Treating hepatitis B and C (in South Africa)

Dr Mark Sonderup
Division of Hepatology and Department of Medicine
University of Cape Town & Groote Schuur Hospital
Treatment of Hepatitis B
Evolution of HBV Therapy

- Interferon alfa-2b (1990)
- Lamivudine (1998)
- Peginterferon alfa-2a (2002)
- Adefovir (2005)
- Entecavir (2006)
- Tenofovir (2008)
- Telbivudine (2008)
4 Phases of Chronic HBV Infection

Current Understanding of HBV Infection

<table>
<thead>
<tr>
<th>Phase</th>
<th>Immune Tolerant</th>
<th>Immune Clearance</th>
<th>Immune control</th>
<th>Reactivation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver</td>
<td>Minimal inflammation and fibrosis</td>
<td>Chronic active inflammation</td>
<td>Mild hepatitis and minimal fibrosis</td>
<td>Active inflammation</td>
</tr>
</tbody>
</table>

REVEAL Study: Risk of HCC and Cirrhosis according to baseline HBV viral load

HCC Incidence in TDF Studies Lower Than Predicted by REACH-B Risk Model

- Analysis of actual HCC incidence vs REACH-B predictions in 152 cirrhotic, 482 noncirrhotic pts treated with TDF for 8 yrs in studies 102 (HBeAg-) and 103 (HBeAg+)
- Noncirrhotics: 8 observed cases vs 18 predicted over 7 yrs
  - Significant difference from Wk 240: 55% reduction in HCC
- Cirrhotics: observed cases matched prediction over first 4 yrs; no observed cases in last 3 yrs
- Combined analysis: 50% lower HCC incidence at Yr 7

*Statistically significant.

# Treatment Criteria for Chronic Hepatitis B

**HBeAg pos and HBeAg neg Disease**

<table>
<thead>
<tr>
<th>Liver Society Guidelines*</th>
<th>HBeAg Positive</th>
<th>HBeAg Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HBV DNA, IU/mL</td>
<td>ALT</td>
</tr>
<tr>
<td>APASL 2008[2]</td>
<td>≥ 20,000</td>
<td>&gt; 2 x ULN†</td>
</tr>
<tr>
<td>AASLD 2009[3]</td>
<td>&gt; 20,000</td>
<td>&gt; 2 x ULN† or (+) biopsy</td>
</tr>
</tbody>
</table>

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*Although ALT and HBV DNA are primary tests used to determine treatment candidacy, the levels of elevation that warrant consideration of treatment are not universally agreed upon.

†Laboratory normal.

‡30 U/L for men and 19 U/L for women.

**In patients older than 40 yrs of age, 2000 IU/mL should be considered as a cutoff for treatment.

South African guideline for the management of chronic hepatitis B: 2013

C W N Spearman,1 MB ChB, FCP (SA), MMed, PhD; M W Sonderup,1,2 MB ChB, BPharm, FCP (SA); JF Botha,1,3 MB ChB, FCP (SA); S W van der Merwe,4,5 MB ChB, MSc, MMed, PhD; E Song,6,7 MB ChB, FCP (SA), FRCP (London); C Kassianides,8,9 MB ChB, FCP (SA); K A Newton,2,10 MB ChB, FCP (SA); H N Hairwadi,1 MB ChB, MMed, PhD

1 Division of Hepatology, Department of Medicine, University of Cape Town, South Africa
2 South African Gastroenterology Society, Mowbray, Cape Town, South Africa
3 Sandton Clinic, Bryanston, Johannesburg, South Africa
4 Department of Immunology, University of Pretoria, South Africa
5 Department of Clinical and Experimental Medicine, University of Leuven, Flanders, Belgium
6 Department of Medicine, University of the Witwatersrand, Johannesburg, South Africa
7 Donald Gordon Medical Centre, Johannesburg, South Africa
8 Morningside Clinic, Sandton, Johannesburg, South Africa
9 Gastroenterology Foundation of South Africa, Mowbray, Cape Town, South Africa
10 Department of Gastroenterology, Division of Clinical Medicine, University of KwaZulu-Natal, Durban, South Africa

Corresponding authors: C W N Spearman (wendy.spearman@uct.ac.za) and M W Sonderup (msonderup@ammedical.co.za)
NIH Guidelines: Indications for HBV Treatment

