dx.doi.org/10.1016/j.jhep.2018.02.032

23 851 patients Taiwan
596 CHC (2,5%)
Incidence du CHC à 3 ans : 3,56%
Validation du score chez 19 321 patients de Hong Kong

Hsu YC et al. J Hepatol 2018;69:278-85

Le risque de CHC persiste même après séroclearance HBs

- 829 Korean patients who achieved HBsAg seroclearance
- **19 developed HCC**
- **Risk factors for HCC**
  - Male gender
  - Age ≥50 years at seroclearance



Kim GA et al. J Hepatol 2015;62:1092-9

Il faut donc dépister le CHC

6 months thereafter, if no deterioration. Closer renal

Journal o

Please cite this article in press as: European Association for
atitis B virus infection. J Hepatol (2017), http://dx.doi.org/1

Dépistage du CHC chez les patients non traités

- Hommes Asiatiques ou Africains > 40 ans
- Femmes Asiatiques > 50 ans
- Patients avec membre de sa famille de 1er degré avec CHC

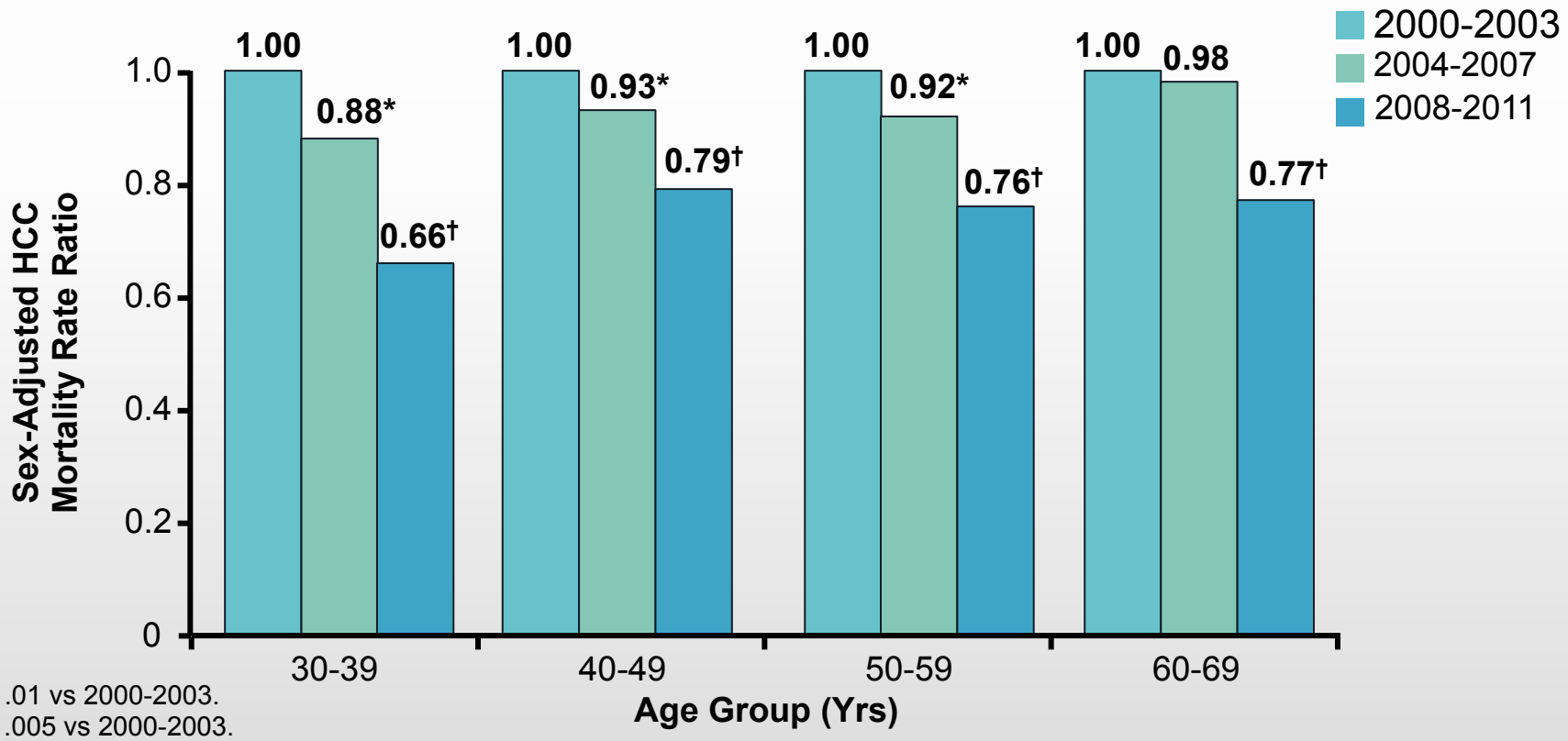
Terrault NA et al. Hepatology 2018;67:1560-99

Effet sur la mortalité

Réduction de la mortalité par CHC grâce au programme national de traitement des hépatites virales B

Pts receiving treatment for chronic hepatitis after start of program in 2003 in Taiwan: 157,570 (HBV) and 61,823 (HCV)

Reduced rate of HCC mortality in all age cohorts by 5-8 yrs after introduction of national therapy program

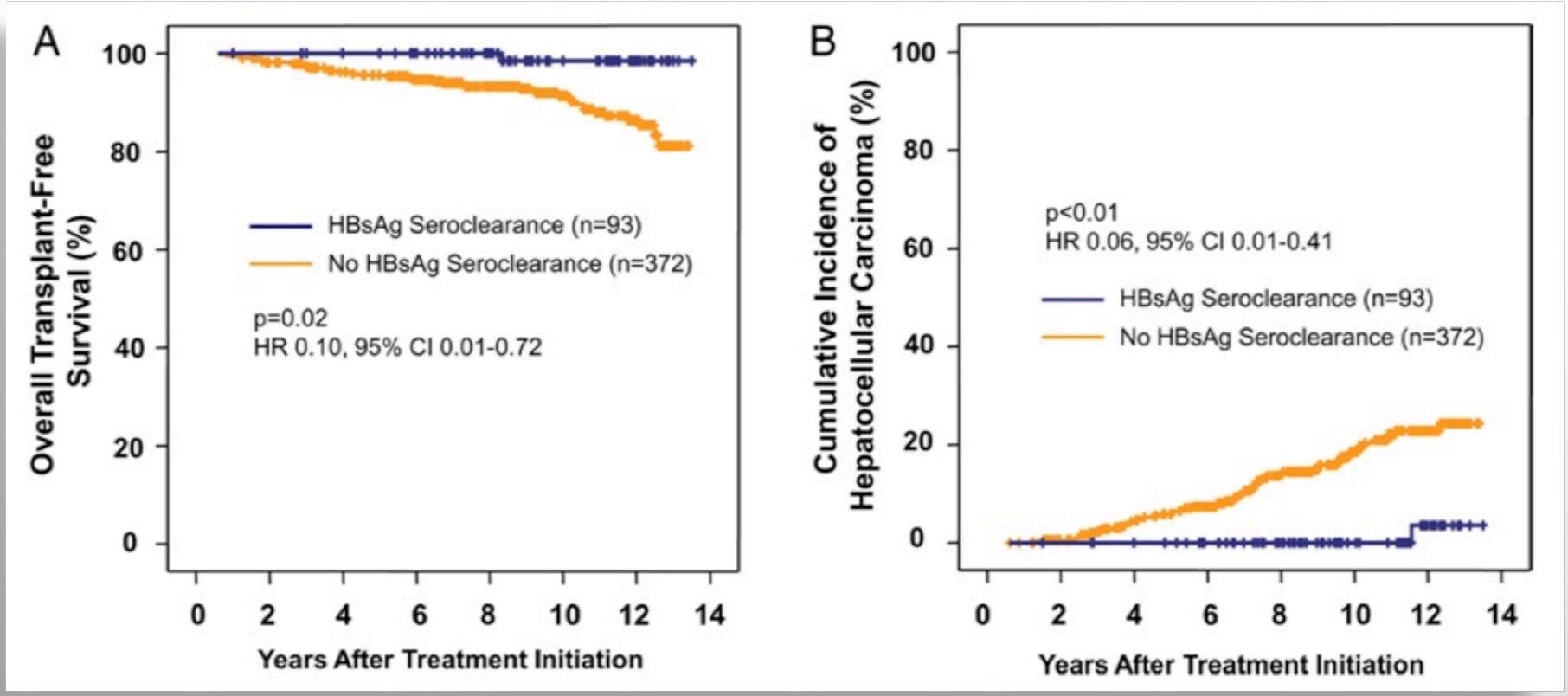


*P < .01 vs 2000-2003.
†P < .005 vs 2000-2003.

Chiang CJ, et al. Hepatology. 2015;61:1154-1162.