Patients for Whom Therapy Is Indicated

Patients who have

- Acute liver failure
- Decompensated cirrhosis
- Cirrhosis or advanced fibrosis and HBV DNA in serum
- Patients who will be receiving cancer chemotherapy or immunosuppressive therapy

NIH Guidelines: Indications for HBV Treatment

Patients for Whom Therapy May Be Indicated

• Active liver disease without advanced fibrosis or cirrhosis
  - HBeAg pos or HBeAg neg chronic hepatitis B

Patients for Whom Immediate Therapy Is Not Routinely Indicated

• Immune-tolerant phase (HBeAg pos, high serum HBV DNA levels, normal ALT or little activity on liver biopsy)

• Inactive carrier or immune control phase (HBeAg neg, low or undetectable levels of serum HBV DNA, and persistently normal ALT)

• Occult HBV infection (serum HBV DNA pos, IgG core pos, HBsAg neg)

Two Treatment strategies for CHB

Interferon-based therapy

• Dual Antiviral and immunomodulatory activity
• Finite course of treatment
• Aim for sustained off-treatment immune control (HBsAg +, HBeAg -) through dual mode of action

Nucleos(t)ide analogue therapy

• Antiviral activity
• Long-term (potentially indefinite) treatment
• Aim for on-treatment viral suppression (HBV DNA -)
• Maintained through continuous antiviral therapy
• Suppression of replication to undetectable levels to avoid resistance
HBeAg Positive Disease

End-points of treatment

• Ideal end-point sAg loss ± sAb

• Durable eAg loss and seroconversion

• Durable suppression of HBV DNA to low or undetectable
HBeAg loss and seroconversion in HBeAg+ Patients after 1 Yr of Treatment

-Not head-to-head trials; different patient populations and trial designs

head trials; different patient populations and trial designs

HBeAg Loss

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Outcome (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAM</td>
<td>17-32</td>
</tr>
<tr>
<td>ETV</td>
<td>21</td>
</tr>
<tr>
<td>TDF</td>
<td>18</td>
</tr>
<tr>
<td>Peg-IFN</td>
<td>33</td>
</tr>
</tbody>
</table>

HBeAg Seroconversion

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Outcome (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAM</td>
<td>22</td>
</tr>
<tr>
<td>ADV</td>
<td>12-18</td>
</tr>
<tr>
<td>ETV</td>
<td>21</td>
</tr>
<tr>
<td>TDF</td>
<td>21</td>
</tr>
<tr>
<td>Peg-IFN</td>
<td>30</td>
</tr>
</tbody>
</table>

Lowest HBsAg levels at week 12 are associated with highest rate of sustained immune control.

HBeAg positive patients treated with PEG-IFNα-2a +/- lamivudine for 48 weeks.

![Bar chart showing HBeAg seroconversion rates.](chart.png)

- **Low (<1500)**: 57% (51/90)
- **Medium (1500–20,000)**: 32% (72/223)
- **High (>20,000)**: 16% (14/86)

P<0.0001 for <1500 IU/mL vs higher levels

Piratvisuth et al. APASL 2010
HBeAg Negative Disease

End-points of Treatment

- Ideal end-point sAg loss ± sAb
- Durable suppression of HBV DNA to low or undetectable levels
- NUC therapy long-term as relapse common after stopping treatment
Virologic response in HBeAg- Patients (Undetectable* HBV DNA at Wk 48-52)

Not head-to-head trials; different patient populations and trial designs

*By PCR based assay (LLD ~ 50 IU/mL) except for some LAM studies.

Undetectable HBV DNA Over Time in HBeAg-Negative Patients

*Not head-to-head trials; different patient populations and trial designs*

Extended Treatment With Nucleos(t)ide Analogues vs Limited Duration (1 Yr) Peginterferon Treatment

<table>
<thead>
<tr>
<th></th>
<th>1 Yr</th>
<th>2 Yrs</th>
<th>3 Yrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entecavir</td>
<td>93</td>
<td>91</td>
<td>87</td>
</tr>
<tr>
<td>Tenofovir</td>
<td>63</td>
<td>15</td>
<td>16</td>
</tr>
<tr>
<td>Peginterferon</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Single center study.*
HBsAg Loss Over Time in HBeAg Negative Patients

*With sustained undetectable HBV DNA.

HBsAg, more than HBV DNA, can distinguish between relapsers and responders to PEG-IFN in HBeAg Negative patients.

*Moucari et al. Hepatology 2009*

*HBV DNA undetectable by PCR 1 year post-treatment*

**HBV DNA undetectable at EOT but detected in following 24 weeks**
To assess and Rx a patient with chronic HBV in 2013 you need the following as a minimum:

1. ALT/AST
2. TBr/albumin/INR
3. HBeAg, eAb
4. HBV viral load
5. US liver
6. Exclude HIV/HCV
7. Access to TDF [LAM]
8. IFN for selected patients
Treatment of Hepatitis C
Definitions: Virological Response

Rapid virological response (RVR)
- *Undetectable HCV RNA 4 weeks after initiating treatment*

Complete early virological response (cEVR)
- *Undetectable HCV at 12 weeks of treatment*

Sustained virological response (SVR)
- *Undetectable HCV RNA levels at 24 weeks post-treatment*
SVR = Viral Cure

- Nearly 100% of patients who achieve SVR remain undetectable during long-term follow-up\(^{[1-4]}\)

Outcomes in advanced fibrosis with/without a SVR

Liver-Related Death

5-yr occurrence

**SVR:** 4.4% (CI: 0% to 12.9%)  
**No SVR:** 12.9% (CI: 7.7% to 18.0%)  
\( P = .024 \)

Liver Failure

5-yr occurrence

**SVR:** 0%  
**No SVR:** 13.3% (CI: 8.4% to 18.2%)  
\( P = .001 \) (log likelihood)

Evolution of hepatitis C therapy

- Discovery of HCV genome
- Treatment with IFN alfa for 24 or 48 weeks – 3x weekly dosing – Poor outcomes
- Addition of RBV to IFN alfa improved outcomes
- Peg-IFN mono – once-weekly dosing
- Peg-IFN α plus RBV becomes gold standard

1989 to 2011
Results of HCV Rx: overall SVR rates

- **IFN 24 wk 1998**: 6
- **IFN 48 wk 1998**: 13
- **IFN + RBV 1998**: 41
- **PEG-IFN 2000-2002**: 39
- **PEG-IFN + RBV 2001-2004**: 63

References:
Peginterferon α-2a + Ribavirin: SVR According to Genotype

Predicting an SVR

Viral kinetics: Response guided therapy
Early virological response (EVR): HCV RNA ↓ ≥ 2 logs or Undetectable at Week 12

PegIFN/RBV

66% SVR

2 log decline

Limit of detection

EVR

SVR
EVR is an essential predictor of achieving SVR: 12-week stopping rule

All patients (n=453)

EVR*
Yes
86%
(n=390)

No
14%
(n=63)

SVR
65%
(n=253)

NPV=97%

Early virological response = >2 log_{10} drop in HCV RNA or undetectable at week 12

Ferenci P, et al.
Rapid Virological response (RVR): HCV RNA Undetectable at week 4

HCV RNA (log<sub>10</sub> IU/mL)

Weeks

0 1 2 3 4 5 6 7 8

PegIFN/RBV

90% SVR

2 log decline

Limit of detection

RVR

SVR
SVR in Patients Who Achieved an RVR
Similar Across Genotypes

RVR = HCV RNA negative (<50 IU/mL) at week 4; genotypes 1 and 4, patients were treated for 48 weeks; genotypes 2 and 3 patients were treated for 24 weeks
HCV negative at week 4 and 12

eRVR = Extended RVR
IL28B
A Polymorphism on Chromosome 19 Predicts SVR

IL28B rs12979860 polymorphism genotype frequency by population

- **European Americans**
  - C/C: 38%
  - C/T: 50%
  - T/T: 12%

- **African Americans**
  - C/C: 16%
  - C/T: 48%
  - T/T: 36%

- **Hispanics**
  - C/C: 35%
  - C/T: 46%
  - T/T: 19%

IL28B genotype drives phase 1 viral kinetics

- Genotype 1 HCV, IL28B rs12979860

Day 1, \(P < 0.001\)
Response Rates by \textit{IL28B} Polymorphism: GT 1 Treated With PegIFN/RBV

The polymorphism on chromosome 19, rs12979860 (T/T, T/C, or C/C), was strongly associated with SVR in all patient groups.