La séroconversion HBs est associée à un excellent pronostic

Patients traités par lamivudine ou entecavir



Kim GA et al. Gut 2014;63:1325-32

Quand arrêter les analogues?

- **Après perte de l'antigène HBs**
- Chez les patients non-cirrhotiques
  - ✓ **HBeAg positive** avec seroconversion HBe et ADN VHB indétectable
  - ✓ **HBeAg-negative** avec au moins 3 ans d'ADN VHB indétectable

Surveillance rapprochée si arrêt

Indications au traitement

Que l'antigène HBe soit + ou −

Tous les patients avec hépatite chronique virale B définie par

- ✓ HBV DNA > 2,000 IU/ml,
- ✓ ALT > ULN
- ✓ Et/ou au moins A2 ou F2

Indications au traitement

- Les patients avec **cirrhose** compensée ou décompensée doivent être traités, quelle que soit la charge virale ou le taux d'ALAT

- Les patients antigène HBe + ou – avec **histoire familiale de CHC ou de cirrhose** doivent être traités

Indications au traitement

- Patients avec **ADN VHB > 20 000 UI/ml et ALAT > 2xULN** quelque soit le score de fibrose

- Patients HBeAg-positive avec ALAT N et forte charge virale **âgés de plus de 30 ans** quelque soit le score de fibrose

Cas particuliers

Professionnels de santé

- Ne pas exclure d'une profession de santé un patient infecté par le VHB

- Ne pas hésiter à traiter tout professionnel de santé infecté

Réactivation virale B

- Tous les patients qui vont recevoir une chimiothérapie ou un traitement immunosuppresseur doit avoir une sérologie VHB
- Tous les patients HBs positif doivent être traités par un NUC
- Les patients avec anticorps anti-HBc isolé doivent être traités en fonction du risque de réactivation

Dialysés et transplantés rénaux

- Tous les patients dialysés et transplantés rénaux doivent être dépistés pour le VHB
- Les patients HBsAg-positive dialysés ou transplantés rénaux doivent être traités par entecavir ou TAF
- Les patients avec anticorps anti-HBc isolé doivent être monitorés régulièrement

Traitement des patients VIH-VHB

- Tous les patients VIH avec coinfection VHB doivent être traités par antirétroviraux, quel que soit le taux de CD4.

- Tous les patients HIV-HBV devraient être traités par un schéma antirétroviral à base de Tenofovir.

Traitement des enfants et adolescents

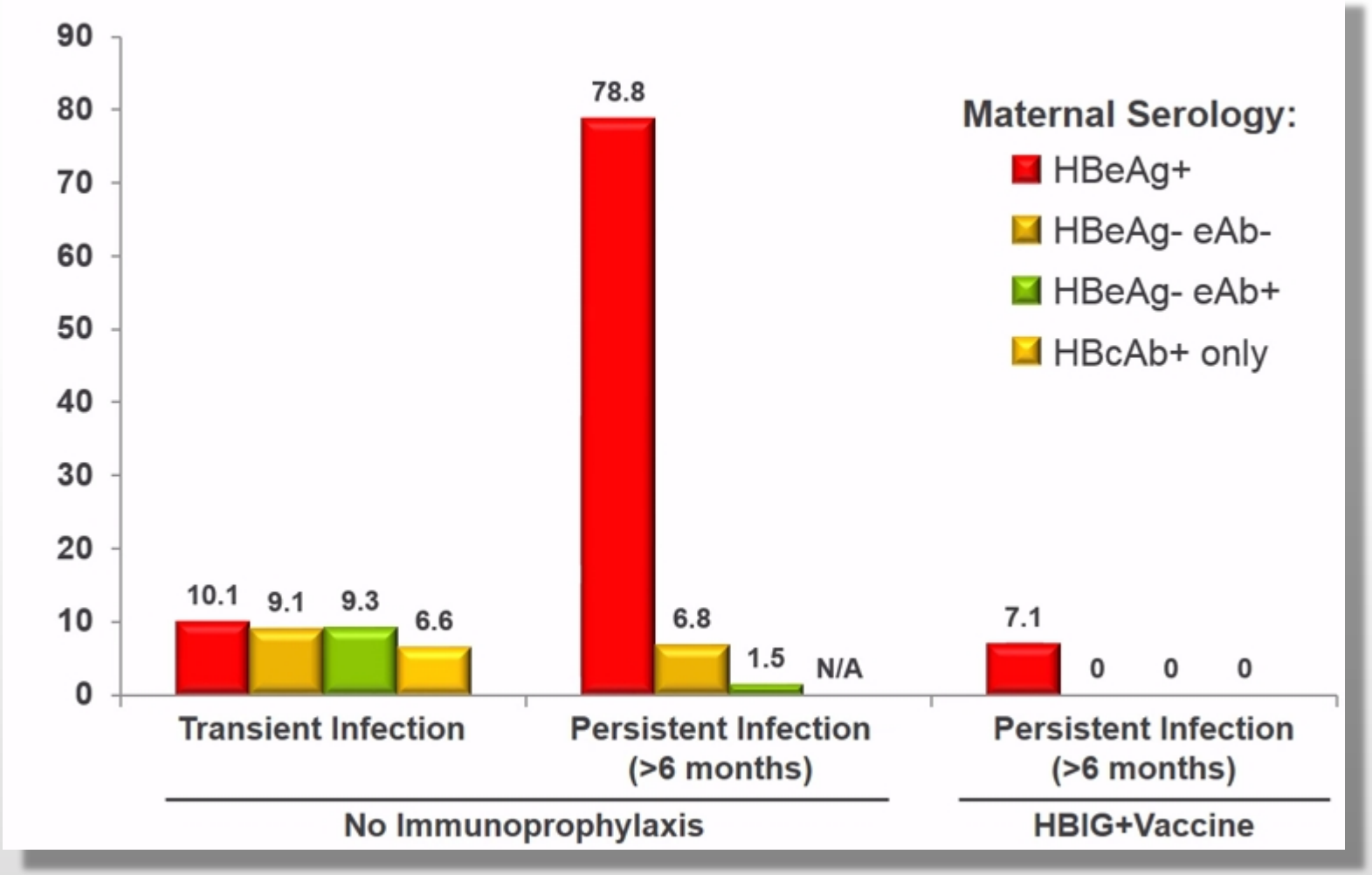
- Chez les enfants, la maladie est souvent minime et n'a pas les critères pour être traitée.