*IL28B* Polymorphisms and Response to PegIFN/RBV by HCV Genotype

• **CC IL-28B** genotype is the strongest pre-Rx predictor of SVR (OR 5.2; 95% CI, 4.1-6.7)

Thompson et al., *Gastroenterology* 2010;139:120-9
IL28B polymorphisms are not predictive in hepatitis C genotype 5 infected South African patients

Mark W. Sonderup¹, Wamda Abuelhassan², C Wendy Spearman¹
1. Department of Medicine and Division of Hepatology, Groote Schuur Hospital and University of Cape Town
2. Department of Gastroenterology, Chris Hani-Baragwanath Academic Hospital, University of the Witwatersrand, Soweto, Johannesburg.

63rd Annual Meeting of the American Association for the Study of Liver Diseases, Boston, MA, USA November 9 - 13 2012
### Demographic data of treated patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N = 32</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male, n (%)</td>
<td>18 (56%)</td>
</tr>
<tr>
<td>Age (y) mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>53.2±11.5</td>
</tr>
<tr>
<td>Woman</td>
<td>53.4±10.3</td>
</tr>
<tr>
<td>Ethnic group — no. (%)</td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>17 (53%)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>12 (38%)</td>
</tr>
<tr>
<td>Mixed Ancestry</td>
<td>3 (9%)</td>
</tr>
<tr>
<td>Weight — kg</td>
<td></td>
</tr>
<tr>
<td>median (range)</td>
<td>80 [53 – 130]</td>
</tr>
</tbody>
</table>
IL28B genotype – ethnic distribution

Blacks
- CC: 31
- CT: 47
- TT: 22

- Blacks: 47%
- CT: 41%
- TT: 12%

Caucasians
- CC: 33%
- CT: 58%
- TT: 8%

- Caucasians: 47%
- CT: 58%
- TT: 8%
Treatment outcomes

- **RVR**: 62%
- **cEVR**: 100%
- **SVR**: 78%

*lower level of detection of HCV is <15IU/ml*

*RVR = rapid virological response, cEVR = complete early virological response, SVR = sustained virological response and defined as undetectable HCV RNA at the end of a 24-week follow-up period*
## RVR and IL28B polymorphism genotype

<table>
<thead>
<tr>
<th>IL28B genotype</th>
<th>OR (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CC vs. non CC</td>
<td>1.6 (0.3 – 10.3)</td>
<td>0.58</td>
</tr>
<tr>
<td>CT vs. non CT</td>
<td>1.4 (0.3 – 5.9)</td>
<td>0.64</td>
</tr>
<tr>
<td>TT vs. non TT</td>
<td>0.4 (0.1 – 2.2)</td>
<td>0.33</td>
</tr>
</tbody>
</table>
SVR and IL28B polymorphism genotype

<table>
<thead>
<tr>
<th>IL28B genotype</th>
<th>OR (95% CI)</th>
<th>p- value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CC vs. non CC</td>
<td>0.6 (0.1 – 4.2)</td>
<td>0.62</td>
</tr>
<tr>
<td>CT vs. non CT</td>
<td>2.7 (0.4 – 16.7)</td>
<td>0.28</td>
</tr>
<tr>
<td>TT vs. non TT</td>
<td>0.5 (0.1 – 2.9)</td>
<td>0.45</td>
</tr>
</tbody>
</table>
Influence of ethnicity on achieving a SVR

Caucasian: 58%
Black: 59%

$p = NS$
### SUMMARY: SVR predictive factors

<table>
<thead>
<tr>
<th>Major factors&lt;sup&gt;[1-3]&lt;/sup&gt;</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Viral genotype (non–genotype 1)</td>
<td>Pretreatment HCV RNA (≤ 600,000 IU/mL)</td>
<td>RVR</td>
</tr>
<tr>
<td>IL28B allele (C/C vs. T/T)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Other factors&lt;sup&gt;[1,2,4]&lt;/sup&gt;</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose of Peg-IFN</td>
<td>Lower body weight (≤ 75 kg)</td>
<td></td>
</tr>
<tr>
<td>Dose of RBV</td>
<td>Absence of insulin resistance</td>
<td></td>
</tr>
<tr>
<td>Female sex</td>
<td>Elevated ALT levels (3 x ULN)</td>
<td></td>
</tr>
<tr>
<td>Younger age (younger than 40 yrs)</td>
<td>Absence of bridging fibrosis or cirrhosis</td>
<td></td>
</tr>
<tr>
<td>Ethnicity - non-Black</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Evolution of HCV Therapy

1990-2000

The Empiric Phase

2000-2011

The Refinement Phase
- Viral kinetics
- Optimal dosing
- Special populations
- Non-responders
- Genomics

Weisberg IS, Sigal SH, Jacobson IM. Current Hepatitis Reports. 2007;6:75-82.
DAA – Direct Acting Antivirals: Protease Inhibitors
HCV structure

Structural proteins: C, E1, E2, p7

Non-structural proteins: NS2, NS3, NS4A, NS4B, NS5A, NS5B

Protease

RNA polymerase

HCV (NS3) Protease Inhibitors

- Two protease inhibitors approved in 2011 for HCV genotype 1
- Telaprevir (Incivo/Incivek) and Boceprevir (Victrelis)
- As triple therapy in combination with pegylated interferon (PEG) and ribavirin (RBV)
SVR Rates With BOC or TVR + PR According to Treatment History

SVR Rates With BOC or TVR in GT1 Treatment-Experienced Patients

PEG/RBV + Telaprevir or Boceprevir
SVR: PEG-IFN naïve, GT1a vs. 1b

Telaprevir – T12PR

Boceprevir – BPR RGT

GT1a | GT1b
---|---
71 | 79

GT1a | GT1b
---|---
63 | 73

P=PEG-IFN, R=Ribavirin, RGT=Response Guided Therapy
T=Telaprevir, B=Boceprevir

Jacobson I, NEJM 2011; 364: 2405
Zeuzem S, EASL 2011
PEG/RBV + Telaprevir
GT1, IFN-experienced

Zeuzem S, NEJM 2011; 364: 2417
PEG/RBV + Telaprevir or Boceprevir
SVR: GT1, IFN-naïve, IL28B Genotypes

T12PR vs. PR

BPR RGT vs. PR

P=PEG-IFN, R=Ribavirin, RGT=Response Guided Therapy
T=Telaprevir, B=Boceprevir

Jacobson I, Poordad F
### Investigational HCV Regimens in Phase III Clinical Trials

#### Regimens With 1 DAA + PegIFN alfa/RBV
- Faldaprevir* (BI 201335, PI)
- Daclatasvir* (BMS-790052, NS5A)
- Sofosbuvir* (GS-7977, NI)
- Simeprevir* (TMC435, PI)
- Vaniprevir† (MK-7009, PI)

#### Regimens With 2 DAAs + PegIFN alfa/RBV
- Daclatasvir + asunaprevir*
- PegIFN lambda-1a + daclatasvir + RBV

#### IFN-Free Regimens
- Sofosbuvir + RBV
- Sofosbuvir + GS-5885 (FDC) ± RBV
- Daclatasvir + asunaprevir
- ABT-450/RTV + ABT-267 ± ABT-333 ± RBV

#### New Interferons
- PegIFN lambda-1a + RBV

#### Alternative Dosing
- TVR BID* (approved PI)

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*Studied with pegIFN-α2a. †Studied with both pegIFN-α2a and pegIFN-α2b.

ClinicalTrials.gov.
Sofosbuvir for Previously Untreated Chronic Hepatitis C Infection

Toward a Future of Personalized Medicine for HCV Therapy

Direct-Acting Antivirals

- Nuc + RBV
- NNI + PI ± RBV
- Nuc + NS5A Inh ± RBV
- PegIFN + RBV+ DAA
- Others?
<table>
<thead>
<tr>
<th>Tertiary Centre</th>
<th>Availability/# of patients per annum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gauteng - CMAH</td>
<td>Yes - ±6/year</td>
</tr>
<tr>
<td>Gauteng - CHB</td>
<td>Yes - 5/year</td>
</tr>
<tr>
<td>KZN - IALCH</td>
<td>Yes - no limit*</td>
</tr>
<tr>
<td>WC - Tygerberg</td>
<td>Yes – 5/year</td>
</tr>
<tr>
<td>WC - GSH</td>
<td>Yes – 6/year</td>
</tr>
</tbody>
</table>