- Chez les enfants ou les adolescents, les même antiviraux que ceux utilisés chez les adultes peuvent être prescrits.

Hépatite B et femmes enceintes

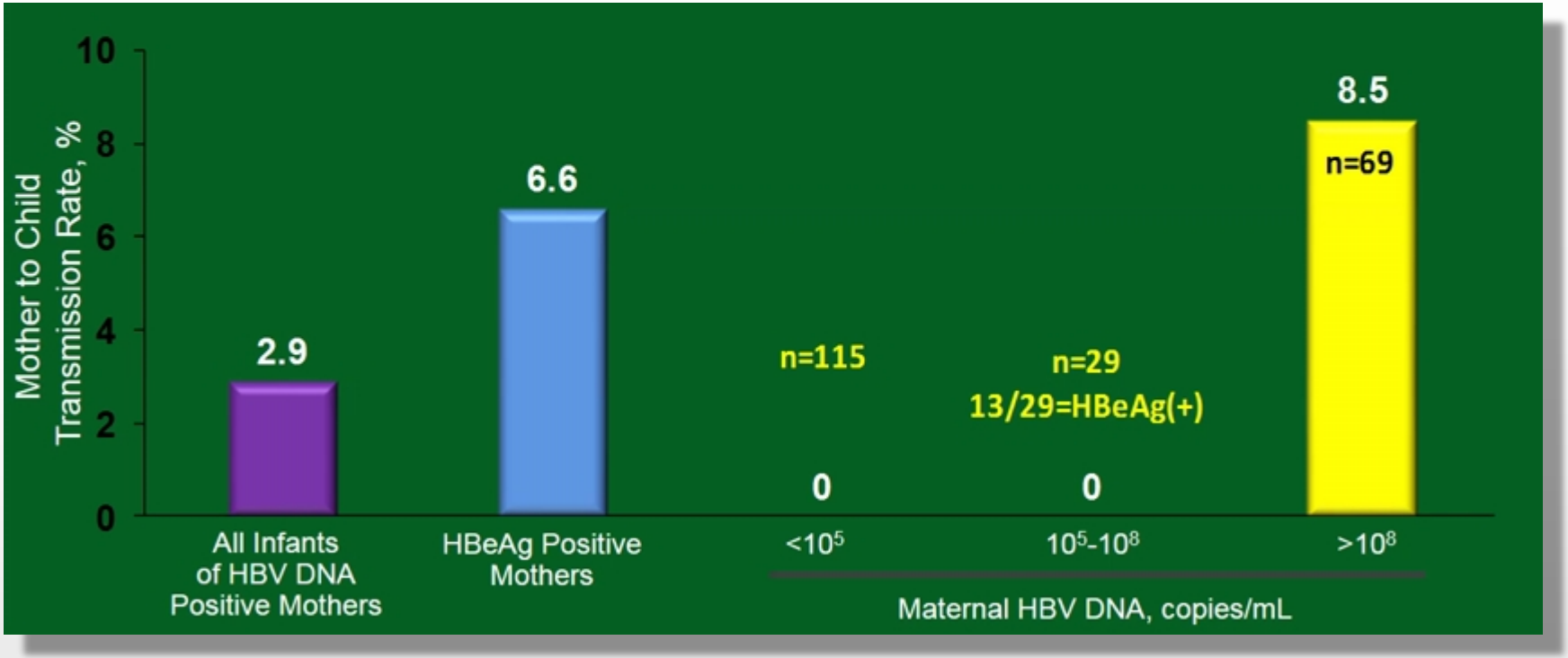
- Le dépistage de l'antigène HBs durant le 1er trimestre de grossesse est fortement recommandé.