* Quaternary EDL – Peg-IFN/RBV not listed
# HEPATITIS B Rx – AVAILABILITY OF Std and/or PEG-IFN AT TERTIARY CENTRES IN SA

<table>
<thead>
<tr>
<th>Tertiary Centre</th>
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<tbody>
<tr>
<td>Gauteng - CMAH</td>
<td>No</td>
</tr>
<tr>
<td>Gauteng - CHB</td>
<td>Yes – Peg-IFN</td>
</tr>
<tr>
<td>KZN - IALCH</td>
<td>Yes – Peg and Std IFN</td>
</tr>
<tr>
<td>WC - Tygerberg</td>
<td>No</td>
</tr>
<tr>
<td>WC - GSH</td>
<td>Only STD IFN</td>
</tr>
</tbody>
</table>
# HEPATITIS B Rx – AVAILABILITY OF Tenofovir/Lamivudine

<table>
<thead>
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<tbody>
<tr>
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<td>WC - Tygerberg</td>
<td>Yes</td>
</tr>
<tr>
<td>WC - GSH</td>
<td>Yes</td>
</tr>
</tbody>
</table>
Private sector – HEPATITIS C

• hepatitis C not a “PMB”
• variation amongst funders
• **Discovery Health:**
  ■ only consider funding if on classic/executive series
  ■ 20% co-payment
• **Medscheme administered funds:**
  ■ mostly fund – variability on co-pay – usually no co-pay
• **LIMS** – usually don’t consider funding Rx

Usually will fund Rx – problems arise with:
1. Funding blood tests whilst on Rx
2. Ability to access GM-CSF (Neupogen\textsuperscript{R}) and EPO if needed sometimes problematic
Private sector – HEPATITIS B

• hepatitis B not a “PMB”

• **Discovery Health**:  
  ■ NO funding for IFN or antivirals

• **Medscheme administered funds**:  
  ■ Will consider funding IFN with motivation  
  ■ Antivirals – often not

With motivation and appeal – may consider chronic benefits for long term antiviral Rx
OPINION: When the private health sector falls short

PROONENTS on both sides of the National Health Insurance (NHI) debate have emphasised improving service at public health facilities. They are right. Many clinics and hospitals are understaffed and poorly managed, with shortages of essential medicines and equipment. Patients have to wait in long queues. They are lucky if they see a pharmacist for assistance on the correct use of medicines.

The reasons for this are many and complex. The fragmented apartheid health system, the poor leadership until 2008 on the HIV/AIDS crisis, under-resourcing and poor management skills have affected the public health system. The skewed distribution of resources between private and public healthcare is a crucial factor.

Medical schemes are the predominant way most private patients finance their healthcare. According to the Council for Medical Schemes, in 2009 there were about 8-million medical scheme beneficiaries — about 17% of the population. However, schemes don’t cover all expenses and many private-sector users have to pay for medical services.

By contrast, 70% of people predominantly use the public health system. According to the Health Systems Trust, per capita health expenditure in the private sector was nearly five-and-a-half times per capita public sector expenditure in 2009. Despite this, schemes do not cover the treatment of many diseases, with many patients falling into the void between the public and private sectors. Perhaps spurred by the NHI discussions, private health providers are beginning to acknowledge that they need to do more and absorb a greater share of SA’s disease burden.

About two weeks before the release of the NHI green paper, the World Health Organisation marked World Hepatitis day. It passed fairly unnoticed in SA, a country where hepatitis B virus infection is endemic. Chronic hepatitis B infection accounts for about half of all cases of liver cancer in SA. Hepatitis C has a far lower prevalence but can cause chronic liver disease with high morbidity and mortality. Both infections are complicated in people with HIV. It is then ironic that treatments for chronic viral hepatitis are available in the public sector, but access in the private sector is more difficult where chronic viral hepatitis is not a prescribed minimum benefit (PMB).

Treatment of hepatitis C is with pegylated interferon, a medication injected weekly, together with ribavirin, a tablet taken daily. Treatment is expensive, requires specialist care and lasts for 24 or 48 weeks. Nevertheless, a public-sector patient with hepatitis C can access treatment. This is not the case for patients on medical schemes because hepatitis C is not a PMB.