Le risque de transmission materno-fœtale est plus important chez les mères AgHBe +



Pan CQ et al. Clin Gastroenterol Hepatol 2012;10:452-9

Le risque de transmission materno-fœtale dépend de la charge virale



Les enfants ont reçu des Ig 100 UI dans les 12 heures suivants leur naissance et une vaccination à J0 M2 M4 et M6.

Wiseman E et al. J Med Austr 2009;190:489-92

Quel traitement prescrire chez la femme enceinte?
Recommandation FDA

Category B

- Telbivudine
- Tenofovir-DF

Pregnancy category B:
Animal studies do not indicate a risk to the fetus and there are no controlled human studies, or animal studies show an adverse effect on the fetus but well-controlled studies in pregnant women have failed to demonstrate a risk to the fetus

Category C

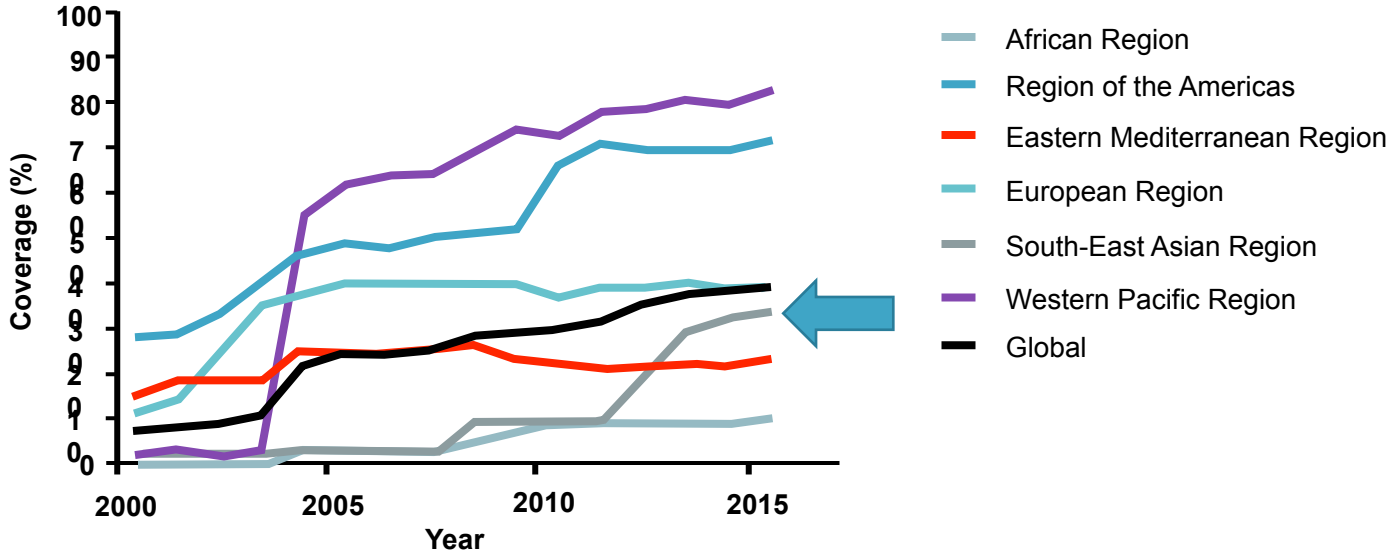
- Interferon alfa
- Peginterferon alfa-2a
- Peginterferon alfa-2b
- Lamivudine
- Adefovir
- Entecavir

Pregnancy category C:
Studies have shown that the drug exerts animal teratogenic or embryocidal effects, but there are no controlled studies in women, or no studies are available in either animals or women.