Several schemes do cover the cost of treatment as an ex gratia benefit and patients thus benefit. However, the country’s biggest health insurer, Discovery Health, does not. Discovery offers treatment only for hepatitis C on its top two most expensive options and then a substantial co-payment is levied. Even for the well-off, this creates an invidious choice: risk financial ruin or risk morbidity and even death. Treatment for hepatitis B is also not covered by medical schemes. The exception to this is HIV-positive patients with hepatitis B, as they can readily access antiretroviral therapy, in which two of the drugs used are active against the hepatitis B virus as well.

We therefore have a clear situation in which public-sector patients with hepatitis are better off than private medical scheme ones. This paints a distinctly more complicated picture of the differences in public and private healthcare in SA to the one we usually read about. In this case, the public sector is absorbing a great burden and providing good service, while medical schemes leave patients without care. Not only is private medical care much more expensive but, in this case, it offers less.

Medical schemes such as Discovery must do their share and cover the full cost of chronic viral hepatitis treatment. The Council for Medical Schemes must take steps to ensure that treatment for chronic viral hepatitis becomes a PMB. These are all opportunities for the private sector to show whether it is interested in doing more to relieve the burden on the public health system or whether it is just making rhetorical noise in response to the perceived threat of NHI.

• Sonderup and Spearman are hepatologists at UCT and Groote Schuur Hospital. Geffen is with the Treatment Action Campaign.
Does the private health sector fall short?

Author: Jasson Urbach

Critics of private health care in SA argue that the private sector does not do enough, that it dumps patients on the public sector and is only interested in "money first". Sonderup, Spearman and Geffen recently argued in the Business Day article, *When the private sector falls short*, 3 Oct. 2011, that medical schemes are evil because they “do not cover the treatment of many diseases causing many patients to fall into the void between the public and private sectors”. That “it is then ironic that treatments for chronic viral hepatitis are available in the public sector, but access in the private sector is more difficult where chronic viral hepatitis is not a prescribed minimum benefit (PMB)”. 

**However, just because a condition is not a PMB, does not mean medical aids do not cover it.** In fact, any legitimate treatment will be covered subject to the rules of the scheme. The only difference is that, if the condition is not a PMB, the scheme is not obliged to pay the doctors in full regardless of what they charge for their services.

Sonderup, Spearman and Geffen then suggest that “The Council for Medical Schemes must take steps to ensure that treatment for chronic viral hepatitis becomes a PMB”. The fact that two of the critics in this case happen to be hepatologists and are arguing that chronic viral hepatitis should be covered as a PMB is equally ironic. **They are surely arguing in their own self-interest and wish to have viral hepatitis declared a PMB to enhance their own incomes.** It is government that determines which conditions are included in the list of PMBs, not medical schemes, and certainly no one individual scheme. And herein lies an important point. When benefits are determined politically rather than by medical schemes responding to what individuals want, the benefit packages expand and their costs increase.

**Date:** 10 October 2011

The consequence is that low cost medical schemes that cover the basic needs of low-income people can no longer be efficiently designed and the unfortunate low income earners are denied cover. It is then that they are driven into “the void between the public and private sector”.

Medical schemes are not charities; they are obliged by economic realities and the interests of the members of their schemes to take great care in managing available resources. If scheme managers were to recklessly pay claims that are not included in the agreements with scheme members they would be guilty of dereliction of duty and would threaten the solvency and continued existence of the schemes they are managing. They have to stick to the rules and ensure that they do not bankrupt the schemes.

**Eminent economists have declared that because people’s health, or lack of it, lies largely and increasingly within their own - and earlier their parents’ - control, many, if not most health risks are actually uninsurable.** Risk pooling and intense actuarial and managerial effort is employed in an attempt to overcome the innate problems that are consequently bound to face private medical scheme managers. Theirs is an almost impossible task and regulatory interventions make their task even more difficult, very often to the detriment of the majority of medical scheme members.

**AUTHOR** Jasson Urbach is a director of the Health Policy Unit (a division of the Free Market Foundation). This article may be republished without prior consent but with acknowledgement to the author. The views expressed in the article are the author’s and are not necessarily shared by the members of the Foundation.
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Every doctor/HCW is an activist
"Knowing is not enough, we must apply.

Willing is not enough, we must do,"

Goethe