Taux de couverture de la dose initiale à la naissance par région

Au niveau mondial seulement **39%** des enfants ont la première injection à la naissance

En Afrique seulement **11 pays sur 54** ont adopté cette stratégie



WHO. Global Hepatitis Report 2017. Available at: http://apps.who.int/iris/bitstream/10665/255016/1/9789241565455-eng.pdf (accessed November 2018); Dionne-Odom J, et al. Clin Ther 2018;40:1255–67

Vaccination (3 doses) selon les continents

Progress has been made since 1992, when the World Health Assembly formulated a resolution recommending the inclusion of hepatitis B vaccine in the EPI by 1997 (WHA 45.17) *(1)*.This resolution paved the way for nations to incorporate hepatitis B vaccine into their national immunization programmes. In 2015, 185 of 194 WHO Member countries (95%) had included hepatitis B vaccine in the EPI. An additional nine countries used schedules that started later in life or that targeted high-risk populations. Between 1990 and 2015, hepatitis B vaccine coverage in infants increased from 1% to 84% (WHO–UNICEF joint reporting form data), in part due to the support of the Global Alliance for Vaccines and Immunization *(74)* and to facilitated procurement through the revolving fund of the Region of the Americas.

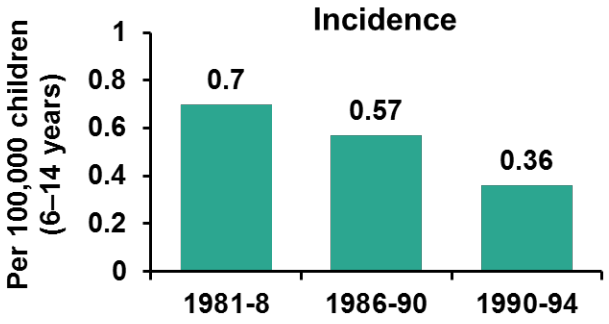
PREVENTION OF MOTHER-TO-CHILD TRANSMISSION OF HBV REMAINS LOW IN FOUR REGIONS

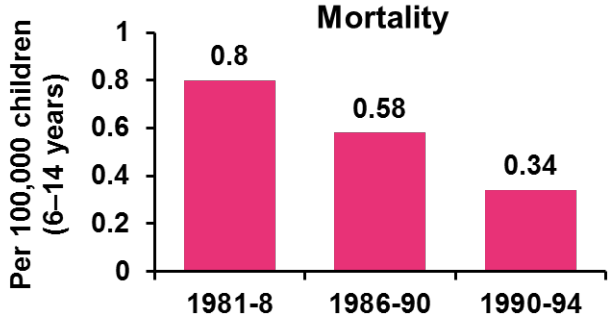
Following the progressive evolution in 2004 *(75)* 2009 *(1)* of the global WHO recommendation t hepatitis B immunization at birth, coverage of th dose increased, reaching 39% globally in 2015 (I In 2015, the birth dose of hepatitis B vaccine ren the cornerstone of prevention of transmission of from mother to child. Ideally, the birth dose sho given within 24 hours of birth. However, the exa of administration of the birth dose is not clear as always readily reported. While a birth dose can s partially effective against mother-to-child transn

Impact de la vaccination sur la prévalence du portage de l'Ag HBs et l'incidence du CHC

pays	Nb	Suivi (years)	Couverture vaccinale (%)	% de portage de l'AgHBs avant	% de portage de l'AgHBs après
Gambie	675	9	100	10	0.6
Indonésie	2519	4	90	6.2	1.9
Arabie Saoudite	4791	1–8	85	6.7	0.3
Thaïlande	3373	0–5	90.4	5.4	0.8

Carrier rate: 9.8% in 1984, 1.3% in 1994, 0.7% in 1999*





Namgyal P. J Hepatol 2003;39 S77–S82;

Chang MH, et al. New Eng J Med 1997;336 1855-59

*Nationwide vaccination in Taiwan, implemented July 1984

Conclusion

- Nouvelles définitions de l’hépatite B
- Le traitement prévient la cirrhose, le CHC et améliore la survie
- Le risque de CHC persiste
- Dépistage du CHC+++
- Programmes de dépistage et de vaccination

Merci

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